Fiber type-specific muscle glycogen sparing due to carbohydrate intake before and during exercise

K. De Bock, W. Derave, M. Ramaekers, E. A. Richter, and P. Hespel

Research Center for Exercise and Health, Faculty of Kinesiology and Rehabilitation Sciences, Katholieke Universiteit Leuven, Leuven, Belgium; and Copenhagen Muscle Research Centre, Institute of Exercise and Sports Sciences, University of Copenhagen, Copenhagen, Denmark

Submitted 20 July 2006; accepted in final form 25 September 2006

De Bock K, Derave W, Ramaekers M, Richter EA, Hespel P. Fiber type-specific muscle glycogen sparing due to carbohydrate intake before and during exercise. J Appl Physiol 102: 183–188, 2007. First published September 28, 2006; doi:10.1152/japplphysiol.00799.2006.—The effect of carbohydrate intake before and during exercise on muscle glycogen content was investigated. According to a randomized crossover study design, eight young healthy volunteers (n = 8) participated in two experimental sessions with an interval of 3 wk. In each session subjects performed 2 h of constant-load bicycle exercise (~75% maximal oxygen uptake). On one occasion (CHO), they received carbohydrates before (~150 g) and during (1 g·kg body weight^{-1}·h^{-1}) exercise. On the other occasion they exercised after an overnight fast (F). Fiber type-specific relative glycogen content was determined by periodic acid Schiff staining combined with immunofluorescence in needle biopsies from the vastus lateralis muscle before and immediately after exercise. Preexercise glycogen content was higher in type Ila fibers [9.1 ± 10^−5 optical density (OD)/μm^2] than in type I fibers [8.0 ± 10^−5 OD/μm^2; P < 0.0001]. Type Ila fiber glycogen content decreased during F from 9.6 ± 10^−5 OD/μm^2 to 4.5 ± 10^−5 OD/μm^2 (P = 0.001), but it did not significantly change during CHO (P = 0.29). Conversely, in type I fibers during CHO and F the exercise bout decreased glycogen content to the same degree. We conclude that the combination of carbohydrate intake both before and during moderate- to high-intensity endurance exercise results in glycogen sparing in type Ila muscle fibers.

insulin; human muscle; glycogen breakdown

IT HAS BEEN WELL DOCUMENTED that glycogen depletion plays an important role in fatigue during prolonged exercise (3, 4, 39). Accordingly, it is generally assumed that dietary strategies to spare muscle glycogen can enhance endurance performance. Conversely, procedures causing exaggerated glycogenolysis may impair performance. Literature data on the effects of exogenous carbohydrate intake on exercise-induced net glycogen breakdown are equivocal. It has been shown that elevated starting plasma insulin level due to the ingestion of preexercise carbohydrates can inhibit fat oxidation during the initial stage of exercise, which in turn is compensated for by increased net glycogen degradation to maintain the required rate of ATP production (8, 10, 25). However, this greater glycogen breakdown can be avoided by adequate carbohydrate intake during exercise. Thus euglycemia is maintained and blood-borne glucose serves as an extra energy source (16), which in turn may result in decreased glycogen breakdown. Data concerning the degree of glycogen sparing during exercise with carbohydrate intake are currently lacking. Notwithstanding this, the use of carbohydrates is common practice even during cycling exercise of short (~1–2 h) duration, and it is associated with increased performance (7, 16, 47). Furthermore, published studies have shown that fatigue during prolonged exercise under 90% of maximal oxygen uptake is not only highly correlated with glycogen depletion (4) in the active muscles but probably also plays a role in the etiology of muscle fatigue (3, 39). Moreover, glycogen appears to be a signaling molecule that can regulate muscle gene transcription (20). Decreases in glycogen breakdown can therefore have substantial influence on exercise capacity and muscle metabolism.

Current knowledge on the effect of dietary interventions on muscle glycogen breakdown during exercise is largely based on measurements of glycogen concentration in mixed muscle tissue. However, muscles are composed of type I and type II motor units, which exhibit very diverse metabolic properties (48). Net glycogen degradation in muscle fibers during contractions depends on the balance between glycogen synthase and phosphorylase activity. It is well established that contraction intensity (27), the glycogen concentration existing in the muscle cell (23, 24), as well as circulating insulin level (8) play a pivotal role in regulation of this balance. Type I and type II motor units are differentially recruited during prolonged endurance exercise (2, 19). Furthermore, the glycogen synthase and phosphorylase enzymes are differentially expressed between fiber types, with type I fibers exhibiting lower phosphorylase vs. higher glycogen synthase activity compared with type II fibers (11, 29, 36). Therefore, it is reasonable to assume that the effect of carbohydrate intake on net glycogen degradation during exercise can differ between muscle fiber types.

