Introduction

The average Western diet contains approximately 45–50% carbohydrates and 35–40% fat, with approximately 15% derived from protein. Interestingly, the normal diet of endurance athletes is very similar to this, although in absolute terms their intake of carbohydrates is higher because of their higher energy expenditure.

Carbohydrate and fat are the most important energy sources for endurance exercise. Both sources are stored in the body with fat representing 92% of all endogenous stored energy and carbohydrate only about 2%. It is therefore not surprising that sport scientists, nutritionists and athletes have experimented with different diets in which the relative contribution of these nutrients has been manipulated. Whereas for many years athletes have used carbo-
Carbohydrate and fat metabolism during exercise

Carbohydrate is stored in the body in the form of glycogen in the liver and in skeletal muscle. It has been estimated that about 80–100 grams of glycogen can be found in the liver in the hours after a meal, whereas the total amount in muscle depends mainly on muscle mass and dietary carbohydrate intake. However, in muscle glycogen stores are typically around 400 grams although in trained subjects with a large muscle mass, and after a high-carbohydrate meal this may increase up to 900 grams. An overview of the various endogenous substrates is given in Table 1. These carbohydrate stores are relatively small compared to the large fat stores. Most of the fat is stored in subcutaneous adipose tissue with smaller amounts stored as intramuscular triacylglycerol.

At the onset of exercise, lipolysis in adipose tissue will be stimulated and fatty acids will be mobilized from the adipose tissue and transported to the muscle. These fatty acids can be taken up by the muscle and together with fatty acids derived from intramuscular fat sources they can be transported into the mitochondria and be oxidized. Liver glycogen breakdown will also be increased at the onset of exercise: the hepatic glucose output will increase as a function of the exercise intensity. Glucose will be transported to the muscle where it can be taken up. In the muscle glucose can be oxidized along with glucose derived from muscle glycogen. Muscle glycogen becomes the most important substrate when the exercise intensity increases.

Carbohydrate and fat are always oxidized as a mixture but the relative contribution of these two substrates is dependent on a variety of factors. These factors include the exercise intensity and duration, the aerobic capacity, the diet and what has been eaten in the hours before exercise.

1. The exercise intensity. In absolute terms, fat oxidation increases as the exercise intensity increases from low to moderate intensities even though the percentage contribution of fat may actually decrease (Fig. 1). For the transition from light to moderate intensity exercise, the increased fat oxidation is a direct result of the increased energy expenditure. At higher intensities of exercise (>75% \( \text{VO}_2 \text{ max} \)) fat oxidation will be inhibited and both the relative and absolute rates of fat oxidation will decrease to negligible values. Above approximately 65% \( \text{VO}_2 \text{ max} \) fat oxidation decreases despite high rates of lipolysis. The blood flow to the adipose tissue may be decreased (due to sympathetic vasoconstriction) and this may result in a decreased removal of fatty acids from adipose tissue. During high intensity exercise, lactate accumulation may also increase the reesterification and inhibit the oxidation of fatty acids.

2. The duration of exercise. Fat oxidation increases and carbohydrate oxidation decreases as the exercise duration increases. Typical fat oxidation rates are between 0.2 and 0.5 g·min\(^{-1}\) but values of 1.0–1.5 g·min\(^{-1}\) have been reported after 6 hours of running. The contribution of fat to energy expenditure can even increase to as much as 90%. This increased fat oxidation is likely to be caused by a reduction in muscle glycogen stores towards the later stages of prolonged exercise.

3. Level of aerobic fitness. Trained individuals can oxidize fat at higher rates both at the same absolute and relative exercise intensity. It has been demonstrated that trained individuals use more intramuscular triacylglycerols compared with untrained individuals whilst no difference has been observed in the oxidation of plasma derived fatty acids.

4. Diet. The composition of the diet has a marked effect on substrate utilization (Fig. 2). Generally a high-carbohydrate diet will increase carbohydrate oxidation and decrease whole body fat oxidation whereas a low-carbohydrate, high-fat diet will have the opposite effect. This will be discussed in more detail below.

