Metabolism and performance following carbohydrate ingestion late in exercise

ANDREW R. COGGAN and EDWARD F. COYLE

Human Performance Laboratory, Department of Kinesiology and Health Education, The University of Texas, Austin, TX 78712

ABSTRACT

COGGAN, A. R. and E. F. COYLE. Metabolism and performance following carbohydrate ingestion late in exercise. Med. Sci. Sports Exerc., Vol. 21, No. 1, pp. 59–65, 1989. To determine whether a single carbohydrate feeding could rapidly restore and maintain plasma glucose availability late in exercise, six trained cyclists were studied on two occasions during exercise to fatigue at 70 ± 1% of \( \dot{VO}_{2max} \). After 135 min of exercise, the men were fed either an artificially sweetened placebo or glucose polymers (3 g·kg⁻¹ in a 50% solution). Prolonged exercise led to a decline in plasma glucose from 4.6 ± 0.1 mM at rest to 3.9 ± 0.2 mM after 135 min (\( P < 0.05 \)). Plasma glucose decreased further (\( P < 0.05 \)) to 3.2 ± 2.0 mM at fatigue following placebo ingestion but increased (\( P < 0.05 \)) and was then maintained at 4.5–4.9 mM following carbohydrate ingestion. Respiratory exchange ratio (R) fell gradually during the placebo trial from 0.88 ± 0.01 after 10 min of exercise to 0.81 ± 0.01 at fatigue (\( P < 0.01 \)). R also reached a minimum of 0.81–0.82 in each subject after 135–180 min of exercise during the carbohydrate feeding trial but increased again to 0.84–0.86 as plasma glucose rose following the carbohydrate feeding. Exercise time to fatigue was 21% longer (205 ± 17 vs 169 ± 12 min; \( P < 0.01 \)) during the carbohydrate ingestion trial. Plasma insulin did not increase significantly, whereas plasma free fatty acids and blood glycerol plateaued following carbohydrate ingestion. These data indicate that a single carbohydrate feeding late in exercise can supply sufficient carbohydrate to restore euglycemia and increase carbohydrate oxidation, thereby delaying fatigue.

ENDURANCE, HYPOGLYCEMIA, RESPIRATORY EXCHANGE RATIO

Both muscle glycogen and plasma glucose are oxidized by skeletal muscle to supply energy during prolonged exercise (1–4,19,22,31,32). Although the underlying mechanisms are uncertain, there appears to be a gradual shift from intramuscular glycogen toward blood-borne glucose as the predominant carbohydrate energy source as exercise proceeds and as muscle glycogen is depleted (8,12,19,25,31,32). The contribution of glucose to oxidative metabolism may be limited, however, by a decline in plasma glucose concentration late in exercise (1,2,8,12). This was demonstrated in a recent study in which it was possible to increase carbohydrate oxidation and reverse fatigue following pro-
longed exercise at 70% of maximal oxygen consumption (\( \dot{VO}_{2max} \)) by infusing glucose intravenously (8). The maintenance of euglycemia during an additional ~45 min of exercise required the infusion of glucose at ~1.1 g·min⁻¹, which could have supplied three fourths of total carbohydrate oxidation during this time (8).

Oral ingestion of glucose polymers at fatigue also initially increased plasma glucose levels and respiratory exchange ratio (R) in these subjects (8). Plasma glucose and R steadily declined as exercise proceeded, however, with fatigue again rapidly ensuing (8). Tabata, Atomi, and Miyashita (30) have also observed declining blood glucose concentrations when further exercise is performed following carbohydrate ingestion at fatigue. These results suggest that the ingestion of carbohydrate solutions after fatigue has already occurred cannot supply glucose into the blood at a sufficient rate to meet the demands of the exercising musculature.

In this context, the purpose of the present investigation was to determine whether a single carbohydrate feeding late in exercise, but before the actual point of fatigue, could supply glucose into the blood rapidly enough to reverse the decline in plasma glucose concentration and in carbohydrate oxidation and thereby delay fatigue. Based on gastric emptying studies (9,15,16,23), we reasoned that ~30 min should allow sufficient time for significant quantities of carbohydrate to be absorbed. Fatigue in trained cyclists during exercise at 70–75% of \( \dot{VO}_{2max} \) was anticipated to occur in ~150–180 min in the absence of carbohydrate supplementation (8,12); therefore, the carbohydrate feeding was supplied after 135 min of exercise.

