

Fat Burning During Exercise: Can Ergogenics Change the Balance?

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In Brief: Endurance athletes and dieters are eager to burn more fat during exercise; athletes hope to conserve carbohydrate stores, while dieters wish to decrease fat stores. This article briefly reviews the role of fat as an energy source for physical activity, discusses how exercise intensity and duration affect fat and carbohydrate metabolism, and assesses the nutrition strategies athletes are most likely to use in attempts to promote fat burning during exercise: caffeine ingestion, L-carnitine supplements, medium-chain triglyceride supplements, and high-fat diets. Of this group, caffeine ingestion is the only strategy scientifically proven to enhance athletic performance.

In recent years, a multitude of dietary supplements and nutrition strategies have been promoted as "magic bullets" to boost fat metabolism, reduce body fat, and improve athletic performance. Though some of these substances may enhance exercise capacity and, in particular, fat metabolism, most claims are based on anecdote, testimony, and inventive marketing, rather than sound science.

The search for strategies to improve athletic performance has prompted a recent surge of interest in nutrition practices that, in theory, could promote fatty acid oxidation, slow carbohydrate utilization, and improve exercise capacity. However, most of these interventions have little or no scientific basis and should not be recommended for use by healthy individuals or athletes to improve exercise performance.

Fat as an Energy Source

Compared with the body's limited carbohydrate stores, triglyceride reserves are plentiful. In a healthy, untrained individual, between 70,000 and 100,000 kcal of energy is stored as fat, mainly in the peripheral adipocytes. Even highly trained athletes who have little adipose tissue have fat stores that far exceed their athletic requirements. Although most fat is stored in adipose tissue, endurance athletes have small but physiologically important amounts of triglyceride within muscle cells; active muscle mass may contain up to 300 g of fat, most stored within the myocyte as small lipid droplets.

As a stored source of energy, fat has an advantage over carbohydrate: the energy density is higher while the relative weight is lower. Fatty acids provide more adenosine triphosphate (ATP) per molecule than glucose. However, to produce the equivalent amount of ATP, the complete oxidation of fatty acids requires more oxygen than the oxidation of carbohydrate.

Exercise Intensity and Fuel Use

The relative contributions of fat and carbohydrate to energy vary with exercise intensity. Low-intensity activities such as walking strongly stimulate lipolysis from peripheral adipocytes, while intramuscular triglycerides contribute little or nothing to total energy expenditure (1). The rate of carbohydrate use is also low: carbohydrate needs are met predominantly by circulating blood glucose, with little or no muscle glycogen breakdown (figure 1: not shown). The rate of appearance of fatty acids into the plasma peaks during low-intensity exercise (25% to 30% of VO₂ max) and then declines as exercise intensity increases.

In contrast, the rate of fat *oxidation* is highest during moderate activity such as easy jogging (65% of VO₂ max). At such an intensity, plasma free fatty acids and intramuscular triglyceride contribute equally to the overall rate of fat oxidation. During high-intensity exercise (85% of VO₂ max), the rate of total fat oxidation falls, mainly because the appearance of fatty acids into the plasma is suppressed. At the same time, lipolysis of intramuscular triglycerides does not rise substantially when exercise intensity increases from 65% to 85% of VO₂ max. This would not affect recreational athletes because most cannot sustain high-intensity exercise for more than 10 to 15 minutes without accumulating high (greater than 10 mM) concentrations of lactic acid in the working muscles and blood, which would cause discomfort and stop activity.

When low-intensity exercise continues more than 90 minutes, the pattern of substrate metabolism changes little relative to the first 20 to 30 minutes of exercise. The same is true of moderate-intensity exercise (65% of VO₂ max): the rate of total fat or carbohydrate oxidation changes little after 2 hours of jogging or cycling at this intensity compared with the first 30 minutes. However, this level of exercise induces a progressive increase in the mobilization of fatty acids from peripheral adipocytes into the plasma (1). Therefore, the contribution of intramuscular substrates (triglyceride and glycogen) to total energy expenditure probably decreases when the duration of moderate-intensity exercise increases beyond 90 minutes.

