

Carbohydrate Intake and Recovery From Prolonged Exercise

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The influence of increased carbohydrate intake on endurance capacity was investigated following a bout of prolonged exercise and 22.5 hrs of recovery. Sixteen male subjects were divided into two matched groups, which were then randomly assigned to either a control (C) or a carbohydrate (CHO) condition. Both groups ran at 70% VO_{2max} on a level treadmill for 90 min or until volitional fatigue, whichever came first (T1), and 22.5 hours later they ran at the same % VO_{2max} for as long as possible to assess endurance capacity (T2). During the recovery, the carbohydrate intake of the CHO group was increased from 5.8 (± 0.5) to 8.8 (± 0.1) g kg^{-1} BW. This was achieved by supplementing their normal diet with a 16.5% glucose-polymer solution. An isocaloric diet was prescribed for the C group, in which additional energy was provided in the form of fat and protein. Run times over T1 did not differ between the groups. However, over T2 the run time of the C group was reduced by 15.57 min ($p < 0.05$), whereas those in the CHO group were able to match their T1 performance. Blood glucose remained stable throughout T1 and T2 in both groups. In contrast, blood lactate, plasma FFA, glycerol, ammonia, and urea increased. Thus, a high carbohydrate diet restored endurance capacity within 22.5 hrs whereas an isocaloric diet without additional carbohydrate did not.

Key Words: carbohydrate metabolism, endurance running, rehydration, diet

Optimal muscle glycogen stores are an important factor in successful endurance performance (20, 23). Prolonged exercise at 60 to 80% VO_{2max} will reduce muscle glycogen, and this is believed to be a major contributor to the onset of fatigue (6, 18). This carbohydrate store must be replenished if endurance capacity is to be restored, where endurance capacity refers to the exercise time to exhaustion at a constant running speed. The restoration of normal muscle glycogen resting values is dependent upon an individual's postexercise carbohydrate intake (1, 4, 5). Several studies have reported on the time course of muscle glycogen resynthesis following exercise (8, 21, 22, 34). Costill et al. (14) demonstrated normalization

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of muscle glycogen within 24 hours after prolonged running, when a diet containing between 525 g (7.3 g kg⁻¹ BW) and 648 g (8.2 g kg⁻¹ BW) of carbohydrate was consumed. Such a high carbohydrate intake may be satisfactorily incorporated into a nutritionally well balanced diet (17).

Keizer et al. (24) conducted one of the few studies to examine physical performance following exhaustive exercise and glycogen restoration. Maximal physical work capacity (MPWC) was determined before and after exhaustive intermittent cycling and a prescribed diet. The recovery diets provided between 577 g (7.8 g kg⁻¹ BW) and 602 g (8.2 g kg⁻¹ BW) of carbohydrate, which resulted in the restoration of muscle glycogen within 22 hours. Despite the body's carbohydrate stores being replenished in this time, MPWC was reduced by 7.3%.

However, there is little information on the return of endurance capacity along with glycogen repletion. Thus the aim of this study was to examine the ability to sustain exercise following a bout of prolonged running and a recovery diet containing additional carbohydrate.

Methods

Subjects

Sixteen male subjects took part in the study. All were engaged in various training programs, of which submaximal running was a central feature (average weekly training distances ranged from 40 to 100 km). The physiological characteristics of the subjects are described in Table 1. The procedures used were approved by the University Ethical Advisory Committee. Prior to receiving their written consent, subjects were informed of the demands of the study and the possible risks and discomforts. They were also required to complete a medical history questionnaire and provide general details of their running ability.

Dietary Analysis

Before the start of the study, subjects weighed and recorded their food intake over a continuous 7-day period. Analyses of the nutritional content of their normal diets were made (33) from these food diaries. Thus, guidance was provided for the dietary prescriptions during the recovery period.

Table 1
**Physiological Characteristics for Subjects
in the Control and Carbohydrate Groups**

Group	Age (yrs)	Height (cm)	Weight (kg)	$\dot{V}_{E\text{max}}$ (L·min ⁻¹)	HR _{max} (b·min ⁻¹)	$\dot{V}\text{O}_{2\text{max}}$ (ml·kg ⁻¹ ·min ⁻¹)
Control	25.2	175.8	70.3	130.9	186	65.0
$\pm SE$	2.1	3.0	2.4	7.1	3	2.3
CHO	25.6	176.7	72.7	136.3	188	65.0
$\pm SE$	1.8	2.8	2.5	2.5	3	2.2

Over the 48 hours prior to Trial 1, subjects were prescribed their normal diet, as previously determined from the 7-day dietary analysis. This ensured that they did not inadvertently modify their energy consumption. The subjects were divided into two matched groups, which were then randomly assigned to either a control (C) or a carbohydrate (CHO) condition. For the 22.5-hr recovery period, the subjects' food intake was prescribed to include additional energy. This was calculated on the basis of an increase in the carbohydrate component of each subject's habitual diet to 9.0 g kg^{-1} BW. The supplementary energy for the CHO group was provided in the form of a 16.5% glucose-polymer solution, whereas the isocaloric equivalent in the form of dietary fat and protein was prescribed for the C group. The relative additional amounts of fat and protein, which were part of the main meals, were determined in proportion to the subjects' normal levels of ingestion. Dietary prescriptions were prepared by a trained dietitian, with reference to each subject's habitual nutrient intake, and thus the foods prescribed were familiar and acceptable.

