Substrate utilization during exercise in active people[1–3]

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ABSTRACT When people walk at low intensity after fasting, the energy needed is provided mostly by oxidation of plasma fatty acids. As exercise intensity increases (eg, to moderate running), plasma fatty acid turnover does not increase and the additional energy is obtained by utilization of muscle glycogen, blood glucose, and intramuscular triglyceride. Further increases in exercise intensity are fueled mostly by increases in muscle glycogen utilization with some additional increase in blood glucose oxidation. Muscle glycogen and blood glucose contribute equally to carbohydrate energy production over 2–3 h of moderate-intensity exercise; fatigue develops when these substrates are depleted. Active people can deplete muscle glycogen with 30–60 min of high intensity, intermittent exercise. When the ingestion of dietary carbohydrate is optimal, it is possible to resynthesize muscle glycogen to high concentrations in ≈24 h, which is the major factor in recovery of exercise tolerance. However, this requires that a 70-kg person eat at least 50 g carbohydrate per every 2 h, beginning soon after exercise, and ingest 500–600 g in 24 h (ie; ≈7–9 g/kg body wt). Carbohydrate foods eliciting high glycemic and insulinemic responses promote more rapid glycogen resynthesis than do foods eliciting lower glycemic responses. Therefore, foods ingested for energy before, during, or after exercise should be classified according to their glycemic index. Although carbohydrate ingestion before and during exercise adds exogenous substrate to the body, it usually attenuates plasma fatty acid mobilization and oxidation. *Am J Clin Nutr* 1995;61(suppl):968S–79S.

KEY WORDS Exercise, muscle glycogen, blood glucose, fat, free fatty acids, fatigue, simple carbohydrate, complex carbohydrate, glycemic index, insulin

Introduction

The energy used to power steady-state aerobic exercise in people is derived predominantly from the oxidation of carbohydrate and fat (1). Normally, bodily protein oxidization does not contribute significantly to energy production (2). Therefore, the four major sources of energy for exercise are muscle glycogen, blood glucose, plasma fatty acids, and intramuscular triglyceride (3, 4). People cannot oxidize fat at high enough rates to provide all the energy required by moderate- to high-intensity exercise. At these intensities, carbohydrate oxidation must provide the energy not available from fat (3, 5). Consequently, fatigue often occurs when muscle glycogen and blood glucose stores become depleted (5). This is the rationale for dietary carbohydrate supplementation before, during, and after exercise. To better understand the importance of adequate dietary carbohydrate to exercise performance, it is helpful to first discuss the factors that limit fat metabolism during exercise.

The amount of energy stored in the form of triglycerides within adipocytes throughout the body is large, totaling 200–625 MJ (≈50 000–150 000 kcal) in men and women with a normal body composition of 10–30% body fat. Because ≈400 kJ are expended to walk or jog 1.6 km (1 mile), most people have enough energy stored in body fat to travel a total of 800-2400 km. Triglycerides stored in adipocytes can be hydrolyzed (ie, lipolysis) into glycerol and free fatty acids, the latter must bind to the protein carrier albumin for transport via the circulation (ie; plasma fatty acids) to the exercising muscles (6). Additional triglyceride, amounting to ≈12 000–20 000 kJ, is stored in droplets within the muscle fibers and is available for oxidation following intramuscular lipolysis. Therefore, the two forms of fat for oxidation by muscle during exercise are plasma fatty acids and intramuscular triglycerides. Despite the large amount of potential energy in these body fat stores, the rate at which they can be oxidized is limited and thus carbohydrate metabolism is needed to provide the additional substrate for oxidation as the intensity of exercise is increased.

Carbohydrate is stored as glycogen, both within the muscle fiber and the liver (7). Depending on diet and activity pattern, ≈10–30 g glycogen are stored in each kilogram skeletal muscle; thus, ≈8400 kJ are available to power exercise. Additionally, ≈80 g glycogen is stored in the liver. Liver glycogen can be hydrolyzed back to glucose and transported via the blood to the muscles for oxidation. Because carbohydrate oxidation is needed to maintain strenuous exercise for prolonged periods, yet bodily stores are limited, active people can become undernourished in carbohydrate and thus experience muscle fatigue while exercising.

The purpose of this review is to first describe the extent to which the various substrates are used for energy during prolonged exercise throughout a wide range of intensities. Recent findings regarding limitations in the mobilization and oxidation

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[2] This article is an update of a previous review by the author (Coyle EF. Timing and method of increasing carbohydrate intake to cope with heavy training, competition and recovery. J Sports Sci 1991;9:29–52).

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of fatty acids are presented, emphasizing the importance of carbohydrate for energy. Furthermore, methods will be presented for increasing carbohydrate intake in active people to speed recovery from exercise training and to improve performance. The conditions that benefit from exogenous carbohydrate supplementation, the critical timing of supplementation, and the amount and type of carbohydrate that is best, are discussed. The recommendations developed will be most applicable to people who frequently exercise intensely or for prolonged periods of time (ie; ≥ 1 h), such as athletes. However, these guidelines also apply to normal individuals who become more active for certain periods of time.

Substrate use during exercise of various intensities

The contribution of the four major substrates to total energy expenditure during exercise at a wide range of intensities is shown in **Figure 1**, which is from recent studies using stable isotope infusion to quantify substrate turnover (4). These measures represent values after 30 min of exercise in the fasted state in endurance-trained people. Exercise duration, diet, and state of training modify these responses. Plasma triglycerides are a potential source of energy for muscle. However, triglyceride entry into muscle is catalyzed by lipoprotein lipase, which is not capable of meeting more than a small percentage of the energy needs of strenuous exercise (8). Plasma triglycerides, however, are important for recovering intramuscular triglyceride during the long periods between exercise bouts (8).

Almost all of the energy for exercise at the low intensity of 25% maximal oxygen uptake ($\dot{V}O_2$max), comparable to walking, is derived from plasma fatty acids, with an additional small contribution from blood glucose when performed in the fasted state. During this low-intensity exercise, the rate of appearance (Ra) of fatty acids in plasma is very similar to the rate of fatty acid oxidation (ie; 26 μmol · kg^{-1} · min^{-1}) in endurance-trained people. This pattern does not change over the course of 2 h of exercise, or probably longer, because all of the energy requirements are met by mobilization of fatty acids from the large triglyceride stores in adipocytes throughout the body.

However, as exercise intensity is increased from 25% to 65% and then to 85% $\dot{V}O_2$max, the Ra of plasma fatty acids declines progressively and as a result the concentration of fatty acids in the blood is proportionally reduced as shown in **Figure 2**. This reduced plasma fatty acid mobilization occurs despite a maintained high rate of lipolysis from adipocytes, as determined from direct measures of the Ra of glycerol, which is an index of lipolysis (4). The Ra of plasma fatty acids declines with increasing intensity of exercise because of insufficient blood flow and albumin delivery to carry fatty acids from adipose tissue into the systemic circulation (6, 9). Therefore, it appears that fatty acids become trapped in adipose tissue as exercise intensity is increased. This theory is supported by the observation that cessation of exercise at 65% and 85% $\dot{V}O_2$max results in a large increase in the Ra of plasma fatty acids as well as the plasma fatty acid concentration without a concomitant increase in the Ra of glycerol (ie; lipolysis) (4). Whatever the mechanism, the availability of plasma fatty acids for oxidation by muscle declines as the intensity of exercise is increased (Figures 1 and 2).

When exercising at moderate intensities (ie; 65% $\dot{V}O_2$max), comparable to the pace chosen when running for 1–3 h, total fat oxidation increases despite the reduction in the Ra of fatty acids. The substantially higher rate of total fat oxidation compared with entry of fatty acids into plasma reflects an increased

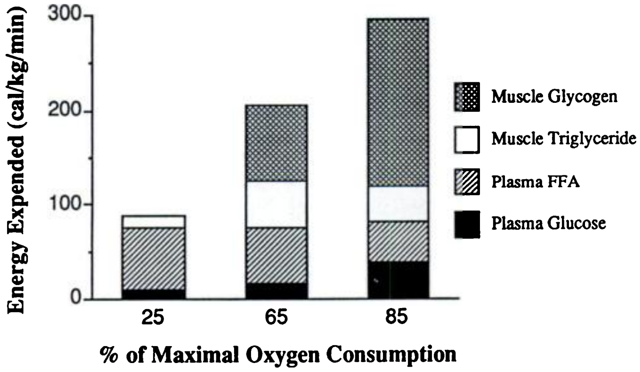


FIGURE 1. Contribution of the four major substrates to energy expenditure after 30 min of exercise at 25%, 65%, and 85% of maximal oxygen uptake. FFA, free fatty acids. For energy, cal × 4.186 = J. Reproduced with permission from Romijn et al (4).