Nutrition Tools to Change Metabolism

Endogenous carbohydrate reserves are limited, and muscle and liver glycogen depletion often coincides with fatigue during endurance events and many team sports (2). Consequently, methods that promote fatty acid oxidation and conserve carbohydrate stores might improve exercise capacity. Both endurance training and nutrition strategies are used in pursuit of this goal.

The effects of endurance training on fat metabolism are well documented: it enhances total fatty acid oxidation by increasing intramuscular triglyceride storage and maximal fatty acid flux. These processes conserve endogenous carbohydrate stores and prolong intense exercise.

As for nutrition strategies, many so-called ergogenic aids have been investigated for their potential to increase fat utilization. Among them are caffeine, L-carnitine, medium-chain triglycerides, and high-fat, low-carbohydrate diets.

Caffeine

The use of caffeine as a potential ergogenic aid is not new; the Medical Commission of the International Olympic Committee (IOC) first banned caffeine in 1962, rescinded the ban a decade later, and recently reclassified it as a restricted drug (an illegal dose is greater than 12 mg/L in urine). Most athletes consume caffeine as strong, black coffee; others take over-the-counter antidrowsiness preparations that contain caffeine.

Once ingested, orally administered caffeine is almost completely absorbed. Plasma caffeine concentration peaks about 45 to 60 minutes after a single 250-mg dose, although individuals vary in their response. Under normal ingestion regimens, it is highly unlikely that any individual could exceed the current IOC limit.

Caffeine affects almost every organ system, with the most obvious being the central nervous

system. The stimulant increases alertness, reduces perceived effort during exercise, and decreases reaction time. At high doses (more than 15 mg/kg body weight), caffeine can also produce bradycardia, hypertension, nervousness, irritability, insomnia, and gastrointestinal distress (3).

In the first study (4) of caffeine as an ergogenic aid, a single dose (5 mg/kg body weight) ingested 60 minutes before exercise increased time to fatigue by 20% during intense cycling (80% of VO₂ max) (4). Other laboratory (5) and field (6) studies confirmed the benefits of caffeine for endurance performance. The postulated mechanism for the improved exercise capacity was a rise in circulating free-fatty-acid concentration, an increase in fatty acid oxidation, and a reduction in carbohydrate utilization during exercise.

Evidence of a glycogen-sparing effect--most apparent during the early stages of exercise--has been found in every study that has determined muscular glycogen levels after caffeine ingestion (7,8). There is little doubt among scientists that caffeine positively affects fat metabolism, and that ingestion in legal quantities can improve performance in continuous, moderate-intensity exercise (submaximal exercise lasting more than 15 minutes) (3). When compared with placebo, caffeine (150 to 250 mg) has also been shown to improve 5-minute running and cycling performance in moderately or well-trained athletes who perform at or near their VO₂ max (3). In contrast, caffeine has no ergogenic effect on maximal anaerobic (sprint) events lasting less than 30 seconds or maximal graded exercise to exhaustion (9).

L-Carnitine Supplementation

Carnitine plays a central role in the metabolism of fatty acids by transporting them from the cytosol to the mitochondrial matrix for beta oxidation. Long-chain fatty acid oxidation in all tissues is carnitine dependent; therefore, hereditary and acquired carnitine deficiencies cause triglyceride to accumulate in the skeletal muscles, impair fatty acid utilization, and reduce exercise capacity. Carnitine supplementation can usually reverse these changes (10).

It has been hypothesized that carnitine supplementation in healthy people increases fatty acid transport into the mitochondria and subsequent oxidation. If this were true, supplementation would significantly benefit endurance athletes and individuals wishing to lose weight.