Preliminary Measurements

Following familiarization with running on a motorized treadmill, subjects were required to complete three preliminary tests. The first test determined the oxygen cost of running over a range of submaximal speeds. The speeds were selected with reference to each subject's running ability and ranged from 2.79 to $5.04 \text{ m}\cdot\text{s}^{-1}$, with increments of $0.45 \text{ m}\cdot\text{s}^{-1}$. This was a continuous test with subjects running for 4 min at four different speeds; expired air was collected over the last minute of each stage. The second preliminary test determined maximal oxygen uptake ($\text{VO}_{2\text{max}}$) during uphill treadmill running (36). The treadmill gradient was initially set to 3.5% and was subsequently increased by 2.5% every 3 min. Subjects ran at a constant submaximal speed throughout the test, and 1-min expired air collections were taken over the 3rd minute of each stage. Subjects aimed to run for as long as possible. A final expired air collection was taken when subjects felt they could only maintain the required exercise intensity for 1 more minute. From this collection $\text{VO}_{2\text{max}}$ was calculated.

The third preliminary test was a speed-lactate test. This determined the relationship between oxygen consumption and blood lactate concentrations over a range of submaximal running speeds. Four speeds were calculated from data obtained during the previous tests, such that the respective VO_2 demands were equivalent to 60 to 90% of $\text{VO}_{2\text{max}}$. Blood samples were taken during the final minute of each 4-min stage, along with the collections of expired air.

Experimental Design

The treadmill runs of Trial 1 (T1) and Trial 2 (T2) were performed in the mornings of 2 consecutive days (Figure 1). The subjects arrived at the laboratory after an overnight fast of 9 to 10 hours, which began at 10 p.m. the previous evening. On arrival, the subjects emptied their bladders before any preliminary measurements were made. They were weighed nude before and after each exercise bout. Immediately before T1 and T2, four ECG chest electrodes were taped to each subject for monitoring the heart rate during exercise.