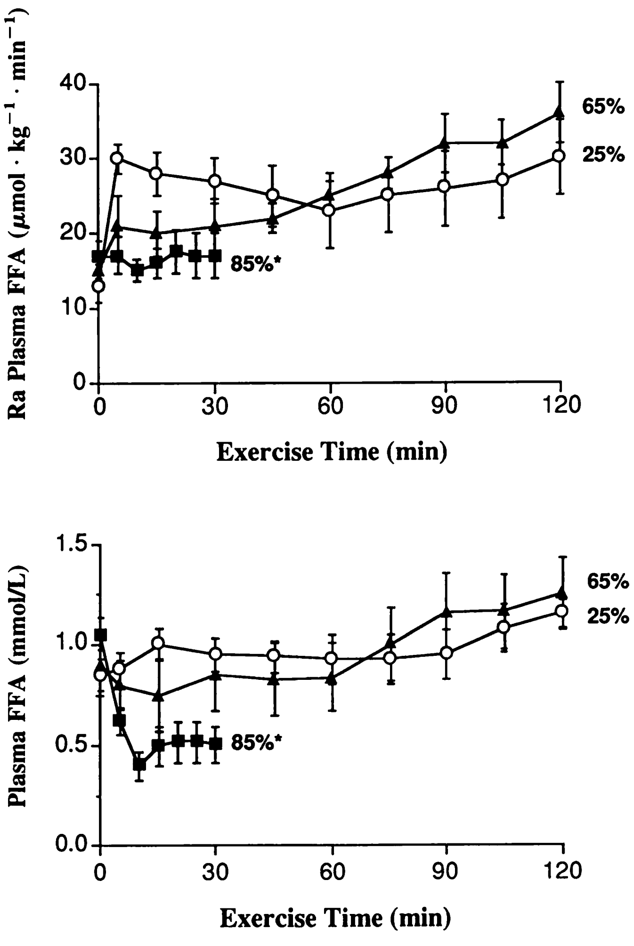


FIGURE 2. Rate of appearance (Ra) of free fatty acids (FFA) in plasma (upper panel) and plasma FFA concentration (lower panel) during exercise at 25%, 65%, and 85% of maximal oxygen uptake. $\bar{x}$ ± SE. *Values during exercise at 85% maximal oxygen uptake are significantly lower compared with those at 25% and 65% maximal oxygen uptake ($P < 0.05$). Redrawn with permission from Romijn et al (4).

oxidation of intramuscular triglycerides (Figure 1) (4). In fact, during moderate-intensity exercise in endurance-trained people, plasma fatty acids and intramuscular triglyceride contribute equally to total fat oxidation (Figure 1) (10, 11), and total fat oxidation is highest during moderate-intensity exercise (ie; $> 40\ \mu mol \cdot kg^{-1} \cdot min^{-1}$). However, fat cannot be oxidized at sufficiently high rates to provide all of the energy for moderate-intensity exercise (ie, 60–75% $\dot{V}O_2max$) and therefore about one-half of the total energy must be simultaneously derived from carbohydrate oxidation (ie, muscle glycogen and blood glucose) (Figure 1). During moderate-intensity exercise, all four major substrates contribute substantially to energy production. This is the intensity generally chosen by people performing aerobic exercise to develop fitness.

High-intensity exercise (85% $\dot{V}O_2max$) is performed at a level that promotes relatively high rates of muscle glycogen breakdown and thus carbohydrate oxidation (Figure 1). This results in accelerated rates of lactic acid production, which accumulates in muscle and blood. High-intensity exercise represents the greatest intensity that a person can maintain for a 30–60-min period, with great effort and with the sensation of fatigue in the exercising muscles. At high intensities, carbohydrate oxidation provides more than two-thirds of the needed energy with the remainder coming from plasma fatty acids and intramuscular triglycerides. However, the absolute rate of fat oxidation is significantly decreased (ie, from 43 to 30 $\mu mol \cdot kg^{-1} \cdot min^{-1}$) when the intensity of exercise is increased from 65% to 85% $\dot{V}O_2max$. Part of this decline appears to be due to the marked suppression of the Ra of plasma fatty acids during high-intensity exercise and the resulting reduction in the concentration of fatty acids in the plasma, as shown in Figure 2. When this reduction in plasma fatty acid availability is reversed by intravenous lipid infusion to restore the plasma fatty acid concentration to > 1 mmol/L, fat oxidation is increased somewhat and muscle glycogen utilization is decreased during high-intensity exercise (12). However, fat oxidation does not completely return to the high levels observed during moderate-intensity exercise. This indicates that when the availability of fatty acids is high, the muscle becomes limited in its ability to oxidize fat during intense exercise. Plasma fatty acid concentrations cannot be appreciably increased by ingesting or intravenously infusing triglycerides alone—triglycerides must be hydrolyzed to fatty acids through the action of lipoprotein lipase, which in these experiments is released into the blood from the capillary endothelium by heparin injection. Therefore, there are no practical methods yet available to increase long-chain fatty acid concentrations in plasma via exogenous supplementation alone, leaving this responsibility to the adipocytes. Medium-chain triglycerides can be absorbed into the portal circulation as fatty acids (13). However, the extent to which their ingestion raises plasma fatty acids in the systemic circulation and increases total fat oxidation by muscle during exercise is currently unclear (13, 14).

Therefore, the answer to the question "is body fat always readily available for oxidation during exercise?" depends on the intensity of exercise. At low intensities (ie, 25% $\dot{V}O_2max$) and when the subject has fasted, the answer is apparently yes, as evidenced by the progressive increase in plasma fatty acid concentration throughout exercise (Figure 2). During moderate exercise (ie, 65% $\dot{V}O_2max$) in endurance-trained people, plasma fatty acid mobilization is not sufficient to meet the muscle's ability to oxidize fat; under these conditions intramuscular triglycerides become an important fuel, as do muscle glycogen and blood glucose. During the first 30 min of moderate-intensity exercise, plasma fatty acid concentrations decline and the Ra of fatty acids is not high; therefore, plasma fatty acid availability may be suboptimal (Figure 2). However, throughout the remainder of moderate-intensity exercise, the Ra of fatty acids and the concentration of plasma fatty acids increase without proportional increases in fat oxidation, suggesting that the availability of plasma fatty acids is adequate. Therefore, besides possibly the early portion of moderate-intensity exercise, the fat-oxidative ability of muscle seems to be met by sufficient plasma fatty acid availability, with intramuscular triglycerides providing the substrate for fat oxidation not met by mobilization of plasma fatty acids from adipocytes, at least in endurance-trained subjects. Additionally, carbohydrate oxidation provides the remaining energy that cannot be met by fat oxidation. During high-intensity exercise, when muscle glycogenolysis is heavily stimulated, plasma fatty acid mobilization is simultaneously suppressed and the delivery of fatty acids to muscle is very low. As a result, muscle glycogenolysis is stimulated slightly more than when plasma fatty acid concentrations are high, and this response is consistent with the muscle's shift to carbohydrate oxidation.

Note that these are the responses of people who have fasted for 8–12 h before exercise. Carbohydrate meals during the 12-h period before exercise appear to be a major factor regulating fat mobilization during exercise. The attenuation of fat oxidation during exercise of low to moderate intensity by preexercise carbohydrate feedings may partly be due to suppressed fatty acid mobilization as well as to metabolic alterations in the exercising muscles that increase carbohydrate oxidation (15–17).

Although these observations indicate that mobilization and oxidation of triglyceride in adipocytes are greatest during low-intensity exercise after fasting, they cannot be used as a basis at this time for recommending that people limit their exercise to these conditions when attempting to lose body fat. Body fat reduction is a function not only of oxidation during exercise but also of the composition and amount of energy intake over extended time periods. Low-intensity exercise that does not oxidize much glycogen requires less glycogen resynthesis from subsequent dietary carbohydrate. In my opinion, the ideal exercise and diet programs for regulating body fat mass have yet to be convincingly established. However, it is clear that energy expenditure through exercise is a very important component of body fat mass regulation.

Muscle's limited ability to oxidize fat makes carbohydrate essential

When carbohydrate oxidation declines as the result of depletion of muscle glycogen and hypoglycemia, people are unable to oxidize fat at rates sufficient to meet the energy requirements of even moderate-intensity exercise (60–75% $\dot{V}O_2max$). As people fatigue, they must reduce the work rate to the lowest intensity (ie, 30–50% $\dot{V}O_2max$) that matches their ability to predominantly oxidize fat (3, 18). This is necessary even when high concentrations of plasma fatty acids are present (3, 18). Therefore, high absolute rates of fat oxidation do not seem to be limited by plasma fatty acid availability but appear instead to be limited by muscle's oxidative ability. This may explain the reason why further elevation of plasma fatty acid concentrations during low- to moderate-intensity exercise fails to increase total fat oxidation in some studies (19, 20).

The reason for the muscle's limited ability to oxidize fat, and thus its dependence on carbohydrate, is not entirely clear. One line of thinking is that the limitation is in the transport of fatty acids into the mitochondria. Traditionally, this has been thought to be limited by carnitine palmitoyltransferase (CPT) activity for transport of fatty acids across the mitochondrial membrane. Additionally, free fatty acid binding proteins control the transport of fatty acids through the interstitium and cell membranes as well as the cytoplasm (21, 22). Fat oxidation during exercise may be limited by transport in numerous sites. Additionally, CPT activity is inhibited by malonyl-CoA, the first intermediate in the conversion of glucose to fat (23). Carbohydrate availability in muscle may reduce fat oxidation through malonyl-CoA inhibition of CPT-stimulated fatty acid transport across the mitochondrial membrane (24).