The normal carnitine pool in a healthy 70-kg adult is about 100 mmol; more than 98% resides in skeletal and cardiac muscle, 1.6% in the liver and kidneys, and only 0.4% in the extracellular fluid (11). More than 50% of the daily need for carnitine is normally supplied by the diet from meat, poultry, fish, and some dairy products; the rest is endogenously biosynthesized from methionine and lysine. Daily urine losses are usually less than 2% of the total body carnitine store.

Many well-controlled studies have examined the effects of carnitine supplementation on metabolism and athletic performance in moderately trained individuals (12,13) and well-trained athletes (14,15). The doses used in these studies have varied from 2 to 6 g/day, and the length of supplementation from 5 days to 4 weeks. The results of these and many other investigations (16) convincingly demonstrate that carnitine supplementation has no effect on fuel utilization at rest (12) or during exercise (12,14).

Because supplementation does not alter lipid metabolism during exercise, it is not surprising that the rate of muscle glycogen utilization does not change (15). Lactate metabolism is not reduced (12,14) and blood pH does not change during submaximal (15) or maximal (14) exercise. Even when carbohydrate availability has been compromised before exercise by reducing muscle glycogen stores, carnitine supplementation still fails to alter lipid metabolism during submaximal exercise (17).

Because of carnitine's role in fatty acid metabolism, it is not surprising that it has been targeted as a potential promoter of fat loss. Carnitine is vigorously marketed to athletes in sports that require making weight or maintaining low body fat (wrestling, rowing, gymnastics,

bodybuilding). However, there is no scientific evidence that carnitine enhances fatty acid oxidation, helps reduce body fat, or helps athletes "make weight."

Finally, many studies have shown little or no loss of carnitine from skeletal muscle during low- or high-intensity exercise (16), suggesting that training does not substantially reduce muscle carnitine levels in healthy athletes eating conventional diets. Massive doses of carnitine increase muscle carnitine levels by only 1% or 2% (18). Therefore, there is little or no reason for moderately active individuals or athletes in hard training to take carnitine supplements.

Medium-Chain Triglycerides

Medium-chain triglycerides contain predominantly fatty acids with a chain length of C6-10. Appreciable amounts of medium-chain triglycerides are not found in the diet. Compared with long-chain fatty acids, medium-chain triglycerides--when ingested with carbohydrate--are emptied very rapidly from the stomach and are absorbed almost as fast as glucose. Consequently, recent interest has focused on the potential ergogenic effect of ingesting medium-chain triglyceride solutions for endurance events.

The first investigators to compare the effects of medium-chain triglyceride ingestion to glucose during exercise (2 hours of cycling at 65% of VO₂ max) found that the contributions to total energy requirements during exercise were similar (19). A more recent study (20) combined carbohydrate with medium-chain triglyceride during 3 hours of moderate-intensity (57% VO₂ max) exercise in well-trained cyclists. About 70% of the triglyceride was oxidized when ingested with carbohydrate, compared to 33% when ingested alone. Toward the end of exercise, the rate of medium-chain triglyceride oxidation approached the rate of ingestion. Even so, the maximum contribution of ingested medium-chain triglycerides to total energy expenditure was only 7%.

In a separate study (21), the same researchers examined the effects of medium-chain triglyceride ingestion on the rates of muscle glycogen utilization during 180 minutes of moderate-intensity cycling. Ingesting medium-chain triglyceride (10 g/hr) did not affect the rate of total carbohydrate oxidation or the rate of muscle glycogen utilization. Even when subjects commenced exercise with low muscle glycogen, medium-chain triglyceride ingestion had no effect on carbohydrate utilization (21).

To date, only one study (22) has reported a beneficial effect of medium-chain triglyceride ingestion on performance. Large doses (about 30 g/hr) of medium-chain triglyceride in a carbohydrate solution improved performance 2.5% over a carbohydrate solution alone in a 40-km cycle time trial undertaken after 2 hours of moderate-intensity exercise (22). Researchers attributed the enhanced performance to the larger doses of medium-chain triglyceride used in this study relative to those used in studies that did not show an effect. The higher dose produced higher levels of fatty acids in the blood and, presumably, subsequent increased oxidation. As noted, however, this study is the exception. Moreover, ingestion of larger amounts (30 g/hr) is likely to produce gastrointestinal problems in most athletes, which would probably hurt performance.