Our laboratory has recently reported that 2 h of moderate- to high-intensity (~70% maximal oxygen uptake) exercise in the fasted state, compared with and identical exercise bout in conjunction with carbohydrate intake before and during exercise, stimulated net breakdown of intramyocellular lipids in type I fibers but not in type Ila fibers (12). Surprisingly, net glycogen degradation over the 2-h exercise period, assessed by biochemical assay of pre- and postexercise glycogen concentrations in mixed muscle samples, was not significantly different between dietary conditions. However, using such an approach, it may be difficult to demonstrate glycogen sparing because of 1) the presence of a significant fraction of inactive fibers in the tissue sampled, which may mask the effects in the active fibers; 2) differential fiber composition of muscle biopsies obtained before and after exercise; and 3) diverse regulation of net glycogenolytic rate between fiber types. Therefore,
we decided to readdress the effect of exercise in the fasted state, vs. exercise with adequate carbohydrate intake, on muscle glycogen degradation using a fiber-specific histochemical method. The data show that carbohydrate intake resulted in glycogen sparing in type IIa fibers.

METHODS

Protocol

Subjects. Eight healthy, physically active men (age, 23.0 ± 0.4 yr; body weight, 74.9 ± 2.4 kg) volunteered to participate in the study, of which detailed methodology as well as results of biochemical glycogen and blood analyses have been previously published (12). The study protocol was approved by the local Ethics Committee (Katholieke Universiteit Leuven, Faculty of Medicine). Subjects gave their written informed consent after they were informed in detail of all experimental procedures and risks possibly associated with the experiments.

Protocol. Briefly, the study was designed as a balanced and randomized crossover study in which all subjects participated in two experimental sessions with a 3-wk interval during which the subjects were instructed to keep their exercise pattern as normal as possible. In one session, the subjects performed a 2-h exercise bout in the fasted state (F), whereas in the other condition they received carbohydrate supplements before and during exercise (CHO). Subjects received a standardized diet containing 3,000 kcal/day (61% carbohydrates, 23% fat, and 15% protein) for 3 days before each session and were also instructed to abstain from any strenuous physical exercise during this period.

On the morning of the experiments, the subjects arrived at the laboratory between 6:00 and 9:00 AM after an 11-h overnight fast. They were assigned to either of two experimental conditions. During CHO, one-half of the subjects received a standardized carbohydrate-rich breakfast (722 kcal: 85% carbohydrates, 4% fat, 11% protein), whereas in F they remained fasted. After a 2-h rest period, a percutaneous needle biopsy sample was taken from the right vastus lateralis muscle under local anesthetic (2–3 ml lidocaine) through a 5-mm incision in the skin. Furthermore, a blood sample was taken from an antecubital vein. Thereafter, subjects cycled for 2 h at a workload (179 ± 9 W) and cadence (91 ± 3 rpm), which were fixed during the two familiarization sessions preceding the experiments. Before, halfway through and at the end of the exercise bout, oxygen uptake and carbon dioxide output were measured over a 5-min interval (Jaeger, Oxycon Alpha). Capillary blood was sampled from a hyperemic earlobe before and after the first and second hour of exercise. In the CHO condition, subjects received 1 g maltodextrine·kg body weight⁻¹·h⁻¹ in a flavored 15% solution during the exercise bout. Conversely, during F they received an equal amount of water that was sweetened (1.14 g/l sodium cyclamate, 11.4 g/l sodium saccharine) and flavored to be similar in taste and appearance with the maltodextrine solution. At the end of the exercise bout, another muscle biopsy and venous blood sample were taken. The muscle biopsy was taken through the same incision as the preexercise biopsy but with the needle pointing in another direction.

Analysis of Muscle Samples

Muscle biopsy handling. After being freed from any visible nonmuscle material, part of the muscle sample was immediately frozen in liquid nitrogen, and the remaining part was mounted in embedding medium cooled in isopentane. All samples were stored at −80°C for later analysis. Muscle glycogen content in mixed muscle was measured as glucose residues after acid hydrolysis in freeze-dried muscle tissue using a standard enzymatic fluorometric assay (30).

