

Carbohydrate Feeding during Exercise

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Abstract

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During strenuous exercise (i.e. 70% maximal O₂ consumption) there is a progressive shift from muscle glycogen to blood glucose oxidation with increasing duration of exercise. By maintaining blood glucose concentration and the rate of carbohydrate oxidation necessary to exercise strenuously, carbohydrate consumption throughout exercise delays fatigue by 30-60 min in endurance-trained subjects. This requires exogenous glucose supplementation at rates in excess of 1 gram/min (i.e., 16 mg/kg/min) as evidenced by the observation that intravenous glucose infusion at this rate is required to maintain blood glucose at 5 mM. Exogenous glucose must be infused at a rate of 2.6 gram/min (i.e., 37 mg/kg/min), which is similar to the total rate of carbohydrate oxidation, in order to maintain blood glucose at 10 mM after 2 h of exercise. However, carbohydrate supplementation during intense exercise does not spare muscle glycogen utilization in people. This suggests that over the course of 2-4 hours of exercise at 70% VO_{2max}, muscle glycogen and blood glucose contribute equally to total carbohydrate oxidation. Furthermore, during the latter stages of prolonged exercise, exogenous blood glucose supplementation may be capable of supplying almost all of the carbohydrate requirements of exercise at intensities up to 70% VO_{2max}.

Key words

Blood glucose, glucose supplementation, endurance-trained subjects

Introduction

The relative contribution of carbohydrate and fat to energy depends upon the intensity and duration of exercise as well as upon the preexercise diet and the state of physical training (17, 18, 19, 21). Carbohydrate metabolism during exercise has typically been manipulated by altering diet either before or during exercise. In a study of a dog named Joe who ran for longer period on laboratory treadmill when fed glu-

cose, Dill et al. (13) concluded that the limiting factor in the performance of prolonged exercise "seems to be merely the quantity of easily available fuel" in the form of blood-borne glucose.

In contrast to these observations in the dog, early studies of humans focused upon the effects of altering blood glucose concentration upon the central nervous system (16, 22). Carbohydrate ingestion during exercise was therefore thought to improve performance primarily by preventing symptoms of neuroglucopenia. This concept was reinforced by the classic studies of Christensen and Hansen (5, 6, 7).

Carbohydrate Energy and Strenuous Exercise: Importance of Muscle Glycogen

The introduction of the muscle biopsy technique in the 1960's demonstrated that muscle glycogen levels become depleted after intense exercise performed to fatigue (4). Furthermore, the duration that strenuous exercise could be maintained was altered by raising and lowering preexercise muscle glycogen levels through manipulation of diet and exercise (3). This has led to the concept that muscle glycogen is the primary carbohydrate source during exercise. Although this is not incorrect, this oversimplified concept indirectly minimizes the potential of blood glucose to serve as a fuel for carbohydrate oxidation during exercise at 60-80% VO_{2max}.

Carbohydrate Energy and Strenuous Exercise: Contribution of Blood Glucose

Data from studies conducted during the 1970's, which directly measured muscle glucose uptake, indicated that blood glucose could make substantial contributions to energy metabolism. Wahren et al. (23) observed that leg glucose uptake increases during exercise of increasing duration and intensity. Ahlborg et al. (1) also reported that the progressive increase in leg glucose uptake with increasing exercise duration was halted due to a decline in blood glucose concentration secondary to reduced liver glucose output. This was assumed to result from a depletion of liver glycogen stores. Ahlborg and Felig (2) subsequently demonstrated that when blood glucose concentration was maintained throughout exercise by glucose ingestion, that leg glucose uptake was maintained at high levels. Additionally, Gollnick et al. (15) observed that the continued increase in leg glucose uptake was related to the number of muscle fibers low in muscle glycogen. These studies of leg glucose uptake did not typically employ exercise of sufficient intensity or duration and with exogenous blood glucose supplementation and therefore the association between blood glucose concentration, oxidation and endurance was not established.