METHODS

Subjects. Six well-trained male cyclists participated in this study after giving their written, informed consent. All subjects were familiar with general laboratory procedures and with prolonged cycle ergometry, having

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served previously as subjects in similar studies from this laboratory (8,12). Mean (±SE) age, weight, and \( \dot{V}O_2\) were 24 ± 1 yr, 70.3 ± 3.0 kg, and 4.59 ± 0.20 l.min\(^{-1}\), respectively. The study protocol was approved by the Human Studies Committee of the University of Texas.

**Experimental design.** Each subject completed two experimental trials, 1–3 wk apart, which consisted of exercise to fatigue at an intensity just below the individual’s blood lactate threshold (11). \( \dot{V}O_2\) measured periodically during these exercise bouts averaged \( 70 ± 1\% \) (range 66–73\%) of the subjects’ \( \dot{V}O_2\)\(_{max}\). During one trial, the men ingested 3 g of carbohydrate (85\%) glucose polymers–15\% sucrose; Exceed, Ross Laboratories, Columbus, OH) per kg of body weight in a lemon-flavored 50\% solution after 135 min of exercise (105 min in one subject; see “Results”). During the other trial, they received an equal volume of a lemon-flavored placebo drink which was artificially sweetened with aspartame and thickened with fruit pectin. The order of presentation was counterbalanced for the two drinks. The volume, composition, and concentration of the carbohydrate drink were chosen in an attempt to minimize emptying of carbohydrate from the stomach (15,16,23).

Exercise was performed using an electrically braked Quinton 870 cycle ergometer, equipped with toe clips and straps. Power output on this ergometer is constant and independent of pedaling rate between 40 and 100 rpm. Subjects were instructed to pedal at 85–95 rpm, with fatigue defined as a decline in pedaling rate below 50 rpm. One investigator was responsible for motivating the subjects during all trials. Subjects were kept unaware of their exercise duration until the end of the study.

The subjects were instructed to maintain a constant training regimen during the study. Subjects refrained from exercise and consumed a constant diet for 48 h prior to all testing. Experiments were conducted in the morning following a 12–14 h fast. The laboratory was maintained at 20–22°C, and the subjects were cooled with fans during exercise. The men were supplied with 250 ml of cold (4°C) water after 15 min of exercise and every 30 min thereafter, except that no additional fluid was ingested at the time of the placebo or carbohydrate feeding (i.e., at 135 min).

**Sample collection and analysis.** Subjective ratings of perceived exertion (6) were obtained after 10 and 30 min of exercise, every 30 min thereafter (every 15 min from 120 to 180 min), and at the point of fatigue. \( \dot{V}O_2\) and \( R \) were also measured over a 5-min interval at these time points using an open-circuit system (Rayfield Equipment, Chicago), which incorporated a mixing chamber, electronic \( O_2 \) (Applied Electrochemistry S3-A) and \( CO_2 \) (Beckman LB-2) gas analyzers, and a dry gas meter (Parkinson-Cowan CD-4). Outputs from these instruments were directed to a laboratory computer, which calculated \( \dot{V}O_2\) and \( R \) every 30 s. The dry gas meter was calibrated periodically using a Tissot spirometer, and the gas analyzers were calibrated frequently using gases verified by chemical analysis.

Approximately 7 ml of blood were drawn at rest, every 30 min during exercise (every 15 min from 120 to 180 min), and at the point of fatigue from a flexible catheter inserted in a forearm vein and kept patent by flushing periodically with sterile saline. Three milliliters of blood were anticoagulated with EDTA (1.2 mg/ml\(^{-1}\)), plasma separated by centrifugation at 4°C, and stored at –80°C until subsequent analysis for glucose (Yellow Springs Instruments Model 23a glucose analyzer) and free fatty acids (FFA) (27). An additional 3 ml of blood were anticoagulated, preserved with aprotonin (0.5 trypsin inhibitory units·ml\(^{-1}\)), and plasma stored at –80°C for later measurement of insulin (18) using a commercial kit (Radioassay Systems Laboratories, Carson CA). One milliliter of blood was deproteinized in 0.8 M perchloric acid and stored at –80°C until used for the measurement of lactate (20) and glycerol (13).