Fiber typing and periodic acid staining. Serial sections (4 μm) from biopsy samples collected before, immediately after, and 4 h after exercise were laid together on uncoated glass slides. The method to quantify fiber type-dependent glycogen content was adapted from Schaart et al. (38). Briefly, cryosections were fixed for 1 h at 4°C in 3.7% formaldehyde in PBS immediately after removal from the freezer. Slides were rinsed for 5 min in PBS and were then treated with 0.1% Triton X-100 in PBS for 5 min and again washed for 5 min in PBS and 30 s in deionized water. Thereafter, slides were pretreated for 5 min with 1% periodic acid in deionized water followed by a washing step for 1 min with running tap water and a wash dip in deionized water for 5 s. Slides were incubated in Schiff’s reagent for 15 min at room temperature and subsequently washed under running tap water for 5 min. Thereafter, sections were washed for 5 min in PBS and incubated overnight at 4°C with two primary monoclonal antibodies against human myosin heavy chain I (A4.840 supernatant, Developmental Studies Hybridoma Bank, Iowa City, Iowa) and IIa (N2.261 supernatant, Developmental Studies Hybridoma Bank) to determine muscle fiber type I (and IIa, respectively). Incubation was followed by three 5-min washes with wash buffer, after which the appropriate conjugated antibodies (type I: FITC-anti-mouse IgM and Cy3-anti-rabbit IgG, Developmental Studies Hybridoma Bank, Iowa City, IA; type IIa: Alexa Fluor 350 anti-mouse IgG1 Molecular Probes, Leiden, The Netherlands) were added. After a last wash for 3 × 5 min in PBS, coverslips were mounted with Fluorescent Mounting Medium (DakoCytoMAX, Carpinteria, CA).

Slides were examined using a Nikon E1000 fluorescence microscope (Nikon, Boehravendorp, Germany) equipped with a digital camera. Periodic acid Schiff (PAS)-stained sections were examined in bright field, and special care was taken to capture images with identical camera (exposure time: 4 ms) and microscopy (light intensity: 11.3 V; ND filter: 72°) settings. Epifluorescence signal was recorded using FITC and DAPI filter for type I and IIa muscle fibers, respectively (Fig. 1). Fibers, negatively stained for type I and type IIa, were qualitied as type IIX fibers. Captured images (×20 magnification) were processed and analyzed using Lucia G software (LIM, Prague, Czech Republic). The bright-field image of the PAS stain was converted post hoc to gray-scale values, and the mean optical density (OD) of each fiber was semiquantified, resulting in a total of 178 ± 19 muscle fibers analyzed for each muscle cross section (101 ± 12 type I, 65 ± 9 type IIa and 11 ± 4 type IIX muscle fibers). The fraction of type I, IIa, and IIX fibers was 57 ± 3, 36 ± 2, and 6 ± 2%, respectively. Because of the small number of type IIX fibers, it was possible to evaluate glycogen breakdown in both sessions in only three subjects. Therefore, results for type IIX quantification are not included in this paper. Mean OD of a selected region outside the muscle section, which was equal to the OD of the sections after α-amylase treatment, was used for background correction. Fiber-type specific glycogen content was expressed as mean optical density per square micrometers. To reduce any analytical error, all slides were analyzed within the same assay. The intra-assay coefficient of variation was 1.7%.

Analysis of Blood Samples

Venous blood samples were collected into vacuum tubes containing Silica Clot Activator (BD Vacutainer, Becton Dickinson, Franklin Lakes, NJ). Tubes were centrifuged (1,500 rpm for 15 min at 4°C), and the supernatant was stored at −80°C until later analysis. Capillary blood samples were collected (50 μl) into heparinized glass capillaries and were immediately analyzed for blood glucose concentration using an Analox GM7 analyzer (Analox Instruments, London, UK). Plasma insulin was assayed using a commercially available radioimmunoassay kit (Human Insulin, Biotech-IgG, Hamburg, Germany). Plasma catecholamines were determined by means of a high-performance liquid chromatography method using electrochemical detection.

Statistical Analyses

Treatment effects were evaluated using a repeated-measures ANOVA. Two-way ANOVA was performed to examine the interac-
tion between treatment and time. In case the ANOVA yielded a significant effect, a planned contrast analysis was used for post hoc comparisons. Regression analyses were performed using Pearson’s product-moment correlation analysis. A probability level of $P \leq 0.05$ was considered statistically significant. All data are expressed as means ± SE.