After the subject had been seated in a relaxed position for approximately

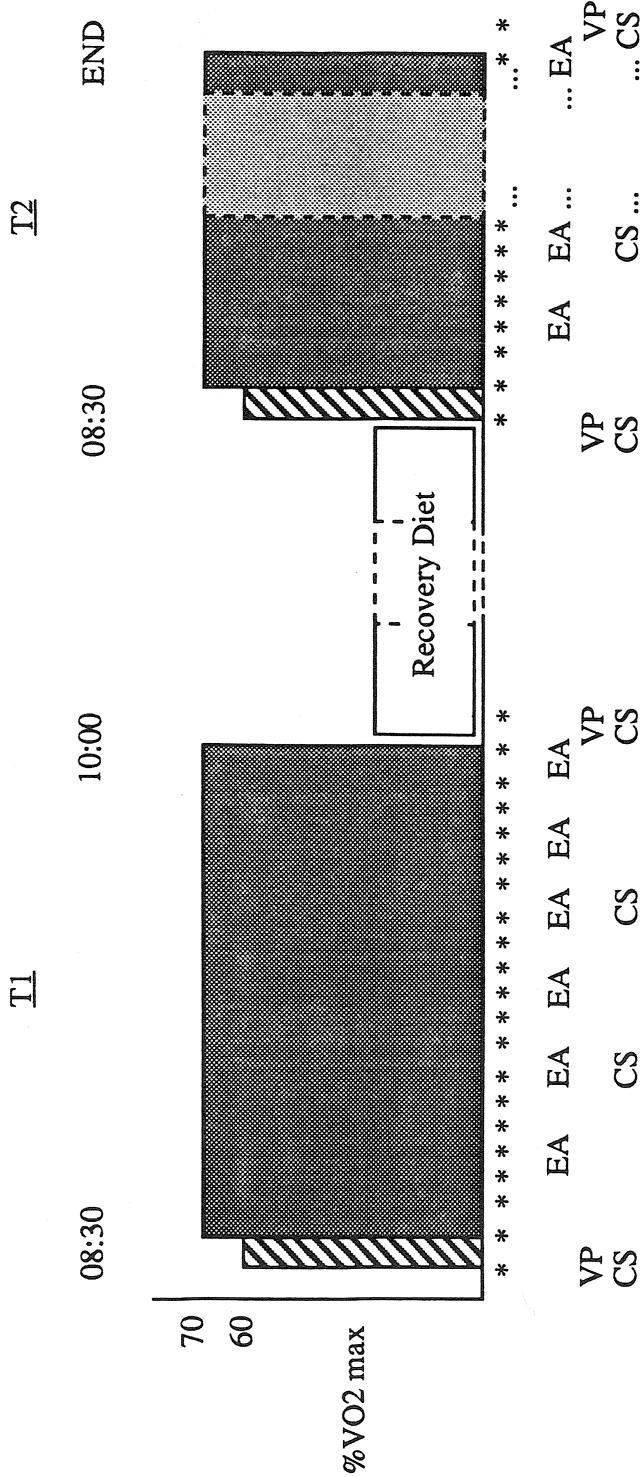


Figure 1 — Schematic representation of the experimental procedures. *Temperature; EA = expired air collection; CS = capillary sample; VP = venipuncture; \square = warm-up; ■ = exercise.

10 min, a 10-ml venous blood sample was taken from an antecubital vein in the forearm. Duplicate 20- μ l capillary blood samples were also obtained from the thumb of a prewarmed hand. Similarly, venous and capillary blood samples were obtained at the end of both trials T1 and T2. In addition, capillary blood samples were taken after 30 and 60 min during each trial.

The recovery period between T1 and T2 was 22.5 hours. A standardized 5-min warm-up, which consisted of steady running at a pace equivalent to 60% $\text{VO}_{2\text{max}}$, was performed prior to each trial. Immediately following the warm-up, the treadmill speed was increased to a pace equivalent to 70% $\text{VO}_{2\text{max}}$. The first trial was an endurance task in which subjects aimed to exercise for 90 min, or until volitional fatigue, whichever came first. Volitional fatigue was defined as the point at which subjects were unable to maintain the required running pace. In the second trial, subjects were required to run for as long as possible, such that endurance capacity could be measured in terms of exercise time to fatigue. In order that a subject might more accurately assess his state of fatigue during the latter stages of each trial, he had the option of reducing the running pace for a period of not more than 2 min on one occasion, after which the speed equivalent to 70% $\text{VO}_{2\text{max}}$ was resumed. When the subject reduced the treadmill speed a second time, the test was terminated.

Expired air samples were collected at 15-min intervals during T1 and T2, using the Douglas bag technique (37). Simultaneously, subjective ratings of perceived exertion were obtained using the Borg scale (9). From subsequent gas analyses, VE, VO_2 , and VCO_2 were determined and the respiratory exchange ratio (R) was calculated.

The laboratory was maintained at a temperature of 20°C. While running on the treadmill, subjects were cooled by electric fans and wet sponges were available for use ad libitum. Likewise, drinking water was freely available. The total fluid ingested was recorded at the end of each trial and was accounted for in each subject's postexercise change in body weight. The subjects maintained a constant level of training from the beginning of the preliminary tests until completion of T2. In addition, subjects refrained from strenuous activity over the 2 days preceding T1 and during the 22.5 hours of the recovery period.

Analyses

The capillary blood samples were deproteinized in 0.38 mmol·l⁻¹ perchloric acid prior to centrifugation (Eppendorf centrifuge 5414). They were stored at -20°C and later analyzed for glucose and lactate (27). The venous blood samples were dispensed into lithium heparin tubes. Hemoglobin concentration was determined of each sample using the cyanmethemoglobin method (Boehringer Mannheim GmbH Diagnostica), and packed cell volume was measured by microcentrifugation (Hawksley Ltd.). Changes in plasma volume were then estimated by the method of Dill and Costill (16). Plasma samples were obtained by centrifugation of the remaining whole blood for 20 min at 3°C (Burkard Koolspin). A 300- μ l aliquot of plasma was stored at -70°C and was analyzed for ammonia within 48 hours (Boehringer Mannheim GmbH Diagnostica). The remainder was stored at -20°C and later analyzed for FFA (11), glycerol (26), and urea (Boehringer Mannheim GmbH Diagnostica).

The performance times of the two groups were compared by analysis of

covariance. Differences in blood biochemical responses were examined using Student's independent *T* test, whereas differences within each group were examined using a paired *T* test. All other physiological responses were analyzed using two-way analysis of variance (ANOVA) for repeated measures. When differences were revealed using ANOVA, then a Tukey post hoc test was applied in order to identify the nature of such differences. Values at the 0.05 level were accepted as being statistically significant.

Results

The run times of the C and CHO groups during the two trials are given in Figure 2. The C group exercised 3.62 min longer than the CHO group during T1 (ns). The absolute differences in performance between T1 and T2 were (a) a reduction in run time of 15.57 min in the C group ($p<0.05$) and (b) an increase in run time of 9.21 min in the CHO group (ns). Thus the restoration of endurance capacity was more complete in the CHO group compared with the C group.