**Statistical analyses.** Exercise time to fatigue was compared between treatments using a paired \( t \)-test. Ratings of perceived exertion, \( R \), and blood metabolite data were analyzed using repeated-measures ANOVA. Significant differences identified by ANOVA were isolated using Newman-Keuls post hoc tests. The significance level for all comparisons was set at \( P < 0.05 \).

**RESULTS**

**Plasma glucose and \( R \).** Plasma glucose concentration increased slightly during the first hour of exercise in both trials but began to decline gradually after this time, falling \( (P < 0.05) \) to just below 4 mM immediately before placebo or carbohydrate ingestion (Fig. 1). Plasma glucose decreased further \( (P < 0.05) \) following ingestion of the placebo drink, averaging 3.2 ± 0.2 mM when the subjects were unable to continue exercise. Ingestion of the carbohydrate drink, in contrast, resulted in a sharp increase \( (P < 0.05) \) in plasma glucose to 4.7 ± 0.4 mM within 15 min following the feeding (Fig. 1). Plasma glucose was then maintained at 4.6–4.9 mM throughout the remainder of the carbohydrate feeding trial (Fig. 1).

\( R \) averaged 0.88 ± 0.01 after 10 min of exercise during the placebo trial, but fell steadily to 0.83 ± 0.01 after 135 min of exercise \( (P < 0.05) \) and declined further \( (P < 0.05) \) to 0.81 ± 0.01 at fatigue (Fig. 1). \( R \) values initially fell similarly during the carbohydrate ingestion trial, reaching a minimum of 0.81–0.82 in each subject after 135–180 min of exercise. \( R \) then increased to 0.84–0.86 as plasma glucose concentrations rose following carbohydrate ingestion. This increase, however, ap-
peared to lag 15–30 min behind the increase in plasma glucose and occurred at different times in individual subjects. Mean R thus plateaued at 0.84 ± 0.01 following carbohydrate ingestion (Fig. 1).

**VO₂, total carbohydrate oxidation, and ratings of perceived exertion.** Since VO₂ was constant and did not differ between treatments (Table 1), total carbohydrate oxidation estimated from VO₂ and R paralleled these changes in R. Total carbohydrate oxidation during the placebo trial thus fell gradually from 2.3 ± 0.2 g·min⁻¹ after 10 min of exercise to 1.4 ± 0.1 g·min⁻¹ at fatigue (Table 1). In contrast, total carbohydrate oxidation plateaued at ~1.7 g·min⁻¹ following carbohydrate ingestion (Table 1).

Ratings of perceived exertion rose gradually during the placebo trial from 11.7 ± 1.0 after 10 min of exercise to 17.8 ± 0.7 at the point of fatigue (P < 0.01) (Table 1). Perceived exertion increased similarly during the carbohydrate ingestion trial, with no significant effect of the carbohydrate feeding (Table 1).

**Exercise performance.** Exercise time to fatigue averaged 21% longer (205 ± 17 vs 169 ± 12 min; P < 0.01; Table 2) when declines in plasma glucose and R were reversed by carbohydrate ingestion. All of the subjects except one were able to exercise longer when fed carbohydrate, with fatigue delayed by 41–52 min (Table 2). In the sixth subject, plasma glucose and R declined after 105 min of exercise during the carbohydrate ingestion trial to levels very similar to those observed at fatigue (163 min) during his placebo trial. As fatigue seemed imminent, the carbohydrate drink was supplied after 105 min of exercise. Plasma glucose and R subsequently rose, and the individual was able...
to continue exercise for an additional 45 min (i.e., 150 min total; Table 2).

**Plasma FFA and blood glycerol.** Plasma FFA and blood glycerol increased steadily during exercise in the placebo trial, reaching values 4–5-fold and 7–8-fold above basal at the point of fatigue (Fig. 2). During the carbohydrate feeding trial, plasma FFA and blood glycerol also increased progressively during the first 135 min of exercise but then remained constant following the ingestion of the carbohydrate drink (Fig. 2). Plasma FFA and blood glycerol were therefore significantly lower during the carbohydrate feeding trial versus the placebo trial at the time of fatigue.