Because fat oxidation can only provide about one-half of the energy needed for exercise at 70% $\dot{V}O_2$max and no more than one-third of the energy needed for more strenuous exercise lasting 10–30 min (ie, > 85% $\dot{V}O_2$max) (Figure 1), the need for adequate muscle glycogen and blood glucose concentrations becomes clear. Although the muscle is clearly limited in its ability to oxidize fat, there are some conditions in which plasma fatty acid concentration is not optimal, such as during high-intensity exercise and during exercise after a carbohydrate meal. However, at this time there are no practical means of raising plasma concentrations of long-chain fatty acids via ingestion. Besides, that would not appreciably raise fat oxidation. As presented below, the muscle's reliance on carbohydrate oxidation demands that active people eat sufficient amounts of carbohydrate. This provides important substrate for oxidation during exercise as well as for recovery of muscle glycogen after exercise.

Carbohydrate feeding during exercise

Prolonged continuous moderate-intensity exercise

After 1–3 h of continuous exercise at moderate intensities (ie, 60–80% $\dot{V}O_2$max), it is clear that people fatigue as the result of carbohydrate depletion. Carbohydrate feedings (ie, glucose, maltodextrins, or sucrose) during exercise delay fatigue by 30–60 min (3, 5, 25). However, this improvement in performance is not due to a sparing of muscle glycogen use during exercise (3, 26–31). Instead, it appears that the exercising muscles rely mostly on blood glucose for energy late in exercise (3, 5).

These concepts are summarized in **Figure 3**, which displays the substrate shifts during prolonged exercise at 65–75% $\dot{V}O_2$max in endurance-trained subjects after they have fasted overnight (3–5). About 50% of the energy for exercise at 70% $\dot{V}O_2$max is derived from fat, with equal contributions from plasma fatty acids and intramuscular triglycerides during the early period. There is a small increase in plasma fatty acid contribution over time. The remaining 50% of the energy is derived from carbohydrate. During the early portions of exercise, the majority of carbohydrate energy is from muscle glycogen. As exercise progresses, muscle glycogen is reduced and contributes less to the carbohydrate requirements of exercise and there is increased reliance on blood glucose.

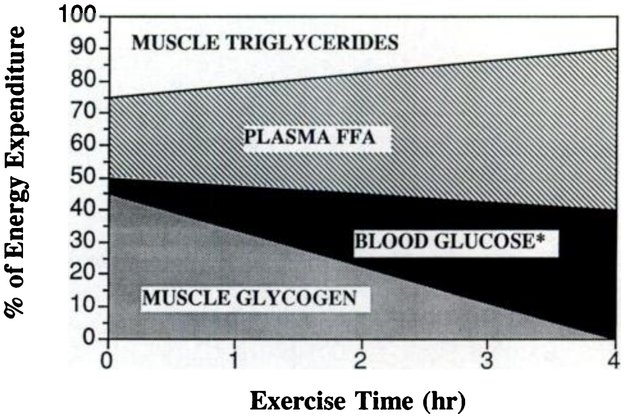


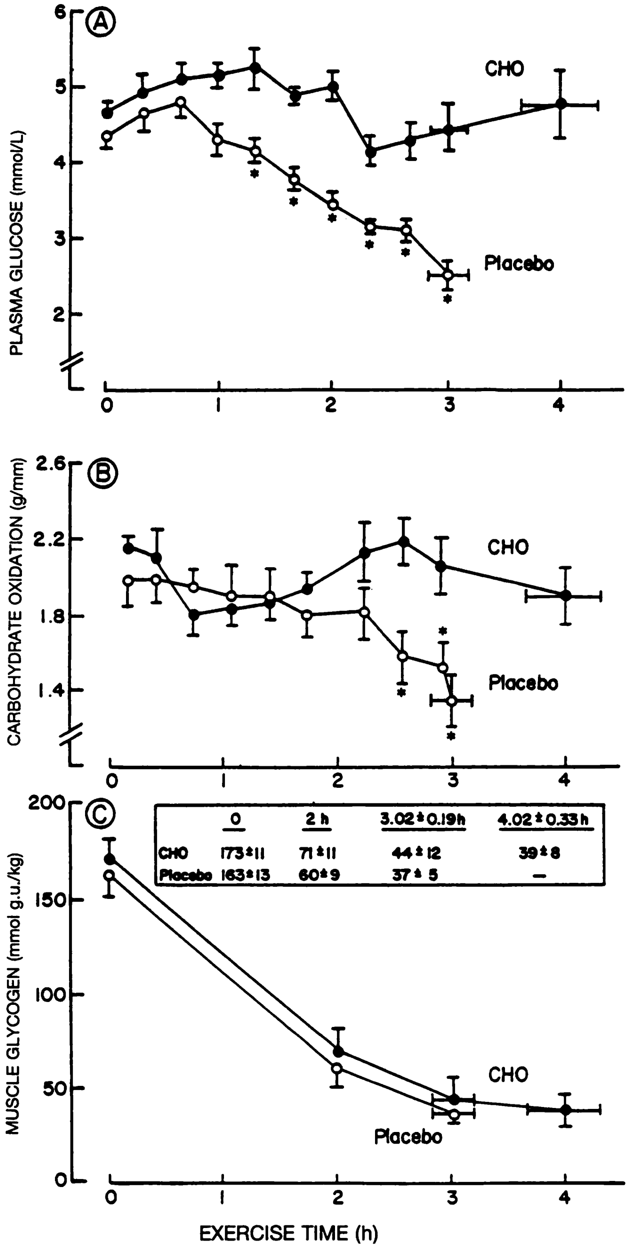
FIGURE 3. Percentage of energy derived from the four major substrates during prolonged exercise at 65–75% of maximal oxygen uptake. Initially, approximately one-half of the energy is derived each from carbohydrate and fat. As muscle glycogen concentration declines, blood glucose becomes an increasingly important source of carbohydrate energy for muscle. *After 2 h exercise, carbohydrate ingestion is needed to maintain blood glucose concentration and carbohydrate oxidation. FFA, free fatty acids. From references 3 and 4.

Figure 4 displays the actual plasma glucose and muscle glycogen responses to prolonged cycling, as well as the rates of carbohydrate oxidation. After 3 h of exercise while drinking only flavored water (ie, fasting placebo), muscle glycogen is low and the majority of carbohydrate energy is derived from the metabolism of glucose, which is transported from the circulating blood into the exercising muscles. When drinking only water (ie, placebo), fatigue occurs after ≈3 h because of a lowering of blood glucose, which causes an inadequate supply of this carbohydrate energy for oxidation. However, when carbohydrate is ingested throughout exercise and glucose in the bloodstream remains high, subjects maintain the necessary reliance on carbohydrate for energy and fatigue is delayed by 1 h (ie, from 3 to 4 h). Remarkably, muscle glycogen use is minimal during the additional hour of exercise despite the fact that carbohydrate oxidation is maintained (Figure 4). This indicates that blood glucose is the predominant carbohydrate fuel during the later stages of exercise.

The shortest duration of moderate- to high-intensity exercise that benefits from carbohydrate feeding is not entirely clear. Blood glucose concentrations decline during the second hour of exercise and therefore it is generally believed to be beneficial for durations of exercise lasting > 2 h. There is preliminary evidence that carbohydrate feedings may also benefit intense exercise (80–90% $\dot{V}O_2$max) lasting 1 h (32). However, there is no compelling evidence that carbohydrate ingestion reduces muscle fatigue during low- to moderate-intensity exercise (40–70% $\dot{V}O_2$max) lasting < 1 h.

Type of carbohydrate

Glucose, sucrose, and maltodextrins appear to be equally effective in maintaining blood glucose concentrations and carbohydrate oxidation and in improving performance (33, 34). Therefore, the selection of carbohydrate for ingestion during exercise should be based on what is best tolerated under the



	0	2 h	3.02±0.19h	4.02±0.33h
CHO	173±11	71±11	44±12	39±8
Placebo	163±13	60±9	37±5	—

FIGURE 4. Plasma glucose (A), carbohydrate oxidation (B), and muscle glycogen (C) responses when cycling at 74% of maximal oxygen uptake when ingesting a placebo (ie, flavored water) or when ingesting carbohydrate (CHO) every 20 min. $\bar{x}$ ± SE. *Significantly different from carbohydrate; $P < 0.05$. g.u., glycogen used. From reference 3.

conditions. Liquids are obviously easier to ingest during exercise than solids and they also provide for fluid replacement. Maltodextrins have become a popular form of carbohydrate for inclusion in sports drinks because they are not very sweet-tasting and therefore solutions in concentrations ≥ 100 g/L are more palatable for most people. However, exercising people can generally meet both their carbohydrate and fluid needs by ingesting 600-1000 mL/h of a carbohydrate solution containing 4–10% carbohydrate (35, 36).

Fructose feedings during exercise have not been observed to be effective for improving performance compared with glucose or sucrose because its conversion to and oxidation as glucose is not rapid enough to supply the carbohydrate energy required late in exercise (34, 37). For the same reason, low glycemic index fruits and foods would probably be of little benefit compared with high glycemic index food if ingested during exercise. However, it has been suggested that the slower conversion to glucose of low glycemic index foods may be of some benefit if ingested before exercise, presumably to meet the carbohydrate oxidation needs late in exercise (38).