RESULTS

Muscle Glycogen (Figs. 1 and 2)

Initial glycogen content in type IIa fibers was ~15% higher than in type I fibers ($P < 0.0001$), but it was similar between experimental conditions. The 2-h exercise bout on average reduced glycogen content in type IIa fibers from 9.6 ± 1 to 4.5 ± 1 × 10^{-2} \text{ OD/\mu m}^2$ in F ($P = 0.001$) vs. from 8.7 ± 1 to 7.1 ± 2 × 10^{-2} \text{ OD/\mu m}^2$ in CHO ($P = 0.29$, not significant, interaction $P = 0.043$). Conversely, exercise-induced glycogen degradation in type I fibers was independent of the experimental condition. Biochemically assayed mixed muscle glycogen contents were 455 ± 40 and 466 ± 19 mmol/kg dry weight preexercise, decreasing to 206 ± 31 and 130 ± 33 mmol/kg dry weight postexercise in CHO and F, respectively. Net glycogen degradation amounted to by a standard enzymatic fluorometric assay was 149 ± 25 mmol·kg dry weight^{-1}·h^{-1} in F vs. 111 ± 24 mmol·kg dry weight^{-1}·h^{-1} in CHO but was not significantly different between experimental conditions ($P = 0.23$, not significant). Mixed-muscle glycogen concentration measured by biochemical assay correlated with glycogen concentration estimated from PAS-stained sections weighted for the proportion of type I and IIa fibers ($r = 0.65$, $P < 0.0001$).

Blood measurements (Table 1). From the start to the end of the 2-h exercise bout, blood glucose concentration gradually decreased in both CHO and F ($P < 0.0001$), but it was higher at any time in CHO ($P = 0.002$, Table 1). Plasma insulin levels dropped during exercise in both conditions ($P = 0.03$), yet they were about eightfold higher in CHO than in F throughout the session ($P = 0.003$). Plasma epinephrine concentration pre-exercise was similar between trials. However, exercise increased plasma epinephrine about threefold in F ($P = 0.01$) but not in CHO (Table 1).

The respiratory gas exchange ratio was measured at the start, midway, and at the end of the exercise bout. The respiratory gas exchange ratio was consistently lower in F compared with CHO ($P = 0.004$) and gradually decreased from 0.88 ± 0.02 to 0.81 ± 0.02 in F and from 0.93 ± 0.02 to 0.88 ± 0.02 in CHO during the 2-h exercise period ($P = 0.002$).

DISCUSSION

In the present study, we show that a 2-h moderate- to high-intensity exercise bout in the fasted state induced substantial glycogen breakdown in both type I and type IIa fibers. However, carbohydrate intake before and during exercise significantly inhibited net glycogen degradation in type IIa fibers but not significantly in type I fibers. This is the first study to demonstrate that supplementary carbohydrate intake before and during high-intensity endurance exercise, for a given preexercise glycogen concentration and exercise intensity, induces fiber type-specific glycogen sparing.

It is well established that carbohydrate intake can enhance endurance performance in both running (7, 43) and cycling (9, 32). For more than 20 yr, considerable controversy has existed as to whether muscle glycogen sparing contributes to this beneficial effect of carbohydrate intake. In fact, to the best of our knowledge, only two studies found decreased glycogen utilization during continuous cycling exercise with carbohydrate intake (4, 14), whereas others reported unchanged net glycogen breakdown (9, 21, 34) in either mixed muscle tissue or in pools of isolated type I and type II muscle fibers (9, 34). Based on the above studies, Tsintzas and colleagues (35, 43,
After an overnight fast, plasma insulin is in the range of intake during subsequent exercise. In this regard, all of the aforementioned studies used either the carbohydrate-fed state or in the fasted state (open bars). Values are means for = 8 subjects. OD, optical density. *P < 0.05 vs. preexercise.

Fig. 2. Effect of exercise on fiber type-specific glycogen content. Muscle glycogen content for type I (A) and type IIa fibers (B) before and immediately after exercise as determined on periodic acid Schiff-stained muscle cross sections. Exercise was performed either in the carbohydrate-fed state (solid bars) or in the fasted state (open bars). Values are means for = 8 subjects. OD, optical density. *P < 0.05 vs. preexercise.