High-Fat Diets

Altering an individual's diet 24 to 48 hours before exercise is a well-known, effective tool for modifying the patterns of substrate utilization and improving performance (2). Consuming a diet high in fat (greater than 60% of energy intake) and low in carbohydrate (less than 15% of energy) for 1 to 3 days significantly reduces resting muscle glycogen content, shifts exercise metabolism in favor of lipid oxidation, and impairs submaximal exercise capacity (23). On the other hand, some evidence suggests that longer (5- to 7-day) high-fat-diet periods may induce adaptations that "retool" the working muscle to increase its capacity for fatty acid oxidation (24).

The most frequently cited study (25) supporting the use of high-fat diets compared the effects

of 28 days of a high-fat diet (85% of energy) with those of an isocaloric high-carbohydrate diet (66% of energy) on submaximal cycle time to exhaustion. Although the high-fat diet reduced resting muscle glycogen content by 47% (143 mmol/kg of wet weight of muscle for the high-carbohydrate diet vs 76 mmol/kg of wet weight of muscle for the high-fat diet), the mean exercise time for the five subjects under investigation did not differ significantly (147 minutes for the high-carbohydrate diet, 151 minutes for the high-fat diet). These results, however, should be interpreted cautiously because one subject rode almost 60% longer after the high-fat diet, skewing the average result.

Probably the longest exposure to a carbohydrate-restricted diet was a recent investigation (26) in which two groups of 10 untrained subjects participated in a 7-week endurance program while consuming a high-fat (62% of energy) or high-carbohydrate (65% of energy) diet. Cycling time to exhaustion at 70% of VO₂ max increased by 191% after the high-carbohydrate diet, but only 68% after the high-fat diet. To determine if the performance impairment in the high-fat group could be reversed, subjects switched to a high-carbohydrate diet during the eighth week of the study and repeated the exercise task. Even after a week of ingesting carbohydrate, the mean performance time improved by only 12 minutes, leading researchers to conclude that "a combination of training and a fat-rich diet did not reveal an additive effect on physical performance (26)."

Recently, the idea of "nutritional periodization" for endurance training has been proposed. Athletes might train for most of the year on a high-carbohydrate diet, consume a high-fat diet for 2 to 3 days early in the week before a major event, then carbohydrate-load 48 hours before competition (27). Such periodization would permit endurance athletes to train hard throughout the year and maximize their endogenous carbohydrate stores before competition while, in theory, also optimizing their working muscles' capacity to oxidize fatty acids during a major race. The hypothesis requires scientific testing before any recommendation can be made to athletes.

Even if such a dietary regimen were shown to enhance performance, high-fat diets still increase the risk of a number of diseases (28,29). Though regular physical activity attenuates these risks (28), individuals should limit their long-term exposure to high-fat diets. Short-term use of high-fat diets is associated with insulin resistance in the liver (30), which results in a failure to suppress hepatic glucose output and leads to a reduction in liver glycogen synthesis. For these reasons, caution should be exercised when recommending high-fat diets to athletes.

Individualized Fueling Strategies

Athletes use many nutritional strategies to promote fat oxidation, conserve carbohydrate stores, and improve athletic performance. However, many of these practices, such as the "zone diet (31,32)," have not been rigorously tested.

Even agents shown to have an ergogenic effect when tested under well-controlled conditions may be ergolytic in certain individuals. Negative effects may not be known because there are likely to be many scientific studies that, because they lacked a positive finding, were never published. Accordingly, it is important to recognize that individuals vary in their response to ergogenic substances. Nutrition strategies require the supervision of qualified medical personnel, and should *always* be fine-tuned during daily training.

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