Evidence That Carbohydrate Feedings Delay Fatigue

Although it was recognized that muscle glucose uptake can increase to high levels especially when ingesting carbohydrate (2), in 1982 Felig et al. (14) concluded that glucose feedings do not delay fatigue during bicycle ergometer exercise at 60 to 65% of VO_{2max} . An indication that carbohydrate ingestion may improve endurance performance in people was provided by Ivy et al. (20) yet this concept was not generally accepted (9). Coyle et al. (11) demonstrated that carbohydrate feedings delay fatigue and improve endurance in people by preventing blood glucose from declining to levels which, in most of the subjects, causes local muscle fatigue during the latter stages of prolonged exercise.

Carbohydrate Feedings Do Not Affect Muscle Glycogen Use During Continuous Strenuous Exercise

In a subsequent study we measured the pattern of decline in muscle glycogen concentration in the vastus lateralis during exercise to fatigue at 74% VO_{2max} on two occasions, with and without carbohydrate feedings (10). Fatigue occurred after 3.0 ± 0.2 h when fed a placebo, whereas fatigue was delayed until 4.0 ± 0.3 h when fed carbohydrate (i.e., approximately 70 g of maltodextrins in a 50% solution at 20 min followed by 28 g in a 10% solution every 20 min thereafter). As shown in Fig. 1C, the pattern of decline in muscle glycogen concentration was similar during the first three hours of exercise with and without carbohydrate feeding. Remarkably, the additional hour of exercise made possible by carbohydrate feedings occurred without a further decline in muscle glycogen concentration.

We interpret these observations to indicate that the lowering of blood glucose during the latter stages of prolonged strenuous exercise (Fig. 1A) plays a major role in the development of muscular fatigue by not allowing leg glucose uptake to increase sufficiently to offset reduced muscle glycogen availability. When plasma glucose was maintained at 4–5 mM, through carbohydrate ingestion, it was theorized that blood glucose can largely replace muscle glycogen in providing carbohydrate for oxidation during the latter stages of prolonged strenuous exercise. These concepts are summarized in Fig. 2. An interesting question regards the cause of fatigue during exercise with the carbohydrate feedings.

Carbohydrate Supplementation at Fatigue

In order to more directly test the hypothesis that carbohydrate feedings improve exercise performance by preventing the decline in blood glucose concentration and oxidation late in exercise, we reasoned that it should be possible to reverse the decline in carbohydrate oxidation as well as fatigue during exercise when fasted by restoring euglycemia (8). Therefore, on three separate occasions subjects first exercised at 70% VO_{2max} to the point of fatigue when fasted, displaying a decline in plasma glucose concentration (i.e. to 3.1 mM) and RER (i.e. to 0.81) prior to fatigue. After a 20 min rest, the subjects were encouraged to perform further exercise with three different treatments. When they received a placebo solution to drink during the rest, the subjects tolerated only an additional