**Plasma insulin and blood lactate.** Plasma insulin averaged 11 ± 1 μU·ml⁻¹ at rest and decreased slightly (NS) to 9 ± 1 μU·ml⁻¹ after 135 min of exercise (Table 3). Plasma insulin continued to decline slightly following placebo ingestion, averaging 7 μU·ml⁻¹ at fatigue (P < 0.01 vs pre-exercise). Circulating insulin concentrations did not increase significantly following carbohydrate ingestion (Table 3).

Exercise at 70 ± 1% of VO₂max resulted in a small but significant increase in lactate from 0.9 mM at rest to 1.4–1.9 mM during exercise, with no difference between treatments (Table 3).

**DISCUSSION**

We have recently reported that increasing plasma glucose availability via intravenous glucose infusion following fatiguing exercise at 70% of VO₂max can increase carbohydrate oxidation and reverse fatigue (8). The maintenance of euglycemia during an additional ~45 min of exercise required the infusion of glucose at ~1.1 g·min⁻¹, which could have supplied three fourths of total carbohydrate oxidation during this time (8). The present results indicate that a single carbohydrate feeding late in exercise can also supply glucose into the circulation at a rate sufficient to restore and maintain plasma glucose levels during continued exercise. In keeping with our previous observations (8,12), this increase in plasma glucose availability was associated with an increase in carbohydrate oxidation and with the postponement of fatigue.

A number of previous studies have observed that exercise performance is improved when supplemental carbohydrate is supplied during prolonged exercise (5,7,8,10,12,21,24,33). This improvement in performance has been variously ascribed to an alteration in the rate of muscle glycogen depletion (5,10,21,24) or to relief of CNS distress due to hypoglycemia (7,28,33). Alternatively, we have hypothesized (8,12) that plasma glucose is oxidized at high rates late in exercise and that carbohydrate feedings during exercise delay fatigue by maintaining the availability of this carbohydrate source when liver and muscle glycogen levels are low and the uptake of glucose by skeletal muscle is increased (19). The present results are consistent with this last hypothesis.

Theoretically, a slowing in the rate of glycogen utilization might explain the postponement of fatigue observed during the carbohydrate feeding trial. We have previously reported, however, that glucose polymer ingestion every 20 min during 3–4 h of exercise at 70% of VO₂max has no effect on the rate of decline in muscle glycogen (12). Furthermore, after 2–3 h of exercise by trained cyclists at 70% of VO₂max, only ~30% of initial glycogen stores remain (8,12). A “sparing” of muscle glycogen thus seems improbable in the present study, in which a single carbohydrate feeding was ingested after 135 min of exercise.

![Figure 2—Blood glycerol and plasma free fatty acid (FFA) concentrations during prolonged exercise when subjects were fed a placebo (open circles) or glucose polymers (3 g·kg⁻¹ body weight) (closed circles) after 135 min of exercise (arrow). Values are means ± SE for six subjects. *Significant difference (P < 0.05) between placebo and carbohydrate feeding trial.](image-url)
Prevention or relief of CNS distress due to the low plasma glucose concentration could also potentially have contributed to the ability of the subjects to sustain exercise longer following the carbohydrate feeding. Indeed, Christensen and Hansen (7) reported almost 50 yr ago that, in two subjects fed a high fat diet, the ingestion of 200 g or glucose after 130–160 min of moderate intensity cycling allowed the men to continue exercise for an additional hour, apparently by relieving symptoms of neuroglucopenia. Although three of the present subjects did in fact report mild CNS symptoms sometimes associated with hypoglycemia (i.e., headache, lightheadedness, nausea) during their placebo trial, these symptoms alone did not appear severe enough to explain the subjects’ eventual inability to continue exercise at 70% of VO_{max}. In addition, ratings of perceived exertion in these three individuals during their placebo trial were not different from their carbohydrate feeding trial or from the subjects who did not report such symptoms. It therefore seems unlikely that the subjects’ enhanced exercise capacity was solely due to relief of CNS distress.