Timing of carbohydrate feedings during continuous exercise

Consuming carbohydrate during prolonged continuous exercise ensures that sufficient carbohydrate will be available during the later stages of exercise. If carbohydrate feedings are withheld until the point of exhaustion, fatigue can only be reversed, and exercise continued for another 45 min, if glucose is intravenously infused at a high rate (ie, > 1 g/min) (25). This of course is not practical but it indicates that the object of carbohydrate feeding throughout exercise is to provide the exercising muscle with approximately an additional 1 g exogenous glucose/min late in exercise. Most people cannot wait until they are exhausted before drinking concentrated carbohydrate solutions because they are unable to absorb it rapidly enough to maintain the energy needs of the exercising muscles (25). The latest that a person can delay carbohydrate ingestion is 30 min before the time of fatigue when ingesting only water (39). However, a better approach is to ingest carbohydrate at regular intervals throughout exercise according to the guidelines given below.

Rate of carbohydrate ingestion

Sufficient carbohydrate should be ingested to supply the blood with exogenous glucose at ≈1 g/min late in exercise. Therefore, ≈60 g exogenous glucose must be readily available within the body. To ensure this, it seems that larger amounts of carbohydrate must be ingested. In the majority of studies in which carbohydrate ingestion throughout exercise improved performance, subjects were fed at a rate of 30–60 g/h beginning early in exercise (5, 40). This generally agrees with the expected needs and glucose distribution within the body, although it should be recognized that the fate of the ingested glucose that is not oxidized is unclear.

Carbohydrate ingestion during sport, recreation, and intermittent exercise

Carbohydrate feedings are also beneficial during sports involving high intensity intermittent exercise such as soccer and ice hockey, which cause fatigue because of glycogen depletion (41–43). The ingestion of carbohydrate throughout the game, and during the half-time rest period, results in higher muscle glycogen and increased sprinting ability toward the end of the game compared with when no carbohydrate is ingested and muscle glycogen remains low.

This model emphasizes that carbohydrate feeding during prolonged exercise improves performance in events that are performed for > 2 h and thus result in hypoglycemia. The extent to which carbohydrate feedings improve performance in events that are < 2 h in duration and that are not obviously limited by carbohydrate availability is less clear. Several studies have observed carbohydrate ingestion to improve performance even when blood glucose availability and carbohydrate energy were not obviously limiting when only water was consumed (29, 32, 34, 44–46). This suggests that carbohydrate ingestion improves performance in events lasting > 60 min. Additionally, if body carbohydrate stores are reduced before the onset of exercise because of inadequate diet and previous exercise, carbohydrate supplementation can improve performance during exercise lasting 60 min (47).

Carbohydrate types and metabolic responses

Glucose is the only type of carbohydrate (ie, sugar or starch) that skeletal muscle can readily metabolize for energy and that it can store as glycogen. Carbohydrates can be functionally classified according to the extent to which they increase blood glucose concentrations (ie, their glycemic index) and by the extent to which they trigger insulin secretion. The glycemic index is generally determined by the rate at which the ingested carbohydrate is made available to intestinal enzymes for hydrolysis and intestinal absorption (48, 49). This is a function of the gastric emptying time (50) and the physical availability of the sugar or starch to hydrolytic enzymes. The latter is influenced by cooking, which alters the integrity of the starch granule (51) and the degree of gelatinization (48). Another factor is the amylose or amylopectin content of food (52, 53). Adding fiber to glucose also slows the rate of carbohydrate entry into blood and oxidation during exercise (54). It is a misconception to think that the glycemic index is simply a function of whether the carbohydrate is complex (ie, starch) or a simple sugar. Some starchy foods produce glycemic responses that are identical to that of glucose (eg, mashed potato and maltodextrins) (17, 55, 56). On the other hand, the rise in blood glucose after eating fructose or sucrose is less than that observed for a wide range of starchy complex carbohydrates (eg, potato, bread, and corn flakes) (57, 58). Foods for active people should be classified as having a high, moderate, or low glycemic index, as outlined by Jenkins et al (57, 58).

The classification of foods according to their glycemic index does not consider the insulin response. Insulin, of course, has large effects on metabolism and is functionally significant because it regulates glucose disposal. Blood glucose and insulin responses are generally related. However, some foods that do not differ in glycemic response could differ in insulin secretion, which has the potential to alter metabolism (59). Therefore, the classification of foods according to their glycemic index is recognized to be rather simplistic. However, its functional significance for describing the metabolic effects of carbohydrate ingestion is certainly better than the prevalent classification of simple or complex carbohydrate, especially for active people. Future classification of carbohydrate foods should consider their functional influences on metabolism, which will probably be related to insulin action on tissues, especially muscle and adipocytes.

Muscle glycogen resynthesis after exercise

Muscle glycogen restoration after heavy training or competition often dictates the time needed to recover between intense bouts of exercise. It is commonly stated that muscle glycogen becomes depleted after ≈2–3 h of continuous exercise performed at intensities of ≈60–80% $\dot{V}O_2$max. Although this is true, it is not usually appreciated that muscle glycogen can also become depleted after only 15–30 min of exercise performed at very high intensities (90–130% $\dot{V}O_2$max) in "intervals" of 1–5-min exercise bouts followed by a rest period and then another "interval" and rest, etc (60). These patterns of intense exercise are typical of many individual and team sports. Therefore, people who attempt to train daily at intensities that deplete muscle glycogen must increase their carbohydrate consumption (61), which helps but does not always guarantee optimal muscle glycogen storage (62).

In people, muscle glycogen is resynthesized to normally high concentrations at a rate of only ≈5 $\text{mmol} \cdot \text{kg muscle}^{-1} \cdot \text{h}^{-1}$, which corresponds to a rate of ≈5%/h (ie, 5 $\text{mmol} \cdot \text{kg muscle}^{-1} \cdot \text{h}^{-1}$ when attempting to increase muscle glycogen by 100 mmol/kg). Therefore, ≈20 h are required to recover muscle glycogen stores. A longer time will be necessary if the diet is not optimal. The important dietary factors to consider are the following: *1*) the rate of carbohydrate ingestion, *2*) carbohydrate type, and *3*) timing of carbohydrate ingestion after exercise.

Rate of carbohydrate ingestion

Blom et al (63) and Ivy et al (64) fed subjects different amounts of high glycemic index carbohydrates (ie, glucose or maltodextrins) every 2 h after exercise and measured the rates of muscle glycogen synthesis during the first 6 h. They reported that glycogen synthesis increased from 2%/h (ie, 2 $\text{mmol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) when 25 g was ingested every 2 h to 5–6%/h (ie, 5–6 $\text{mmol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) when 50 g was ingested every 2 h. However, they did not observe muscle glycogen synthesis to increase to $>$ 5–6%/h when 100, 112, or 225 g were ingested every 2 h. This plateau in glycogen synthesis does not appear to simply be due to an accumulation of carbohydrate in the gastrointestinal tract because Reed et al (65) reported that intravenous glucose infusion at ≈100 g every 2 h also failed to increase muscle glycogen synthesis above 7–8 $\text{mmol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$. Additionally, this failure of muscle glycogen synthesis to increase with increased carbohydrate ingestion or intravenous glucose infusion (100 g per 2 h) occurred despite the fact that increased carbohydrate administration promoted progressively greater increases in blood glucose and plasma insulin concentration, which remained within the physiological range (63–65).

This suggests that the muscle glycogen synthesis is near optimal (5–7 $\text{mmol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) when ≥ 50 g glucose is ingested every 2 h, most of which enters the blood. This forms the basis for the recommendation that the amount and type of food to be eaten after exercise for near-optimal muscle glycogen resynthesis should be that which promotes glucose entry into the blood and systemic circulation at a rate of ≥ 50 g every two h. This goal can be achieved by considering both the glycemic index, which reflects the rate of absorption, and the amount of carbohydrate ingested.

Carbohydrate type and glycogen resynthesis

As discussed, the rate of glycogen synthesis after exercise and ingestion of glucose or food with a high glycemic index is 5–6%/h (5–6 $\text{mmol} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) (63–66). When sucrose is ingested, it is hydrolyzed to equal amounts of glucose and fructose. Its ingestion elicits a similar rate of glycogen synthesis as does glucose ingestion, despite the fact that the glycemic index of sucrose is 60–70% that of glucose (63), which classifies it as having a moderate to high glycemic index. However, fructose ingestion alone promotes muscle glycogen to be resynthesized at a rate of only 3% per h (3 mmol · kg^{-1} · h^{-1}) because of its low glycemic index (20–30% that of glucose) (63). Fructose ingestion, even in large amounts, cannot produce sufficient entry of glucose into the blood (ie, 50 g every 2 h) probably because of the relatively slow rate at which the liver converts fructose to glucose. Therefore, it appears that glucose and sucrose, which possess high and moderate glycemic indexes, are equally effective in the partial restoration of muscle glycogen during the 4–6-h period after exercise; however, fructose is only one-half as effective because of its low glycemic index.