The energy intake of the groups over the 2 days prior to T1 are given in Table 2. Carbohydrates represented 51% ($\pm 3\%$) and 52% ($\pm 3\%$) of these values for the C and CHO groups, respectively. The energy content of the diets of both groups was increased during the recovery phase (Table 2). The carbohydrate content of the C recovery diet remained the same in absolute terms, but in relative

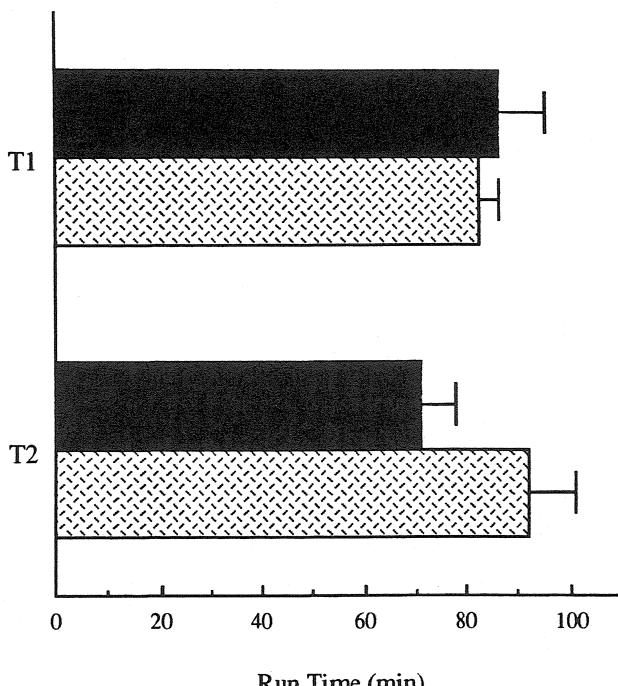


Figure 2 — Run times (min) for the control (solid bars) and carbohydrate (hatched bars) groups during T1 and T2 (mean \pm SE).

Table 2**The Energy Content and Main Dietary Nutrients for Normal and Recovery Diets of Subjects in the Control and Carbohydrate Groups**

Diet		Energy MJ	Protein g	Carbohydrate g	Carbohydrate g·kg ⁻¹ BW	Fat g
Normal	Control	13.4	119.0	431.0	6.1	120.9
	±SE	1.3	14.1	45.5	0.6	12.1
	CHO	13.0	113.0	422.0	5.8	116.8
	±SE	0.9	8.3	32.5	0.5	16.2
Recovery	Control	17.4	171.8	446.5	6.3	197.2
	±SE	0.9	14.9	50.0	0.7	15.1
	CHO	16.3	118.5*	631.8*	8.8*	113.3*
	±SE	0.9	12.1	25.8	0.1	15.7

*Denotes significantly different from the control group, $p<0.01$.

terms it represented a reduction to 36% ($\pm 6\%$) of the total energy intake. The carbohydrate content of the CHO recovery diet was increased such that it represented 63% ($\pm 3\%$) of the total energy intake. The eating patterns of the two groups over the recovery were analyzed by examining the dietary content of breakfast, lunch, dinner, and snacks. It was found that the eating patterns of the C and CHO groups, in terms of energy intake, were the same over the 22.5 hours. There was no difference in the carbohydrate content of the first main meal ingested immediately after T1.

The respiratory exchange ratio (R) did not differ between the groups over the two trials (Figure 3). The energy expenditures of the C group during T1 was estimated to be 5.7 MJ (1,364 kcal), of which fat contributed 48% and carbohydrate 52%. Thus, 173.1 (± 8.4) g of carbohydrate were oxidized during this first bout of exercise. During T2, an estimated 4.8 MJ (1,148 kcal) of energy were expended, of which fat contributed 42% and carbohydrate 58%. The energy expenditure of the CHO group during T1 was estimated to be 5.5 MJ (1,316 kcal), of which fat contributed 57% and carbohydrate 43%. As such, 140.2 (± 8.2) g of carbohydrate were oxidized during this first bout of exercise, whereas during T2 an estimated 6.2 MJ (1,483 kcal) of energy were expended, of which fat contributed 51% and carbohydrate 49%.

Blood glucose concentrations were maintained within the normal range in both groups over T1 and T2 (Figure 4). From the preliminary tests, the running speeds initially eliciting 70% of $\text{VO}_{2\text{max}}$ represented 106% of the $2.0 \text{ mmol}\cdot\text{l}^{-1}$ lactate reference speed in the C group and 110% in the CHO group. Following the onset of exercise, blood lactate concentrations in the C group increased from $0.78 (\pm 0.08) \text{ mmol}\cdot\text{l}^{-1}$ to $5.12 (\pm 0.72) \text{ mmol}\cdot\text{l}^{-1}$ at the end of T1, and from $0.79 (\pm 0.09) \text{ mmol}\cdot\text{l}^{-1}$ to $4.60 (\pm 0.59) \text{ mmol}\cdot\text{l}^{-1}$ at the end of T2. In the CHO group the values increased from $0.96 (\pm 0.11) \text{ mmol}\cdot\text{l}^{-1}$ to $4.51 (\pm 0.60) \text{ mmol}\cdot\text{l}^{-1}$, and from $1.04 (\pm 0.10) \text{ mmol}\cdot\text{l}^{-1}$ to $4.75 (\pm 0.73) \text{ mmol}\cdot\text{l}^{-1}$, during T1 and T2, respectively.