44) suggested that, because increases in plasma glucose and insulin concentrations are less marked in cycling than in running, carbohydrate intake only causes net glycogen sparing in the latter exercise. In fact, Tsintzas and Williams (41) pinpointed the pivotal role of insulin in the potential of carbohydrate intake to induce muscle glycogen sparing during exercise. Besides exercise mode, preexercise carbohydrate intake also is a primary determinant of circulating insulin concentration during subsequent exercise. In this regard, all of the aforementioned studies used an overnight fast as a standard dietary preexercise condition. Nonetheless, ingestion of a carbohydrate-rich (3–5 g CHO/kg body weight) precompetition meal is a standard nutritional guideline to endurance athletes. It is well established that such meal often results in elevated plasma insulin levels during exercise with high rate carbohydrate intake after an overnight fast, plasma insulin is in the range of 5–15 µU/ml (9, 16, 32). In this range of low insulin concentrations, insulin action on glucose uptake in human muscles is conceivably small (37). Conversely, after a carbohydrate-rich meal, plasma insulin concentrations are significantly higher (~40–100 µU/ml), which at least during the initial stages of prolonged exercise is likely to cause substantial stimulation of muscle glucose uptake and inhibition of lipolysis. Therefore, an overnight fast may be an easy way indeed to obtain a standardized preexercise nutritional state; however, extrapolation of data to real-life exercise conditions is uncertain.

It is well established that the metabolic substrate pool for energy production during exercise with carbohydrate intake is shifted from fat to carbohydrate oxidation (1, 16, 26, 28, 33, 40). Interestingly, despite increased rate of overall carbohydrate oxidation with carbohydrate feeding, in the present study net glycogen breakdown tended to be smaller compared with fasting conditions, which means that blood glucose oxidation must have been substantially increased by carbohydrate feeding. Several mechanisms may explain this shift from muscle glycogen to blood glucose oxidation. In CHO, plasma insulin levels were suprabasal throughout exercise (~35 µU/ml at the start to 12 µU/ml at the end), whereas in F, insulin level dropped to an insulin-deficient state (4–1.5 µU/ml). Insulin deficiency is known to suppress muscle glucose uptake during exercise (6, 46). Besides the high circulating insulin level, elevated blood glucose concentration and low plasma free fatty acid levels (12) may also have facilitated blood glucose uptake in CHO and activated the glycogen synthase enzyme. Furthermore, the substantially higher insulin-to-epinephrine ratio (Table 1) during exercise combined with carbohydrate intake is likely to have suppressed net glycogenolytic rate.

Implicit to the above rationale is the assumption that muscle fiber recruitment was identical between nutritional conditions. Vollestad and coworkers (45), based on muscle glycogen-depletion pattern, reported that both type I and type IIa fibers are recruited from the start of exercise at 75% maximal oxygen uptake in the fasted state. However, there are also data to indicate that in the later stages of a high-intensity endurance exercise bout, additional type IIa motor units are progressively recruited to compensate for fatigue in glycogen-depleted type I fibers (19). In the present study, we obtained muscle biopsies only at the start and at the end of the exercise bout, which does not depict the time course of glycogen breakdown during exercise. In addition, the PAS data are semiquantitative, which does not allow to exactly quantify the degree of postexercise glycogen depletion. Still, the comparison of PAS-stained sections with α-amylase-treated sections performed in pilot experiments, indicates that by the end of exercise in the fasted state, type I fiber glycogen content was largely depleted in either experimental condition. Thus we cannot exclude the

Table 1. Effect of exercise on blood glucose and plasma hormones

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Mid</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, mmol/l</td>
<td>CHO 5.7±0.3</td>
<td>4.8±0.2*</td>
<td>4.1±0.2*</td>
</tr>
<tr>
<td>F 4.0±0.6†</td>
<td>4.0±0.6†</td>
<td>3.5±0.1†</td>
<td>11.5±1.8*†</td>
</tr>
<tr>
<td>Insulin, µU/ml</td>
<td>CHO 43.7±1.1†</td>
<td>4.1±0.1††</td>
<td>3.5±0.1††</td>
</tr>
<tr>
<td>F 4.0±0.6†</td>
<td>4.0±0.6†</td>
<td>1.4±0.5††</td>
<td>0.26±0.05††</td>
</tr>
<tr>
<td>Epinephrine, µg/l</td>
<td>CHO 0.09±0.01</td>
<td>0.13±0.03</td>
<td>0.26±0.05††</td>
</tr>
</tbody>
</table>