5. Carbohydrate intake before or during exercise. Pre-exercise carbohydrate ingestion has a very strong inhibiting effect on fat oxidation. The ingestion of 50–100 grams of carbohydrate in the hour before exercise will inhibit lipolysis and will also reduce fat oxidation by about 30–40%.

The regulation of carbohydrate and fat metabolism has been discussed in great detail in a number of recent reviews [Jeukendrup et al. 1998a, b; Jeukendrup 2002; Spriet 2002].

<table>
<thead>
<tr>
<th>Substrate</th>
<th>Mass in kg</th>
<th>Energy in kJ (kcal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma glucose</td>
<td>0.02</td>
<td>328 (78)</td>
</tr>
<tr>
<td>Liver glycogen</td>
<td>0.1</td>
<td>1630 (388)</td>
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<tr>
<td>Muscle glycogen</td>
<td>0.4</td>
<td>6510 (1550)</td>
</tr>
<tr>
<td>Total</td>
<td>0.52</td>
<td>5400 (1290)</td>
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<tr>
<td>Fat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma fatty acids</td>
<td>0.0004</td>
<td>17 (4)</td>
</tr>
<tr>
<td>Plasma triacylglycerol</td>
<td>0.004</td>
<td>164 (39)</td>
</tr>
<tr>
<td>Adipose tissue</td>
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<td>420 000 (100 000)</td>
</tr>
<tr>
<td>Muscle triacylglycerols</td>
<td>0.3</td>
<td>11 100 (2616)</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>447 300 (106 500)</td>
</tr>
</tbody>
</table>

Table 1: Estimated energy stores in an 80 kg man with 15% body fat. Athletes may be somewhat leaner and may have slightly larger glycogen stores (up to about 1000 g). Adapted from Jeukendrup et al., 1998a.
**Diet and performance**

When studying the effects of diet on performance scientists have traditionally measured «performance» by having the athlete exercise in a laboratory, often on a bike or running treadmill, at a steady work rate. They measure the time the athlete can exercise until he fatigues, or fails to keep up with this work rate. Technically, this measurement should be called «exercise capacity» or «endurance». Although these studies are relatively easy to conduct and can show the beneficial effects of a dietary intervention, they do not necessarily mimic the real demands of a sporting event. After all, in most races, the athlete tries to cover a set distance as fast as possible rather than exercise until he is exhausted. Studies that try to simulate a real event, especially in the field rather than in the laboratory, are harder to conduct. However, they have also shown to give more reproducible results [Jeukendrup et al. 1996]. The most complicated types of performance belong to unpredict- able team games or sports involving complex decision-making and motor skills. It is hard to find a way to adequately measure all the components of performance, and it is complicated to organize a protocol in which the same event is conducted twice, before and after an intervention, or with a treatment and a placebo.

It is important to always keep this in mind when interpreting various dietary intervention studies in which such performance measurements were conducted.

**High-carbohydrate diets**

**High-carbohydrate diets and performance**

The link between dietary carbohydrate intake and exercise tolerance was established as early as the 1920s. Krogh and Lindhard [Krogh and Lindhard 1920] observed that a diet high in fat and low in carbohydrate reduced the respiratory exchange ratio and increased the ratings of perceived exertion during exercise. Several years later Christensen and Hansen [Christensen and Hansen 1939] reported that a high-carbohydrate diet (83% carbohydrate) for 3 to 7 days enabled subjects to exercise for 210 min whereas a high-fat diet (94% fat) reduced performance to 88 min. In the 1960s the muscle biopsy technique was redeveloped and it was found that pre-exercise muscle glycogen levels were related to fatigue and that these glycogen levels could be influenced by the diet (Bergström et al. 1967; Hultman and Bergström 1967; Hultman 1967). From these early studies the so-called supercompensation diet developed. This regime included both diet and exercise guidelines to obtain higher than normal (supercompensation) muscle glycogen concentrations and resulted in glycogen stores up to twice the normal resting level (e.g. 500–900 mmol·kg⁻¹ dw). A series of studies indicated that several days of a low carbohydrate diet depleted muscle glycogen stores and reduced cycling endurance compared with a normal carbohydrate diet. However, following up with a high carbohydrate intake over several days caused a supercompensation of muscle glycogen stores and prolonged the cycling time to exhaustion. These pioneering studies resulted in the development of the «classical» seven-day model of carbo-loading. This model consists of a 3–4 day «depletion» phase of hard training and low carbohydrate intake, finishing with a 3–4 day «loading» phase of a high-carbohydrate diet and exercise taper. Early field studies of prolonged running events showed that carbohydrate loading enhanced sports performance, not by allowing the athlete to run faster, but by prolonging the time that race pace could be maintained.