During T1, plasma glycerol (Figure 5) increased sevenfold in the C group

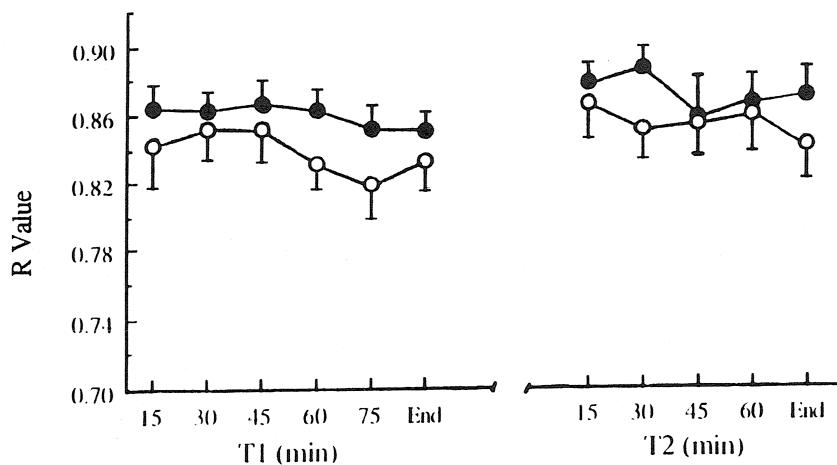


Figure 3 — R values for the control (closed symbols) and carbohydrate (open symbols) groups during T1 and T2 (mean \pm SE).

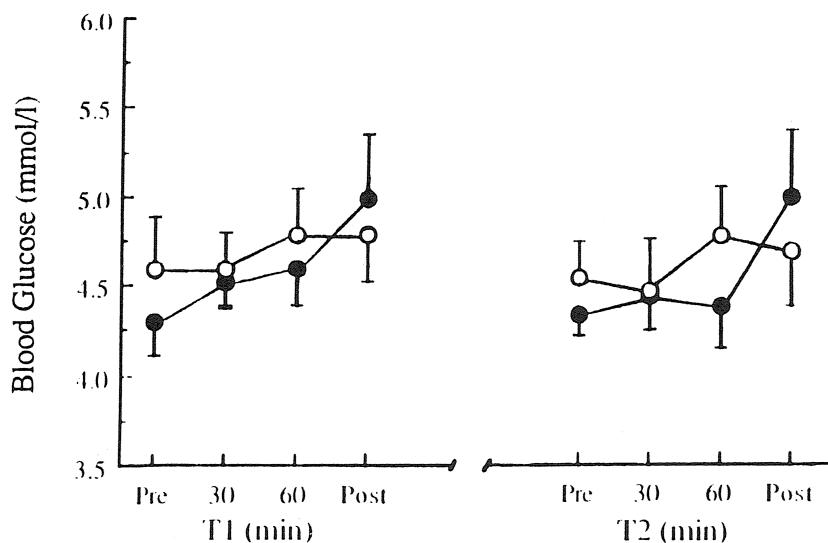


Figure 4 — Blood glucose ($\text{mmol}\cdot\text{l}^{-1}$) for the control (closed symbols) and carbohydrate (open symbols) groups during T1 and T2 (mean \pm SE).

and eightfold in the CHO group ($p<0.01$), whereas FFA (Figure 5) increased twofold in both groups ($p<0.01$). During T2, plasma glycerol increased fourfold in the C group and sevenfold in the CHO group ($p<0.01$). Once again, plasma FFA approximately doubled. However, the change in FFA concentrations of the CHO group ($p<0.01$) was greater than that of the C group ($p<0.05$).