A limited amount of information is available about the rates of glycogen synthesis elicited by eating common foods containing various starches and sugars. Burke et al (67) have recently reported that muscle glycogen resynthesis is 48% greater in 24 h when a variety of high glycemic index foods are eaten compared with when only moderate to low glycemic index foods are consumed. Additionally, it makes little difference if the carbohydrate is in liquid or solid form (60, 65, 68).

Little data exist concerning the extent to which carbohydrate foods with a low glycemic index promote muscle glycogen resynthesis. Legumes possess a low glycemic index largely because the carbohydrate granule is not as accessible to digestive enzymes (51), a factor that can be influenced by food processing and cooking. It is possible, however, that low glycemic index legumes can promote a sufficient rate of glucose entry into the blood for optimal muscle glycogen synthesis if a larger amount is eaten to offset the slow rate at which each gram is digested. However, until more direct data become available, it is assumed that foods with a low glycemic index should not compose the bulk of carbohydrate ingested after exercise, because it is likely that muscle glycogen synthesis would be compromised.

Timing of carbohydrate ingestion after exercise

During the first 2 h after exercise, the rate of muscle glycogen resynthesis is 7–8%/h (7–8 mmol · kg^{-1} · h^{-1}), which is somewhat faster than the normal rate of 5–6%/h, but certainly not rapid (66). A recovering athlete should ingest sufficient carbohydrate as soon after exercise as is practical. The most important reason for this is that it will provide more total time for resynthesis.

Because eating > 50 g carbohydrate (with a high or moderate glycemic index) every 2 h does not seem beneficial for increasing muscle glycogen resynthesis, one might think it best that small, frequent meals be eaten until a sufficient total amount of carbohydrate has been consumed (ie, > 600 g for a 70-kg person). However, this does not appear to be the case. Costill et al (69) fed subjects 525 g carbohydrate over the course of 24 h (70% of energy intake) and reported that muscle glycogen synthesis was similar when two large meals were eaten compared with when seven smaller meals were eaten.

Practical considerations and specific recommendations

People are not usually hungry immediately after exhaustive exercise and often prefer to drink fluids rather than eat solid foods (60). Therefore, beverages that contain glucose, sucrose, maltodextrins, or corn syrups in concentrations of ≥ 60 g/L should be made available. If preferred, there is no reason a person should not eat solid food. However, because appetite is usually suppressed, foods that are more concentrated in carbohydrate and that have a high glycemic index should be available.

When the desire for solid food returns, athletes should eat enough to ensure that a total of ≈600 g carbohydrate (> 8 g/kg body wt) are eaten within 24 h. Most of the food chosen should have a moderate or high glycemic index. The athlete should avoid eating meals that contain less than 70% carbohydrate, and thus have high fat and protein content, especially during the first 6 h after exercise because this often suppresses hunger and limits carbohydrate intake. Realistically, because of other daily activities including sleep, it is usually not possible to eat frequent meals (every 2 h) that contain ≥ 70% and 50 g carbohydrate. Therefore, when a person must go for an extended period between meals, their last meal should contain enough carbohydrate to suffice for that period (ie, 50 g for a 2-h period, 150 g for a 6-h period, or 250 g for a 10-h period). When sufficient carbohydrate is consumed (ie, > 8 g · kg^{-1} · d^{-1}) to maintain high day-to-day muscle glycogen stores, the intensity of training is increased and performance improves compared with when only a moderate amount of carbohydrate is consumed (5 g · kg^{-1} · d^{-1}) (70).

Maximizing muscle glycogen before competition

A few days before a prolonged and intense competitive event, athletes should regulate their diets and training in an attempt to maximize ("super compensate" or "load") muscle glycogen stores. High preexercise glycogen concentrations allow athletes to exercise for longer periods by delaying fatigue. The most practical method of "glycogen loading" involves altering training and diet for 7 d (71). On days 7, 6, 5, and 4 before competition, one should train moderately hard (eg, 1–2 h) and consume a moderately low-carbohydrate diet (ie, 350 g/d). This will make the muscle sufficiently carbohydrate deprived and ready to supercompensate, without making the person sick as sometimes occurs when all carbohydrate is eliminated. During the 3 d before competition, training should be tapered (30–60 min/d of low to moderate intensity) and a high-carbohydrate diet should be consumed (ie, 500–600 g/d; > 8 g · kg^{-1} · d^{-1}). Such a regimen will increase muscle glycogen stores ≥ 20–40% above normal. This "modified" glycogen loading regimen is as effective as the "classic" regimen (7) and is more practical because it does not require athletes to attempt to train while consuming a high-fat diet.

Preexercise nutrition

Although it is agreed that athletes should eat sufficient carbohydrate the day before exercise, there is less agreement as to when, how much, and what type of carbohydrate should be eaten during the hours before exercise.

Exercise that is limited by carbohydrate availability

The goal of a preexercise carbohydrate meal is to optimize the supply of muscle glycogen and blood glucose late in exercise. Preexercise carbohydrate meals have the following effects: *1*) promote additional muscle glycogen synthesis when stores are not already supercompensated, *2*) replenish liver glycogen and store glucose in the body (ie, intestines and glucose space) for potential oxidation during exercise, and *3*) cause increases in carbohydrate oxidation during exercise and decreases in fat oxidation.

Although these first two responses to preexercise carbohydrate feeding are beneficial because more carbohydrate is stored within the body, controversy remains as to whether the increase in carbohydrate oxidation, and concomitant decrease in fat oxidation, is advantageous or disadvantageous. It would seem disadvantageous if the increases in carbohydrate oxidation were greater than the increases in carbohydrate storage because bodily carbohydrate stores would become depleted more rapidly compared with when carbohydrate is not consumed before exercise.

Sugar feedings during the hour before exercise

Eating high glycemic index carbohydrates during the hour before moderately intense exercise (60–75%$\dot{V}O_2$max) causes a decline in blood glucose concentration at the onset of exercise (12, 17). This is due to the effects of the concomitant hyperinsulinemia that increases glucose uptake by the contracting muscles at a time when liver glucose output may be reduced, thus creating an imbalance and hypoglycemia (12, 72, 73). This is usually not perceived by the individual and it does not cause muscle weakness. Preexercise hyperinsulinemia also has the long-lasting effect of reducing the release of free fatty acids from adipocytes and the rate of fat oxidation during exercise (15, 16). Thus, there is a shift in blood-borne fuels from fatty acids to glucose.

There has been much debate as to whether these processes alter muscle glycogen use. Theoretically, muscle glycogen use would be increased if the decline in fat oxidation was not offset by a proportional increase in blood glucose uptake and oxidation by muscle. The two studies that have found preexercise feedings to slightly increase muscle glycogen use also reported a relatively large decline in blood glucose concentration, which may have limited increases in muscle glucose uptake (12, 74). Several other studies have not found sugar feedings during the hour before exercise to increase muscle glycogen use, possibly because the hypoglycemia was not as pronounced or the suppression of fatty acid mobilization was not as great (75–79). More importantly, of the studies that have measured endurance performance after sugar ingestion during the hour before exercise, only one study reported a negative effect (80); four studies observed no significant effect (74, 81–83) and three studies reported improvements in performance (78, 84, 85). Therefore, there is little support for the idea that sugar ingestion before exercise impairs performance.

The notion is widespread, however, that sugar should be avoided during the hour before exercise because it causes hypoglycemia and impairs performance. Instead, it is erroneously recommended that people eat complex carbohydrate during the hour before exercise, because it is thought that complex carbohydrate promotes less of an insulin response. As discussed, this is not necessarily the case. For example, as shown in **Figure 5** and **Figure 6**, the responses to sucrose (ie, simple carbohydrate) and mashed potato (ie, complex carbohydrate) ingestion 30 min before exercise are identical in that blood glucose and insulin were equally elevated after ingestion before exercise and subsequently, blood glucose declined to equally low concentrations during exercise without eliciting any symptoms of hypoglycemia (ie, neuroglucopenia). Figures 5 and 6 also indicate that the glycemic response to rice is moderate and that adding fat, in the form of margarine, blunts the glycemic and insulinemic effect of eating potatoes. Additionally, although the blood glucose and insulin responses varied somewhat after eating 50 g of these moderate and high glycemic foods, all of the meals caused plasma glucose to decline to equally low concentrations early in moderate-intensity exer-

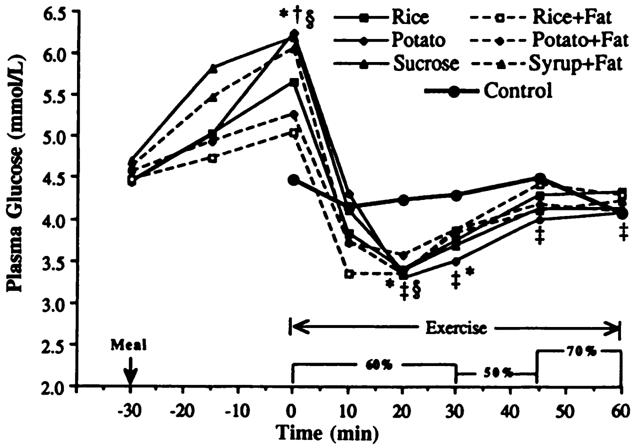


FIGURE 5. Plasma glucose concentration for the 30-min period after eating a 50-g carbohydrate meal and during the subsequent 60 min of exercise. Additionally, 35% more energy was added to the meals in the form of fat. Values are expressed as means of all subjects ($n = 9$). The meals were rice, potato, sucrose, rice and fat, potato and fat, syrup solids (corn syrup and sucrose) and fat, and control or fasting. *All trials significantly different from −30 min ($P < 0.05$). †Potato, sucrose, and syrup and fat significantly greater than rice, rice and fat, and potato and fat ($P < 0.05$). ‡All fed trials significantly less than 0 min ($P < 0.05$). §All fed trials significantly different from control ($P < 0.05$). Reproduced with permission from Horowitz and Coyle (17).