This extreme diet and exercise regime had several disadvantages: athletes developed gastrointestinal problems, especially after the high-fat, low-carbohydrate feeding. Without adequate carbohydrate intake they did not recover from the hard training sessions the week before and the regime did not allow them to perform their normal tapering or training in the week before competition. Also, since every gram of glycogen is stored with 3–5 grams of water, glycogen loading can result in a 2–3% increase in body mass.

Further studies undertaken on trained subjects have produced a «moderate» carbo-loading strategy. Carbo-loading may be seen as an extension of «fuelling up» – involving rest/taper and high carbohydrate intake over 3–4 days [Sherman et al. 1981; Sherman 1983]. The moderate supercompensation protocol offers a more practical strategy for competition preparation, by avoiding the fatigue and complexity of the extreme diet and training protocols associated with the previous depletion phase. Muscle glycogen concentrations after this «moderate» carbo-loading protocol are almost as high as those obtained by the extreme «classical» loading protocol.

We recently studied the effects of high-carbohydrate, low-fat diets on metabolism and performance [Coyle et al. 2001]. Eight well-trained cyclists received a diet containing 88% carbohydrate (901 g·day⁻¹). They trained for 2 hours a day at 70% VO₂ max for 7 days expending approximately 8.2 MJ (2000 kcal) in each training session. After 7 days on this exercise and diet protocol muscle glycogen concentrations were extremely high and in fact among the highest values ever reported (824 mmol·kg⁻¹ dw). Fat oxidation during exercise was reduced by 27% by the high-carbohydrate low-fat diet and this was partly attributed to reduced intramuscular triacylglycerol stores. This may demonstrate that dietary fat may have an important role to play after exercise, in that it helps to restore intramuscular fat stores.

From this and other studies it can be concluded that with adequate refueling strategies it is possible to restore muscle glycogen in less than 24 hours without employing special «supercompensation protocols». Such strategies take the timing and amount of carbohydrate intake into account and have been reviewed recently by Jenjens and Jeukendrup [Jentjens and Jeukendrup 2003]. It is likely that this rapid muscle glycogen resynthesis can only be found in well-trained athletes. Trained individuals have a higher concentration of the glucose transporter GLUT-4 and GLUT-4 may be more insulin sensitive [Hickner et al. 1997]. This means that more GLUT-4 can be found at the sarcolemmal membrane in trained individuals increasing glucose transport and glycogen synthesis [Jentjens and Jeukendrup 2003].

Interestingly, performance in a time trial performed after 2 hours of exercise at 70% VO₂ max was not affected by the carbohydrate content of the diet [Coyle et al. 2001]. A diet containing 58%, 68% or 88% carbohydrate produced the same performance results. The carbohydrate intake in these diets ranged from 581 g·day⁻¹ to 901 g·day⁻¹ and it may be suggested that the lowest carbohydrate intake in this study was sufficient to restore muscle glycogen concentrations to a level that is sufficient to perform well. In addition it is likely that larger glycogen stores are broken down more rapidly as it has been demonstrated repeatedly that the rate of glycogenolysis is directly related to muscle glycogen concentration [Laurent et al. 2000].

In a recent review, Hawley et al. [Hawley et al. 1997] reported that carbo-loading will typically postpone fatigue in exercise lasting 90 min or longer and extend the duration of steady state exercise by ~20%, and improve performance over a set distance or workload by 2–3%. It should probably be added that the ingestion of more than 600 g·day⁻¹ (8 g·kg⁻¹ body mass per day) might result in increased glycogen stores but will not further affect performance.