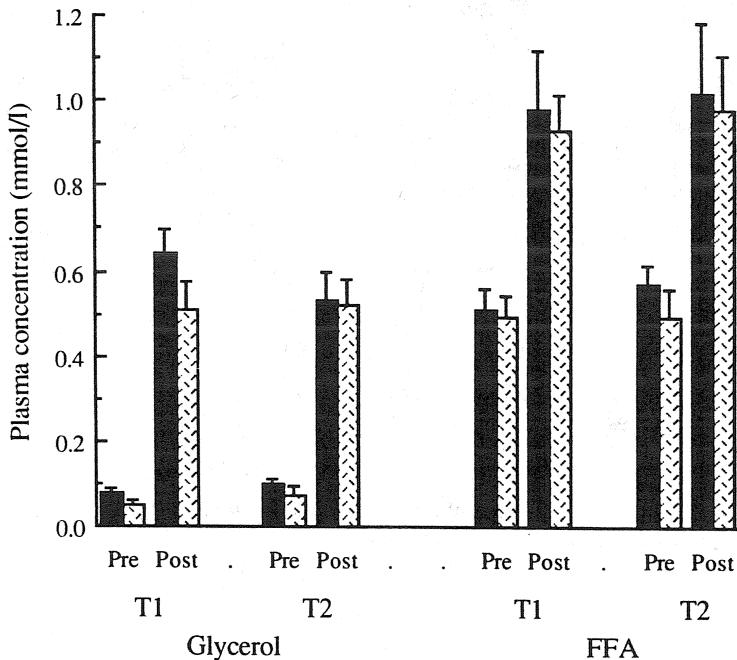


Figure 5 — Plasma glycerol and plasma FFA concentrations ($\text{mmol}\cdot\text{l}^{-1}$) for the control (solid bars) and carbohydrate (hatched bars) groups over T1 and T2 (mean $\pm\text{SE}$).

Plasma ammonia increased in both groups during T1 and T2 ($p<0.01$). There were no differences between the groups, with concentrations ranging from $39.02 \mu\text{mol}\cdot\text{l}^{-1}$ to $54.12 \mu\text{mol}\cdot\text{l}^{-1}$ at rest and rising to between $92.23 \mu\text{mol}\cdot\text{l}^{-1}$ and $110.96 \mu\text{mol}\cdot\text{l}^{-1}$ postexercise. Plasma urea increased in both the C ($p<0.05$) and CHO ($p<0.01$) groups during T1 (Figure 6); though consistent with values for plasma ammonia, there were no differences between the groups. Pre-T1 values were restored prior to T2 in the CHO group, but this was not the case in the C group ($p<0.05$). During the second trial, concentrations remained stable in the C group but increased in the CHO group ($p<0.01$), such that plasma urea was higher in the CHO group post-T2 ($p<0.05$).

The C group had a reduction in body weight of 3.1% during T1 and of 2.3% during T2. These values take into account fluid ingestions of $248 (\pm 59)$ ml in the case of the former, and $220 (\pm 63)$ ml in the case of the latter. In the CHO group, body weight was reduced by 2.7% during T1 and 3.2% during T2. These values take into account fluid ingestions of $385 (\pm 108)$ ml and $447 (\pm 114)$ ml for the two trials, respectively. During the 22.5 hours of the recovery period, pre-T1 body weights were restored in both groups. Plasma volume decreased by 5.7% ($\pm 1.7\%$) over T1 and by 7.1% ($\pm 1.2\%$) over T2 in the C group, and by 7.5% ($\pm 1.6\%$) and 10.8% ($\pm 1.7\%$) over the two trials, respectively, in the CHO group. Heart rates increased during T1 and T2, but there were no differences between the values of the two groups.

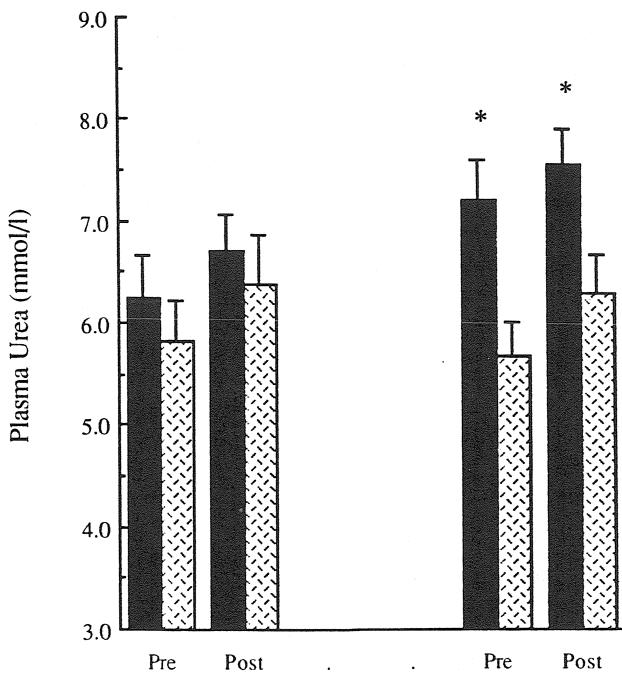


Figure 6 — Plasma urea concentrations ($\text{mmol} \cdot \text{l}^{-1}$) for the control (solid bars) and carbohydrate (hatched bars) groups over T1 and T2 (mean $\pm \text{SE}$). *Denotes control group significantly different from carbohydrate group, $p < 0.05$.