Instead, carbohydrate feeding late in exercise appeared to delay fatigue primarily by maintaining plasma glucose concentration and carbohydrate oxidation during the final stages of the exercise bout (Fig. 1; Table 1). As observed previously (8,12), fatigue during prolonged exercise in the absence of carbohydrate supplementation was accompanied by a decrease in plasma glucose to <3.5 mM and a decline in R to ~0.8 (Fig. 1), suggestive of carbohydrate depletion. In contrast, the ingestion of ~200 g of carbohydrate after 135 min of exercise increased plasma glucose to 4.6–4.9 mM and maintained R values at 0.84 during the final ~1 h of exercise (Fig. 1), enabling the subjects to exercise 36 min longer than when fed the placebo (Table 2). These results support the hypothesis that the carbohydrate feeding delayed fatigue primarily by increasing carbohydrate oxidation by the exercising musculature. The present findings also agree well with our previous observations that the restoration of euglycemia by intravenous glucose infusion at ~1.1 g·min^{-1} can also increase carbohydrate oxidation and enhance performance in trained cyclists during prolonged exercise at 70% of VO_{max} (8).

Notably, the increase in carbohydrate oxidation (Table 1) following the carbohydrate feeding occurred in the absence of a significant increase in plasma insulin (Table 3) or a significant decrease in plasma free fatty acids (Fig. 2). This suggests that the ability of the carbohydrate feeding to maintain carbohydrate oxidation late in exercise and thereby delay fatigue was mediated primarily by changes in the availability of plasma glucose, rather than by associated changes in other substrates or in the hormonal milieu. This may explain the failure of carbohydrate feeding throughout exercise to significantly delay fatigue in those individuals who do not demonstrate a decline in plasma glucose in the absence of such carbohydrate supplementation (5,10).

Interestingly, the extent to which fatigue was delayed in the present study (i.e., by 30–60 min) by carbohydrate ingestion late in exercise is similar to that observed in previous studies in which carbohydrate was ingested throughout the duration of the exercise bout (5,10,12). This suggests that there may be no practical benefit to be gained by the ingestion of carbohydrate supplements throughout exercise. Rather, what appears to be critical is the ability of such carbohydrate supplements to supply glucose into the blood at sufficiently high rates late in exercise and thereby prevent a decline in plasma glucose concentration and R. We have previously reported that the rate of glucose infusion necessary to maintain euglycemia during the final stages of exercise can exceed 1 g·min^{-1} (8). The fact that euglycemia was restored and maintained in the present study when carbohydrate was ingested after 135 min of exercise implies that the carbohydrate feeding was able to match this rate of glucose delivery. Studies of gastric emptying (16,23) and absorption (29) of oral glucose loads indicate that this high rate is possible when the concentration of carbohydrate in the ingested drink is very high. Nevertheless, both we (8) and others (30) have previously observed that blood or plasma glucose concentrations are not maintained during further exercise when carbohydrate is ingested at fatigue, indicating that, under these conditions, the rate of entry of glucose into the bloodstream is unable to keep pace with its rate of removal by the exercising musculature. In contrast, the ingestion of a concentrated carbohydrate solution 30 min before the anticipated time of fatigue in the present study was able to restore euglycemia within
15 min following ingestion and to maintain normal plasma glucose concentrations during an additional ~1 h of exercise (Fig. 1). The underlying cause of this differing response to carbohydrate ingestion during the latter stages of exercise is not readily apparent. It is tempting to speculate that the stress-hormone response to the decline in plasma glucose concentration (17,30) may limit gastric emptying and/or intestinal absorption when carbohydrate ingestion is delayed until fatigue. Gastric emptying is enhanced, however, not impaired, during insulin-induced hypoglycemia in rats (26), and gastrointestinal motility is increased under similar conditions in man (14), which would argue against this possibility. In any case, on a practical basis it appears necessary to begin carbohydrate supplementation some time before the anticipated time of fatigue, in order to ensure that adequate carbohydrate availability is maintained during the final stages of exercise. Based on the present results, the ingestion of carbohydrate ~30 min before the anticipated time of fatigue appears to allow sufficient time for this to occur.

To conclude, the present results indicate that a single concentrated carbohydrate feeding after 135 min of cycling at 70% of VO_{2max} can supply sufficient carbohydrate to restore and maintain euglycemia during continued exercise. This increase in plasma glucose was associated with a significant increase in R, without any change in plasma insulin or FFA concentrations. As a result of this increase in carbohydrate oxidation, fatigue was delayed by ~30 min.

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Present address for A. R. Coggan and address for correspondence: Andrew R. Coggan, Ph.D., Washington University School of Medicine, Section of Applied Physiology, 4566 Scott Ave., Campus Box 8113, St. Louis, MO 63110.

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