**Health effects of a high-carbohydrate diet**

Since the 1960s athletes often ingest diets extremely high in carbohydrate and very low in fat. In sedentary people it has been demonstrated that high-carbohydrate low-fat diets are associated with increases in fasting plasma triacylglycerols and decreased HDL-cholesterol concentrations [Mensink and Katan 1992]. According to epidemiological evidence the latter alterations may be expected to increase the risk of coronary heart disease (CHD) [Hokanson and Austin 1996]. A high-carbohydrate diet also causes postprandial lipaemia which has also been shown to pose a significant increase in atherogenic risk [Paté et al. 1992]. One of the possible causes of the lipaemia may be de novo lipogenesis. We
investigated the effect of reducing the fat intake to very low levels on indicators of lipogenesis in physically active humans who are in energy balance [Jeukendrup et al. 2001]. For this purpose, training and diet of 9 well-trained athletes were strictly controlled for 20 days. Training consisted of 2 h/day at 64% VO_max for 20 days. For 10 days, a control diet (control: 35% fat, 15% protein and 50% carbohydrate) was provided, followed by a 10-day low-fat diet (low-fat diet: 5% fat, 15% protein and 80% carbohydrate). Subjects were kept weight stable and in 24 h energy balance in respiration chambers. Fat oxidation was measured at rest and during exercise. Before and at the end of each diet period whole-body density was measured by densitometry. All subjects were in energy balance and no significant changes were observed in body mass, lean body mass, or fat mass during the entire experimental period. During exercise fat oxidation was reduced by 40% (83 ± 6 vs. 51 ± 5 g·h⁻¹). A negative fat balance was observed with both diets but was significantly greater with the low-fat diet (−89 ± 10 g·24 h⁻¹) compared to control (−43 ± 14 g·24 h⁻¹). These data suggest that physically active people display a relatively high rate of de novo lipogenesis when consuming a low fat diet. Increased plasma triacylglycerol concentrations were also observed in this study. This, however, seems to be in contrast with a study by Koutsari et al. [Koutsari and Hardman 2001] who observed that daily exercise (30 minutes of moderate exercise) could prevent the augmentation of postprandial lipaemia attributable to a short term high-carbohydrate diet. The different results can possibly be explained by the differences in energy expenditure and intake and the fact that subjects in the former study were in a very large negative fat balance.

Therefore whether or not a high-carbohydrate diet will result in increased plasma triacylglycerol concentrations may depend on the absolute amount of carbohydrate rather than the percentage of carbohydrate in the diet. If more carbohydrate is ingested than can be stored as glycogen, it is likely to be converted to fat and this may result in increased triacylglycerol synthesis. However, it must be noticed that epidemiological studies on cardiovascular disease risk factors are generally carried out in relatively sedentary populations and those results may not necessarily translate to the athletic population. If postprandial lipaemia is also a risk factor in well-trained athletes is currently not known.

Summary high-carbohydrate diets

In the absence of muscle damage, muscle glycogen stores can be returned to normal resting levels (to 350–800 mmol·kg⁻¹·dw) with 24 hours of rest and an adequate carbohydrate intake (7–10 g per kilogram body mass per day). Normalized stores appear adequate for the fuel needs of events of less than 60–90 minutes in duration – for example, a half-marathon or basketball game. In fact, supercompensated glycogen levels do not enhance the performance of these events. Extremely high-carbohydrate intakes may result in increased risk factors for cardiovascular diseases.

High-fat diets

Historical overview: High-fat diets

Observations at the beginning of the century in inhabitants of Greenland and their dogs, suggested that high-carbohydrate diets are not always necessary to function «normally». The diet of the Greenland natives was very high in fat, but their exercise capacity did not seem to differ much from people in Western countries who consumed a mixed or high-carbohydrate diet. This led researchers to suggest that there may be an adaptation to such a diet. Initially, studies were performed in rats that were fed a high-fat diet for 2–7 weeks. It has been shown that in rats endurance capacity is very much improved after adaptation to a high-fat diet [Conlee et al. 1990; Miller et al. 1984; Simi et al. 1991]. Miller et al. [Miller et al. 1984] demonstrated that already after 1 week on a high-fat diet, the endurance of rats was increased by 8% and after 5 weeks by 33% compared with a normal diet. Another example of improved endurance after a high-fat diet is depicted in Figure 3. These adaptations have been attributed to the increased concentration of oxidative enzymes and a decreased degradation of a «sparing» of glycogen during exercise. The results suggest that after adaptation to a high-fat diet, the capacity to oxidize fatty acids is increased, because of an adaptation of the oxidative enzymes in the muscle cell. Such changes are similar to those observed after endurance training.