Discussion

The main finding of the present study was that a normal diet supplemented with additional carbohydrate restored endurance capacity following a recovery of 22.5 hours. However, an isocaloric diet in which additional energy was provided as fat and protein did not bring about the same return in exercise capacity.

The groups were well matched in terms of their physiological characteristics (age, height, weight), as well as in terms of their respiratory and cardiovascular responses (V_{Emax} , HR_{max} , $\text{VO}_{2\text{max}}$) (Table 1), running economy, and blood lactate responses to submaximal exercise (Table 3). This was also reflected in their similar run times for T1. In contrast, during T2 the performance of the C group was reduced whereas the subjects in the CHO group were able to match their T1 performance.

This result contrasts with the findings of Keizer et al. (24), who reported that when subjects consumed a prescribed carbohydrate-rich diet after exhaustive exercise, they were unable to reproduce the same physical performance as before the exhaustive exercise bout. Despite restoring muscle glycogen concentrations to resting values 22 hours after intermittent cycle ergometer exercise, MPWC was reduced by 7.3% (24). However, MPWC is more a measure of the maximal

Table 3**Running Speeds and Relative Exercise Intensities at Two Blood Lactate Concentrations and Running Economy for Subjects in Both Groups**

Group	Speed (m·s ⁻¹)		Intensity (% $\dot{V}\text{O}_{\text{max}}$)		Running economy $\dot{V}\text{O}_2$ at 3.8 m·s ⁻¹ (ml·kg ⁻¹ ·min ⁻¹)
	2 mmol·l ⁻¹	4 mmol·l ⁻¹	2 mmol·l ⁻¹	4 mmol·l ⁻¹	
Control	3.44	4.51	66.1	86.0	44.8
$\pm SE$	0.53	0.40	4.3	6.2	1.0
CHO	3.34	4.51	63.7	86.3	43.6
$\pm SE$	0.42	0.21	1.2	2.8	0.4

power generating capacity of muscle than of endurance capacity, as reported in the present study. The ability to generate power is largely determined by the degree of muscle fiber recruitment. The exhaustive cycling exercise may have evoked an inflammatory response (31), which in turn could interfere with the propagation of stimulatory action potentials. As a consequence, the number of muscle fibers recruited may be reduced, resulting in the measurably impaired MPWC. In addition, the biopsies taken during the experimental day and immediately prior to the second MPWC test may have exacerbated the situation, compounding the muscle damage of the prolonged exercise bout.

In the present study, the postrecovery trial (T2) was a submaximal test of uniform intensity whereas the MPWC test was incremental, with the exercise intensity being increased to maximal over time. In addition to the different neural demands of the two activities, they will also differ in the nature of their respective energy demands. The MPWC test will have a substantial contribution from anaerobic metabolism in order to sustain an increasing rate of energy provision. This in itself will limit performance, with the resultant accumulation of metabolic by-products interfering with anaerobiosis and leading to fatigue earlier. However, the submaximal exercise task of the present study, with its lower rate of energy demand, can be sustained over a longer period of time primarily through aerobic metabolism. Thus the MPWC test is more localized in its demands in comparison with the whole-body activity of T2.

The total carbohydrate consumption of the C group over the recovery period was 446.5 g (6.3 g kg⁻¹ BW). This is higher than values for the general population, but it is similar to their usual level of intake and consistent with values reported for endurance runners (12, 32). Nevertheless, this appears to be insufficient to restore endurance capacity within 22.5 hours. The carbohydrate consumption of the CHO group over the recovery period was 631.8 g (8.8 g kg⁻¹ BW), in agreement with the dietary recommendations prescribed by others for muscle glycogen replenishment (14, 17, 35). Both recovery diets contained more carbohydrate than was estimated to have been oxidized during T1. For the C group this was equivalent to a normal level of ingestion. The recovery diet of the CHO group contained additional carbohydrate, such that both the daily requirements of the body and the energy demands of T1 were taken into account.

Although the carbohydrate supplementation in the present study restored endurance capacity, Kirwan et al. (25) reported that an intake as high as 8.0 g kg⁻¹ BW·day⁻¹ did not prevent a cumulative glycogen depletion in subjects running 20 km at 80% VO_{2max} on 5 successive days. This suggests that an even higher carbohydrate intake is necessary for prolonged periods of heavy training. Alternatively, there may be a limitation in the ability to store the ingested fuel, perhaps playing some role in a physiological safety mechanism (25). Thus the upper limit of "useful" carbohydrate ingestion will depend upon its subsequent rate of incorporation into muscle tissue as glycogen. This rate is estimated to range between 5.0 and 10.0 mmol·kg⁻¹ h⁻¹ wet wt under normal conditions, though higher values have been reported (30). Carbohydrate ingested in excess of the body's requirements is redirected toward incorporation into adipose tissue (7). As such, there is an optimal level of daily carbohydrate ingestion that is consonant with both its level of utilization and a maximal rate of glycogenesis.