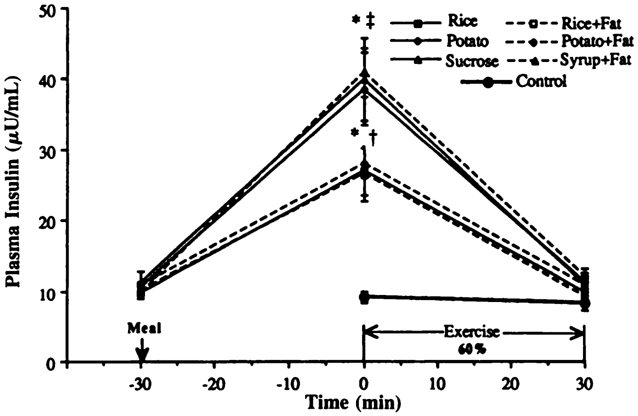


FIGURE 6. Plasma insulin concentration for the 30-min period after the meal and during the subsequent 30 min of exercise. Values are means ± SE for all subjects ($n = 9$). Conditions are the same as in Figure 5. *Significantly greater than both −30 min and 30 min ($P < 0.05$). †Significantly greater than control ($P < 0.05$). ‡Significantly greater than potato and fat, rice, rice and fat, and control ($P < 0.05$). For insulin, μU/mL × 7.175 = pmol/L. Reproduced with permission from Horowitz and Coyle (17).

cise, without affecting sensation of fatigue or ability to complete 1 hour of exercise at moderate intensities (50–70% $\dot{V}O_2$max).

Carbohydrate ingestion during the 6 h before exercise

In an attempt to avoid a decline in blood glucose at the onset of exercise, it is sometimes recommended that carbohydrate meals be eaten 3–4 h before exercise to allow enough time for plasma insulin concentration to return to basal concentrations. However, the insulin effects of a preexercise carbohydrate meal last for several hours after plasma insulin has returned to basal concentrations and thus blood glucose still declines when exercise (ie, 70% $\dot{V}O_2$max) is begun 4 h after a meal (15, 16). It appears that at least 6 h of fasting are necessary after consuming a 150-g high glycemic index meal before carbohydrate oxidation and plasma glucose homeostasis during exercise at 70% $\dot{V}O_2$max are similar to values after an 8–12-h fast (16). There is no reason, however, to recommend that people fast this long before exercise. The decline in blood glucose is not problematic (86). Actually, it can be prevented simply by having the subjects exercise slightly more intensely, which probably causes liver glucose output to increase and match blood glucose uptake by muscle (16). Additionally, the elevation in carbohydrate oxidation should not cause problems if enough carbohydrate was stored in the body as a result of the meal. When muscle glycogen is suboptimal, a substantial amount of the preexercise carbohydrate meal can be converted to muscle glycogen in a 4-h period (15, 87). Liver glycogen undoubtedly increases as well.

Accumulating evidence suggests performance is improved when a relatively large carbohydrate meal is eaten 3–4 h before prolonged exercise compared with when nothing is consumed. Neufer et al (87) reported that a 200-g carbohydrate meal of bread, cereal, and fruit eaten 4 h before exercise, as well as 43 g sucrose eaten 5 min before exercise, resulted in a 22% increase in cycling power compared with placebo. Additionally, Sherman et al (88) fed cyclists various amounts of carbohydrate 4 h before exercise and found that a 312-g feeding of maltodextrin improved power (15%) during the last 45 min of exercise. Mixed meals containing either 45 or 156 g carbohydrate did not significantly improve performance. Apparently, eating ≈150 g carbohydrate (ie, bread and juice) 4 h before exercise does not produce a marked elevation of muscle glycogen, blood glucose, or carbohydrate oxidation after 105 min exercise (15), which may explain why Sherman et al (88) did not observe an improvement in performance with this amount. Finally, Wright et al (89) reported that a 350-g feeding of maltodextrins 3 h before exercise dramatically improves performance.

A relatively large preexercise carbohydrate meal (ie, > 200 g) appears to increase performance by maintaining the ability to oxidize carbohydrates at high rates late in exercise. It is not clear if this is simply due to a greater availability of muscle glycogen. It could also be due to increased blood glucose uptake and oxidation despite the observation that blood glucose concentration is not increased (87, 88). Large preexercise carbohydrate feedings in combination with continued feedings during exercise, which do increase blood glucose concentration, produce even more dramatic improvements in performance than when carbohydrate is only eaten before exercise or when carbohydrate feedings are provided only after exercise has begun (89).

Types of carbohydrate to ingest during the 6 h before exercise

Foods ingested during the 6 h before exercise should be low in fat, fiber, and bulk and be well tolerated. If muscle glycogen stores are not supercompensated, these foods should have a high or moderate glycemic index to best stimulate synthesis. It is sometimes recommended that low glycemic foods, particularly fructose, be consumed during this period to minimize an insulin response. This would seem advisable only in situations when muscle glycogen cannot be further increased and carbohydrate feedings will not be ingested during exercise. The rationale is to make more glucose available during exercise by storing carbohydrate in the body that can be slowly absorbed as glucose during exercise. However, if more glucose is needed during exercise it makes more sense to simply ingest glucose during exercise as discussed below.

Specific recommendations

High and moderate glycemic index foods should be ingested before events that will result in fatigue due to carbohydrate depletion. It is generally recommended that ≈200–300 g carbohydrate be ingested during the 4 h before exercise. Meals should be low in fat, protein, and fiber and, of course, should not cause gastrointestinal discomfort.

Applicability to normally active people

This review has emphasized studies that used mostly endurance athletes such as cyclists and runners as subjects. It is important to note how the responses and dietary needs of these athletes may differ from normally active people. The pattern of substrate use during prolonged exercise at various intensities (Figures 1 and 2) would be generally similar in the two groups. However, at a given percentage of $\dot{V}O_2$max, normally active people oxidize less fat and more muscle glycogen compared with endurance-trained subjects (11, 90, 91). The major difference is that endurance-trained people increase their oxidation of intramuscular triglyceride and spare muscle glycogen (10, 11). Reliance on carbohydrate in general and blood glucose in particular for energy in not increased with endurance training (91). Therefore, theoretically, blood glucose supplementation should be as important to normally active people as to endurance-trained athletes. Practically, however, normally active people are less likely to perform strenuous exercise for long enough durations (> 1–2 h) to require supplementation. When they do, however, they too should benefit from carbohydrate supplementation (5).

People who exercise at relatively low intensities and for short durations (eg, < 1 h), and thus who do not substantially deplete muscle glycogen, obviously do not need to design their diet along the guidelines given for maximizing muscle glycogen resynthesis after exercise. Most notably, ingestion of high glycemic index carbohydrate will not be as important. Additionally, people who allow themselves ≥ 48 h for recovery between exercise bouts, should be able to restore muscle glycogen in that time by consuming a diet containing 60% of energy from carbohydrate, without special attention to carbohydrate type. People who perform aerobic exercise for general fitness and health are recommended to do so 3–5 d/wk (92) with rest days interspersed between training days. This allows recovery, especially of muscle glycogen. When normally active people attempt to perform strenuous aerobic exercise training with only 24 h recovery time, they should follow these guidelines, as should endurance athletes.

Summary

As the intensity of exercise is increased, the rate of plasma fatty acid mobilization declines and thus the exercising muscles become dependent on carbohydrate as a source of energy. This is not due to simply limited availability of fatty acids, but also to a limited ability for fat oxidation in skeletal muscle. Carbohydrate ingestion before and during exercise exerts a large influence on fatty acid mobilization and oxidation, making muscle even more dependent on carbohydrate for energy during exercise. The notion that fatty acid availability is limited and that treatments that increase endogenous or exogenous fatty acid concentrations are beneficial has not been well studied; therefore, carbohydrate is the recommended substrate for ingestion.

On the basis of the fact that fatigue during intense prolonged exercise is commonly due to depletion of muscle and liver glycogen, dietary practices should advocate high carbohydrate intake before, during, and after exercise. The simple goal is to have as much carbohydrate in the body as possible during the last stages of prolonged exercise when the ability for strenuous exercise usually becomes limiting to performance. This theory is put into practice by recommending that carbohydrate intake after exhaustive exercise should average 50 g per 2 hr of mostly moderate and high glycemic carbohydrate foods. The aim should be to ingest a total of ≈600 g in 24 h (> 8 g/kg body wt). Carbohydrate intake should not be avoided during the 4-h period before exercise and in fact it is best to eat ≥ 200 g during this time. When possible, carbohydrate should be ingested during exercise, generally in the form of solutions containing glucose or sucrose or maltodextrins, at a rate of 30–60 g/h. Emphasis has been placed on eating the optimal amount and best type of carbohydrate at the proper times because these practices demand a large amount of food. When diet is not carefully planned according to these guidelines, endurance athletes tend to consume too little carbohydrate because they become satiated with fat and go through periods in the day when recovery of glycogen stores is suboptimal, thus wasting precious time.