Values are means ± SE for (n = 8) The subjects. Values were measured before (Pre), halfway through (Mid; for glucose measurements only), and immediately after (Post) a 2 h-exercise bout exercise was performed either in the fasted state (F) or in the carbohydrate-fed state (CHO). See Methods for further details. *P < 0.05 vs. pre. †P < 0.05 vs. CHO.
possibility that carbohydrate intake suppressed rate of glycogen breakdown in type I fibers during the earlier stages of exercise. However, by the end of the full exercise bout, type I glycogen stores were depleted to nearly similar low levels in either experimental condition, thereby possibly masking early glycogen sparing in type I fibers. Such mechanism is compatible with earlier observations by Tsintzas et al. (42–44) showing substantial glycogen sparing in type I fibers during prolonged submaximal exercise. However, in the latter study, the residual glycogen depot in type I fibers after exercise with carbohydrate intake (≈60% of initial glycogen content) was substantially greater than in the present study (≈30% of initial glycogen content; Fig. 2). Along the same line of reasoning, early glycogen sparing during exercise with carbohydrate intake may have postponed recruitment of additional type IIa motor units to maintain power output during the later stages of the exercise. Such mechanism in turn may explain net glycogen sparing in type IIa fibers from the start to the end of the 2 h cycling bout (Fig. 2). Tsintzas et al. previously reported carbohydrate intake during prolonged submaximal exercise not to affect net glycogen breakdown in type II fibers (42–44). This discrepancy may be partly due to the fact that running and cycling can show markedly different metabolic responses (13) on the one hand and the absence of a carbohydrate-rich preexercise meal in their study compared with ours on the other hand. However, the different findings might also be due to the different approaches used to measure fiber specific glycogen concentration. Tsintzas and co-workers used a biochemical assay on pooled single muscle fibers dissected from needle biopsy samples obtained in only five subjects. In this procedure, type II fibers are isolated from type I fibers by myosin ATPase staining of the isolated fiber tips. The most darkly stained fibers are isolated as type II vs. the unstained ones as type I. The moderately stained fibers are discarded. As a result of such procedure, the number of selected fibers (5–10 per sample) is smaller for type II than for type I fibers. In addition, the pools of type II fibers conceivably contain a fraction of type IIX fibers that are unlikely to be recruited during submaximal exercise. Conversely, in the present study, each glycogen data point represents the mean of 60 type II fibers in eight subjects. Moreover, all fibers measured were identified as type IIa by using antibodies directed against type IIa myosin heavy chains, and type IIX fibers were excluded from the analyses. Therefore, it is probably reasonable to state that our present protocol may be more sensitive to detect changes in exercise-induced breakdown in type IIa fibers.

Our present data indicate that it is important to use a fiber-specific approach to address issues related to metabolism in contracting skeletal muscle. This is particularly true for submaximal exercise during which only a fraction of the total motor unit population is being recruited. In line with literature findings (5, 31), the biopsies we obtained from vastus lateralis muscle at various times during the mixed-muscle tissue, have previously failed to demonstrate significant glycogen sparing due to carbohydrate intake in conjunction with exercise.

In endurance competitions, performance largely depends on the ability to sustain high-power output/speed throughout the race. However, exercise intensity must be well tuned to spare energy for even higher intensity episodes in search of success during the final stages of the race. Power output at the end of a race at least partly depends on glycogen availability in type IIa motor units. Our present observations clearly demonstrate that adequate carbohydrate intake before and during exercise causes glycogen sparing in type IIa fibers. Furthermore, Tsintzas and coworkers (42) have previously shown that carbohydrate intake during exercise also can prevent premature phosphocreatine depletion in type II muscle fibers. Thus improved sprint performance due to carbohydrate intake at the end of an endurance exercise bout (18, 22) could be explained by both increased glycogen and phosphocreatine availability in type II fibers.

In conclusion, this is the first study to clearly demonstrate that adequate carbohydrate intake before and during moderate-to high-intensity endurance exercise, compared with exercise in the fasted state, inhibits glycogen breakdown at least in type IIa fibers. This glycogen sparing may be the reason for enhanced sprint performance observed by others at the end of an endurance exercise bout when carbohydrate is supplied during exercise (15).

GRANTS

This study was supported by grants from the Onderzoeksraad KU Leuven (no. OT04/45), the Fonds voor Wetenschappelijk Onderzoek Vlaanderen (no. G.0233.05), the Copenhagen Muscle Research Centre, the Media and Grants Secretariat of the Danish Ministry of Culture, the Danish Diabetes Association, the Novo Nordisk Foundation, the Danish Medical Research Council, and the European Commission (Integrated Project LSHM-CT-2004-005272). The monoclonal MHC antibodies, developed by Helen M. Blau, were obtained from the Developmental Studies Hybridoma Bank developed under the auspices of the National Institute of Child Health and Human Development and maintained by The University of Iowa, Department of Biological Sciences (Iowa City, IA 52242).

REFERENCES