The difference between the C and CHO groups in estimated amounts of carbohydrate metabolized during T1 cannot be satisfactorily explained from the available data. The running economy and blood lactate responses of the two groups were indicative of similar levels of physiological efficiency (Table 3). One possibility is that the CHO group had a lower oxidation rate (1.70 ± 0.10 g CHO·min⁻¹) in comparison with the C group (2.01 ± 0.01 g CHO·min⁻¹). This may have been further compounded by the marginally longer, but not significantly longer, run time of the C group during T1. Newsholme (29) estimated total body carbohydrate reserves to be 440 g, with 350 g stored in muscle tissue and 90 g stored in the liver. Assuming that 60% of the total muscle mass is active during running, the carbohydrate available to fuel exercise will be of the order of 300 g (10). If all the carbohydrate oxidized were provided by muscle glycogen, then this energy store would be reduced by 58% over T1 in the C group and by 47% in the CHO group.

These values are in agreement with Costill et al. (14) for running exercise of a similar intensity and duration. Consistent with this, blood glucose was maintained within the normal range in the two groups over both T1 and T2. This suggests that hypoglycemia per se was not a cause of fatigue.

The importance of consuming carbohydrate immediately after exercise in order to achieve a rapid glycogen resynthesis has already been demonstrated (21). In the present study, the first meal of the day was provided to both groups on completion of T1 and contained foods high in sugar. This ensured a rapid physiological availability of the ingested carbohydrate (15, 35). The absolute carbohydrate content of the meals was 102.1 (± 15.0) g and 82.7 (± 9.6) g for the C and CHO groups, respectively; this difference was not significant. The remainder of the prescribed diet was taken ad libitum, such that the precise timing of ingestion was noted but not controlled. An optimal rate of muscle glycogen resynthesis can be achieved over the initial 4 to 5 hrs postexercise, by ingesting 1.0 g CHO·kg⁻¹ BW·2 h⁻¹ (8, 22). This is equivalent to approximately 37.0 g CHO·h⁻¹. Thus, the first meal taken following T1 provided more than the minimum amount of carbohydrate recommended for optimal glycogenesis over the early phase of recovery.

The eccentric component of running (i.e., the forced lengthening of exercising muscle as external forces are applied) is associated with muscle soreness (28) and ultrastructural changes indicative of intracellular lesions (19, 31). Such

localized tissue damage has been found to impair muscle glycogen replenishment (13). This was speculated to be due to an infiltration of the traumatized muscle by inflammatory cells (13). As well as being oxidatively active, these cells also release a factor that stimulates a heightened level of carbohydrate metabolism in surrounding tissue. As such, competition for the available glucose develops between the inflammatory cells and the depleted muscle fibers.

Costill et al. (13) observed that a normal level of carbohydrate ingestion ($4.3 \text{ g kg}^{-1} \text{ BW-day}^{-1}$) was inadequate under these conditions for optimal muscle glycogen resynthesis, and suggested that this situation may be appeased with a high carbohydrate diet ($8.5 \text{ g kg}^{-1} \text{ BW-day}^{-1}$). In the present study, subjects in both the C and CHO groups reported joint stiffness and muscle soreness 22.5 hours after T1, which may be indicative of subclinical microtrauma. It would be inappropriate to speculate here on the respective levels of glycogen resynthesis in the two groups following the recovery period. However, it might be argued in light of Costill et al.'s findings (13) that the additional carbohydrate provided in the CHO recovery diet would facilitate the recovery process.

Both plasma ammonia and plasma urea concentrations remained elevated in the C group prior to T2, plasma urea having continued to increase over the 22.5 hours. This was most likely due to additional protein in the recovery diet. Elevated resting concentrations of these potential toxins before T2 may have contributed to the poorer performance of the C group. In the CHO group, plasma ammonia and urea concentrations were restored to pre-T1 values before the start of T2.

Redressing the energy imbalance is not the only concern during recovery; restoring the body's fluid balance is also of paramount importance (3). During T1 and T2, both groups had decreases in body weight that reflected fluid loss. These decrements were in excess of the 2.0% threshold, beyond which exercise performance is impaired (2). In the C group, the fall in plasma volume was greater over T2 than over T1, despite a shorter run time. This fluid loss may have adversely affected the thermoregulatory mechanisms and hastened the onset of fatigue.

In summary, increasing the carbohydrate content of a normal diet to a value of at least $8.8 \text{ g kg}^{-1} \text{ BW-day}^{-1}$ restores endurance capacity within 22.5 hours. An enhanced dietary carbohydrate intake was associated with a reduced accumulation of potentially toxic metabolic waste products, while additional substrate was provided to facilitate muscle glycogen resynthesis when localized tissue damage may have prevailed. However, any consideration of the recovery process should not focus exclusively upon the body's energy status. The restoration of fluid balance is also important (3), as this influences the physiological milieu in which metabolism occurs.

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