The recommendations in this paper are made for a person weighing 70 kg (154 lbs). To apply these recommendations to others, carbohydrate ingestion in grams should be calculated according to the proportion that a given person's body weight differs from 70 kg. For example, a person weighing 100 kg should multiply the recommended intake by 1.4 (ie, 100/70 kg), whereas a person weighing 50 kg should multiply the recommended intake by 0.7 (ie, 50/70 kg).

References

1. Krogh A, Lindhard J. Relative value of fat and carbohydrate as a source of muscular energy. Biochem J 1920;14:290–8.
2. Romijn JA, Wolfe RR. Effects of prolonged exercise on endogenous substrate supply and utilization. In: Lamb DR, Gisolfi GV, eds. Perspectives in exercise science and sports medicine. Vol 5. Energy metabolism in exercise and sport. Dubuque: Brown & Benchmark, 1992:207–34.
3. Coyle EF, Coggan AR, Hemmert MK, Ivy JL. Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. J Appl Physiol 1986;61:165–72.
4. Romijn JA, Coyle EF, Sidossis L, et al. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity. Am J Physiol 1993;265:E380–91.
5. Coggan AR, Coyle EF. Carbohydrate ingestion during prolonged exercise: effects on metabolism and performance. Exerc Sport Sci Rev 1991;19:1–40.
6. Bulow J, Madsen J. Influence of blood flow on fatty acid mobilization from lipolytically active adipose tissue. Pflugers Arch 1981;390:169–74.
7. Bergstrom J, Hultman E. The effect of exercise on muscle glycogen and electrolytes in normals. Scand J Clin Invest 1966;18:16–20.
8. Oasci LB, Essig DA, Palmer WK. Lipase regulation of muscle triglyceride hydrolysis. J Appl Physiol 1990;69:1571–7.
9. Hodgetts A, Coppack SW, Frayn KN, Hockaday TDR. Factors controlling fat mobilization from human subcutaneous adipose tissue during exercise. J Appl Physiol 1991;71:445–51.
10. Hurley BF, Nemeth PM, Martin WH III, Hagberg JM, Dalsky GP, Holloszy JO. Muscle triglyceride utilization during exercise: effect of training. J Appl Physiol 1986;60:562–7.
11. Martin WH III, Dalsky GP, Hurley BF, et al. Effect of endurance training on plasma free fatty acid turnover and oxidation during exercise. Am J Physiol 1993;265:E708–14.
12. Costill DL, Coyle EF, Dalsky G, Evans W, Fink W, Hoopes D. Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. J Appl Physiol 1977;43:695–9.
13. Bach AC, Babayan VK. Medium-chain tryglycerides: an update. Am J Clin Nutr 1982;36:950–62.
14. Massicotte D, Péronnet F, Brisson GR, Hillaire-Marcel C. Oxidation of exogenous medium-chain free fatty acids during prolonged exercise: comparison with glucose. J Appl Physiol 1992;73:1334–9
15. Coyle EF, Coggan AR, Hemmert MK, Lowe RC, Walters TJ. Substrate usage during prolonged exercise following a preexercise meal. J Appl Physiol 1985;59:429–33.
16. Montain SJ, Hopper MK, Coggan AR, Coyle EF. Exercise metabolism at different time intervals following a meal. J Appl Physiol 1991;70:882–8.
17. Horowitz JF, Coyle EF. Metabolic responses to preexercise meals containing various carbohydrates and fat. Am J Clin Nutr 1993;58:235–41.
18. Coyle EF, Hagberg JM, Hurley BF, Martin WH, Ehsani AA, Holloszy JO. Carbohydrate feedings during prolonged strenuous exercise can delay fatigue. J Appl Physiol 1983;55:230–5.
19. Hargreaves M, Kiens B, Richter EA. Effect of increased plasma free fatty acid concentrations on muscle metabolism in exercising men. J Appl Physiol 1991;70:194–201.
20. Ravussin E, Bogardus C, Scheidegger K, LaGrange B, Horton ED, Horton ES. Effect of elevated FFA on carbohydrate and lipid oxidation during prolonged exercise in humans. J Appl Physiol 1986;60:893–900.
21. Knudsen J. Acyl-CoA-binding protein (ACBP) and its relation to fatty acid-binding protein (FABP): an overview. Mol Cell Biochem 1990;98:217–23.
22. Ockner RK. Historical overview of studies on fatty acid-binding proteins. Mol Cell Biochem 1990;98:3–9.
23. McGarry JD, Stark MJ, Foster DW. Hepatic malonyl-CoA levels of fed, fasted and diabetic rats as measured using a simple radioisotopic assay. J Biol Chem 1978;253:8291–3.
24. Elayan IM, Winder WW. Effect of glucose infusion on muscle malonyl-CoA during exercise. J Appl Physiol 1991;70:1495–9