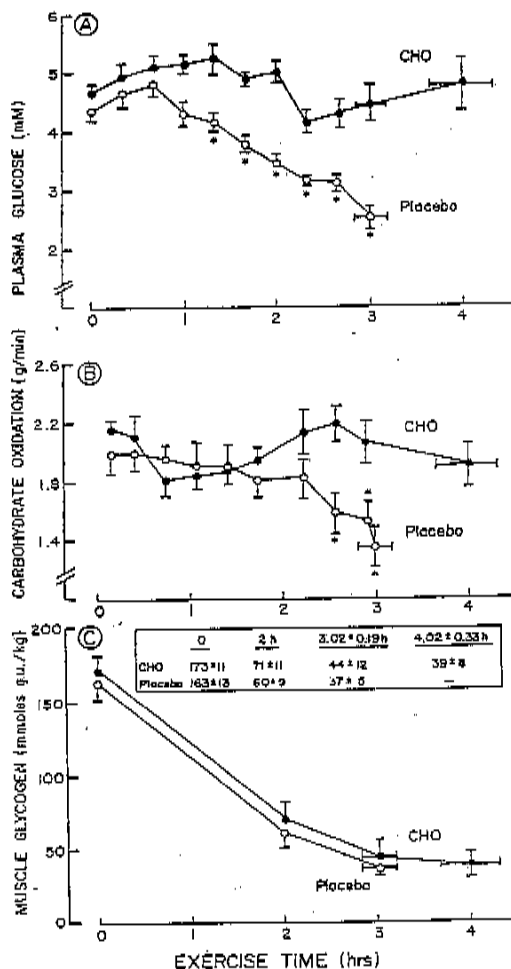


Fig. 1 Responses when cycling at 74% VO_{2max} with a placebo or when ingesting carbohydrate every 20 min (CHO). *Placebo different from carbohydrate, $p < 0.05$. (Reprinted with permission from Ref. 10.)

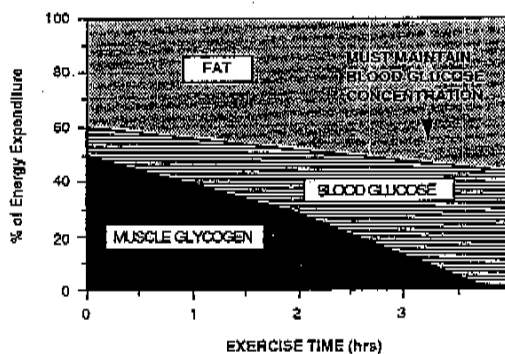


Fig. 2 Various sources of energy during prolonged exercise at 70% VO_{2max} . Note that blood glucose becomes the predominant sources of carbohydrate energy during the latter stages of exercise and thus it is important to maintain blood glucose concentration by eating carbohydrate.

10 ± 1 min of exercise. During a second trial, 200 g of a 50% maltodextrins solution was ingested during the rest period. As a result, during exercise bout 2, plasma glucose concentration and RER were initially increased above levels at fatigue of exercise bout 1, but they could not be maintained, declining progressively to the point of fatigue, which occurred after 26 ± 4 min. During a third trial, glucose was infused (via a pump) intravenously at the beginning of exercise bout 2 at the rate required to maintain plasma glucose concentration at 5 mM. This maintained RER above the levels observed at fatigue during exercise bout 1 and the subjects completed an additional 43 ± 5 min of exercise. It was also observed that muscle glycogen use was minimal during this additional exercise, suggesting that blood glucose was the primary energy source for carbohydrate oxidation, which was occurring at 1.6 g/min.

An important finding of this study was that a glucose infusion rate of over 1.1 g/min was required to maintain euglycemia, suggesting that this exogenous glucose was being oxidized at a high rate under these conditions (i.e. low muscle glycogen, hypoinsulinemic). Since muscle glycogen contributed little to energy, it is likely that endogenous glucose production supplemented the exogenous glucose infusion in providing the carbohydrate needs of exercise at this intensity. We have recently observed that in order to keep blood glucose concentration at 10 mM during exercise, the rate of exogenous intravenous glucose infusion must be increased progressively during the second hour of exercise to a value of 2.6 grams/min which is approximately equal to the rate of total carbohydrate oxidation (12). However, these data cannot provide information as to whether glucose disposal is oxidized in the exercising muscle or taken-up by other tissues.

In conclusion, fatigue during prolonged strenuous exercise is often due to inadequate carbohydrate oxidation. This is sometimes a result of hypoglycemia which limits carbohydrate oxidation and causes muscle fatigue. Carbohydrate feedings during strenuous exercise maintain blood glucose oxidation and delay fatigue by 30–60 min, but do not prevent fatigue, which eventually results from other yet unknown factors. During the latter stages of prolonged cycling, when muscle glycogen is low, it appears that blood glucose can supply carbohydrate energy at rates necessary to exercise at approximately 75% VO₂max in well-trained cyclists.

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