25. Coggan AR, Coyle EF. Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. J Appl Physiol 1987;63:2388–95.
26. Fielding RA, Costill DL, Fink WJ, King DS, Hargreaves M, Kovaleski JE. Effect of carbohydrate feeding frequency and dosage on muscle glycogen use during exercise. Med Sci Sports Exerc 1985;17:472–6.
27. Flynn MG, Costill DL, Hawley JA, et al. Influence of selected carbohydrate drinks on cycling performance and glycogen use. Med Sci Sports Exerc 1987;19:37–40.
28. Hargreaves M, Briggs CA. Effect of carbohydrate ingestion on exercise metabolism. J Appl Physiol 1988;65:1553–5.
29. Mitchell JB, Costill DL, Houmard JA, Fink WJ, Pascoe DD, Pearson DR. Influence of carbohyrate dosage on exercise performance and glycogen metabolism. J Appl Physiol 1989;67:1843–9.
30. Noakes TF, Lambert EV, Lambert MI, McArthur PS, Myburgh KH, Benadé AJS. Carbohydrate ingestion and muscle glycogen depletion during marathon and ultramarathon racing. Eur J Appl Physiol 1988; 57:482–9.
31. Slentz CA, Davis JM, Settles DL, Pate RR, Settles SJ. Glucose feedings and exercise in rats: glycogen use, hormone responses, and performance. J Appl Physiol 1990;69:989–94.
32. Below PR, Coyle EF. Fluid and carbohydrate ingestion individually benefit intense exercise lasting one-hour. Med Sci Sports Exerc 1995; 27:200−10.
33. Massicotte D, Peronnet F, Brisson G, Bakkouch K, Hilliare-Marcel C. Oxidation of a glucose polymer during exercise: comparison of glucose and fructose. J Appl Physiol 1989;66:179–83.
34. Murray R, Paul GL, Seifert JG, Eddy DE, Halaby GA. The effects of glucose, fructose, and sucrose ingestion during exercise. Med Sci Sports Exerc 1989;21:275–82.
35. Owen MD, Kregel KC, Wall PT, Gisolfi CV. Effects of ingesting carbohydrate beverages during exercise in the heat. Med Sci Sports Exerc 1986;18:568–75.
36. Coyle EF, Montain SJ. Carbohydrate and fluid ingestion during exercise: are there trade-offs? Med Sci Sports Exerc 1992;24:671–8.
37. Bjorkman O, Sahlin K, Hagenfeldt L, Wahren J. Influence of glucose and fructose ingestion on the capacity for long-term exercise in well trained men. Clin Physiol 1984;4:483–94.
38. Thomas DE, Brotherhood JR, Brand JC. Carbohydrate feeding before exercise: effect of glycemic index. Int J Sports Med 1991;12:180–6.
39. Coggan AR, Coyle EF. Metabolism and peformance following carbohydrate ingestion late in exercise. Med Sci Sports Exerc 1989;21: 59–65.
40. Coggan AR, Coyle EF. Effect of carbohydrate feedings during high-intensity exercise. J Appl Physiol 1988;65:1703–9.
41. Foster C, Thompson N, Dean J, Kirkendall D. Carbohydrate supplementation and performance in soccer players. Med Sci Sports Exerc 1986;18:S12 (abstr).
42. Muckle DS. Glucose syrup ingestion and team performance in soccer. Br J Sports Med 1973;7:340–3.
43. Simard C, Tremblay A, Jobin M. Effects of carbohydrate intake before and during an ice hockey match on blood and muscle energy substrates. Res Q Exerc Sport 1988;59:144–7.
44. Murray R, Paul GL, Seifert JG, Eddy DE. Responses to varying rates of carbohydrate ingestion during exercise. Med Sci Sports Exerc 1991;23:713–8.
45. Murray R, Eddy DE, Murray TW, Seifert JG, Paul GL, Halaby GA. The effect of fluid and carbohydrate feedings during intermittent cycling exercise. Med Sci Sports Exerc 1987;19:597–604.
46. Murray R, Siefert JG, Eddy DE, Paul GL, Halaby GA. Carbohydrate feeding and exercise: effect of beverage carbohydrate content. Eur J Appl Physiol 1989;59:152–8.
47. Neufer PD, Costill DL, Fink WJ, Kirwan JP, Fielding RA, Flynn MG. Effects of exercise and carbohydrate composition on gastric emptying. Med Sci Sports Exerc 1986;18:658–62.
48. O'Dea K, Nestel PJ, Antonoff L. Physical factors influencing postprandial glucose and insulin responses to starch. Am J Clin Nutr 1980;33:760–5.
49. Gatti E, Testolin G, Noe D, et al. Plasma glucose and insulin responses to carbohydrate food (rice) with different thermal processsing. Ann Nutr Metab 1987;31:296–303.
50. Mourot J, Thouvenot P, Couet C, Antoine JM, Krobicka A, Debry G. Relationship between the rate of gastric emptying and glucose and insulin responses to starchy foods in young healthy adults. Am J Clin Nutr 1988;48:1035–40.
51. Wursch P, Del Vedovo S, Koellreutter B. Cell structure and starch nature as key determinants of the digestion rate of starch in legume. Am J Clin Nutr 1986;43:25–9.
52. Behall KM, Scholfield DJ, Canary J. Effect of starch structure on glucose and insulin responses in adults. Am J Clin Nutr 1988;47:428–32.
53. Goddard MS, Young G, Marcus R. The effect of amylose content on insulin and glucose responses to ingested rice. Am J Clin Nutr 1984; 39:388–92.
54. Jarvis JK, Pearsall D, Oliner CM, Schoeller DA. The effect of food matrix on carbohydrate utilization during moderate exercise. Med Sci Sports Exerc 1992;24:320–6.
55. Crapo PA, Reaven G, Olefsky J. Postprandial plasma glucose and insulin responses to different complex carbohydrates. Diabetes 1977; 26:1178–83.
56. Guezennec CY, Satabin P, Duforez F, Merino D, Peronnet F, Koziet J. Oxidation of corn starch, glucose, and fructose ingested before exercise. Med Sci Sports Exerc 1989;21:45–50.
57. Jenkins DJA, Wolever TMS, Buckley G, et al. Low-glycemic-index starchy foods in the diabetic diet. Am J Clin Nutr 1988;48:248–54.
58. Jenkins DJA, Wolever TMD, Jenkins AL, Josse, RG, Wong GS. The glycemic response to carbohydrate foods. Lancet 1984;2:388–91.
59. Westphal SA, Gannon MC, Nuttall F. Metabolic response to glucose ingested with various amounts of protein. Am J Clin Nutr 1990;52: 267–72.
60. Keizer HA, Kuipers H, Van Kranenburg G, Geurten P. Influence of liquid and solid meals on muscle glycogen resynthesis, plasma fuel hormone response, and maximal physical working capacity. Int J Sports Med 1986;8:99–104.
61. Costill DL, Flynn MG, Kirwan JP, et al. Effects of repeated days of intensified training on muscle glycogen and swimming performance. Med Sci Sports Exerc 1988;20:249–54.
62. Kirwan JP, Costill DL, Mitchell JB, et al. Carbohydrate balance in competitive runners during successive days of intense training. J Appl Physiol 1988;65:2601–6.
63. Blom PC, Hostmark AT, Vaage O, Vardal KR, Maehlum S. Effect of different post-exercise sugar diets on the rate of muscle glycogen synthesis. Med Sci Sports Exerc 1987;19:491–6.
64. Ivy JL, Lee MC, Brozinick JT Jr, Reed MJ. Muscle glycogen storage after different amounts of carbohydrate ingestion. J Appl Physiol 1988;65:2018–23.
65. Reed MJ, Brozinick JT Jr, Lee MC, Ivy JL. Muscle glycogen storage post exercise: effect of mode of carbohyrate administration. J Appl Physiol 1989;66:720–6.
66. Ivy JL, Katz AL, Cutler CL, Sherman WM, Coyle EF. Muscle glycogen synthesis after exercise: effect of time of carbohydrate ingestion. J Appl Physiol 1988;65:1480–5.
67. Burke LM, Collier GR, Hargreaves M. Muscle glycogen storage after prolonged exercise: effect of the glycemic index of carbohydrate feedings. J Appl Physiol 1993;75:1019–23.
68. Mason WL, McConell G, Hargreaves M. Carbohydrate ingestion during exercise: liquid vs solid feedings. Med Sci Sports Exerc 1993; 25:966–9.
69. Costill DL, Sherman WM, Fink WJ, Maresh C, Witten M, Miller JM. The role of dietary carbohydrates in muscle glycogen resynthesis after strenuous running. Am J Clin Nutr 1981;34:1831–6.
70. Simonsen JC, Sherman WM, Lamb DR, Dernbach AR, Doyle JA, Strauss R. Dietary carbohydrate, muscle glycogen, and power output during rowing training. J Appl Physiol 1991;70:1500–5.
71. Sherman WM, Costill DL, Fink WJ, Miller JM. The effect of exercise

and diet manipulation on muscle glycogen and its subsequent utilization during performance. Int J Sports Med 1981;2:114–8.
72. Ahlborg G, Felig P. Influence of glucose ingestion on the fuel-hormone responses during prolonged exercise. J Appl Physiol 1976; 41:683–8.
73. Ahlborg G, Bjorkman O. Carbohydrate utilization by exercising muscle following preexercise glucose ingestion. Clin Physiol 1987;7: 181–95.
74. Hargreaves M, Costill DL, Fink WJ, King DS, Fielding RA. Effect of pre-exercise carbohydrate feedings on endurance cycling performance. Med Sci Sports Exerc 1987;19:33–6.
75. Hargreaves M, Costill DL, Katz A, Fink WJ. Effect of fructose ingestion on muscle glycogen usage during exercise. Med Sci Sports Exerc 1985;17:360–3.
76. Levine L, Evans WJ, Cadarette BS, Fisher EC, Bullen BA. Fructose and glucose ingestion and muscle glycogen use during submaximal exercise. J Appl Physiol 1983;55:1767–71.
77. Koivisto VA, Harkonen M, Karonen S, et al. Glycogen depletion during prolonged exercise: influence of glucose, fructose or placebo. J Appl Physiol 1985;58:731–7.
78. Gleeson M, Maughan RJ, Greenhaff PL. Comparison of the effects of pre-exercise feedings of glucose, glycerol and placebo on endurance and fuel homeostasis in man. Eur J Appl Physiol 1986;55:645–53.
79. Fielding RA, Costill DL, Fink WJ, King DS, Kovaleski JE, Kirwan JP. Effects of pre-exercise carbohydrate feedings on muscle glycogen use during exercise in well-trained runners. Eur J Appl Physiol 1987;56: 225–9.
80. Foster C, Costill DL, Fink WJ. Effects of pre-exercise feedings on endurance performance. Med Sci Sports Exerc 1979;11:1–5.
81. McMurray RG, Wilson JR, Kitchell BS. The effects of fructose and glucose on high intensity endurance performance. Research Q 1983; 54:156–62.
82. Keller K, Schwarzkopf R. Preexercise snacks may decrease exercise performance. Physician Sports Med 1984;12:89–91.
83. Devlin JT, Calles-Escandon J, Horton ES. Effects of preexercise snack feeding on endurance cycle exercise. J Appl Physiol 1986;60: 980–5.
84. Okano G, Takeda H, Morita I, Katoh M, Mu Z, Miyake S. Effect of pre-exercise fructose ingestion on endurance performance in fed men. Med Sci Sports Exerc 1988;20:105–9.
85. Peden C, Sherman WM, D'Aquisto L, Wright DA. 1 h preexercise carbohydrate meals enhance performance. Med Sci Sports Exerc 1989; 21:S59 (abstr).
86. Brouns F, Saris WHM, Beckers E. Metabolic changes induced by sustained exhaustive cycling and diet manipulation. Int J Sports Med 1989;10:S49–62.
87. Neufer PD, Costill DL, Flynn MG, Kirwan JP, Mitchell JB, Houmard J. Improvements in exercise performance: effects of carbohydrate feedings and diet. J Appl Physiol 1987;62:983–8.
88. Sherman WM, Brodowicz G, Wright DA, Allen WK, Simonsen J, Dernbach A. Effects of 4 h preexercise carbohydrate feedings on cycling performance. Med Sci Sports Exerc 1989;21:598–604.
89. Wright DA, Sherman WM, Dernbach AR. Carbohydrate feedings before, during or in combination improve cycling endurance performance. J Appl Physiol 1991;71:1082–8.
90. Coyle EF, Coggan AR, Hopper MK, Walters TJ. Determinants of endurance in well-trained cyclists. J Appl Physiol 1988;64:2622–30.
91. Coggan AR, Kohrt WM, Spina RJ, Bier DM, Holloszy JO. Endurance training decreases plasma glucose turnover and oxidation during moderate-intensity exercise in men. J Appl Physiol 1990;68:990–6.
92. American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiovascular and muscular fitness in healthy adults. Med Sci Sports Exerc 1990;22: 265–74.