In humans, however, these effects are less clear. Early studies reported that adaptation to a high-fat diet for 4–6 weeks resulted in increased fat oxidation and a maintenance of endurance capacity [Phinney et al. 1983a; Phinney et al. 1980]. However, these studies are difficult to interpret because of the small subject numbers and the variation in the results. In the study by Phinney et al. [Phinney et al. 1983a], for example, endurance capacity was measured at 62–64% VO_max in only 5 subjects. On average, there was no change in performance but there was enormous variation amongst individuals with one subject showing a 36% decrease in performance and another subject an unphysiological 57% improvement! Nevertheless, it is remarkable that performance was not reduced in all subjects even though muscle glycogen levels measured before exercise were decreased by almost 50% and fat oxidation during exercise was markedly increased. Interestingly, the high-fat diet seemed to induce similar adaptations to those observed in rats, indicating an increased oxidative capacity of the muscle. Studies have demonstrated increases in β-hydroxy-acyl-CoA-dehydrogenase (HAD) activity, carnitine palmitoyl transferase 1 (CPT I) activity [Fisher et al. 1983], fatty acid binding protein content in the sarcoclemna (FABPpm) [Kiens and Helge 1998] and decreases in hexokinase activity [Fisher et al. 1983] after 4–7 weeks adaptation to a high-fat diet. These changes suggest an increased capacity to oxidize fatty acids after such an adaptation period. It has been demonstrated that adaptation to a high-fat diet will lead to measurable changes in the capacity to store, mobilize, transport and oxidize fat.

High-fat diets and endurance performance

There is much controversy about the effect of dietary fat for prolonged periods of time on physical performance in humans. Studies have reported improved endurance capacity after 1–2 weeks on a high-fat diet [Lambert et al. 1994; Muioio et al. 1994], no change after 4 weeks adaptation [Phinney et al. 1983b; Helge et al. 1998a; Pogliaghi and Veicsteinas 1999; Burke et al. 2002; Carey et al. 2001] or a worsened performance after 2–7 weeks [Helge et al. 1996; Pruett 1970]. Studies with rats seem to produce a more consistent picture, in that the vast majority of the studies have shown a positive effect of a high-fat diet on exercise capacity. However, a recent study suggests that the discrepancy between rat and human studies may be related to the very extreme diets used in rats (typically <1% carbohydrate and >75–85% fat, whereas in

![Figure 3: Effects of a high-fat diet and high-carbohydrate diet (CHO) on endurance capacity in trained rats. The bars represent the number of rats that could maintain the same running pace. Reconstructed from Simi et al., 1991.](image-url)
humans high-fat diets range from 40–70% fat). With a slightly higher carbohydrate content in the rat diet, no effects on running capacity were found [Helge et al. 1998b].

Muuo et al. [Muuo et al. 1994] examined the effects of three diets with relatively small differences in the fat content for 7 days in 6 runners. The contribution of fat in these diets was 15%, 24% and 38%, the contribution of carbohydrate was 50%, 61% or 73%, and protein contributed 12–14%. The authors claimed that the «high-fat» diet (38% fat) increased VO_{2}max and running time to exhaustion. The authors suggested that the mechanism by which performance was improved was through increased β-oxidation and fatty acid oxidation. This study, however, has been criticized for methodological flaws [Jeukendrup et al. 1998c; Coyle and Hodgkinson 1999]. Besides that, the fact that no differences in fat oxidation (respiratory exchange ratio) were observed between the diets, makes these results difficult to interpret. Lambert et al. [Lambert et al. 1994] fed 5 endurance-trained cyclists 14 days a somewhat less extreme high-fat diet or high-carbohydrate diet. The high-fat diet contained 67% fat and 7% carbohydrate whereas the high-carbohydrate diet contained 74% carbohydrate and 12% fat. Although muscle glycogen concentrations were 44% lower after the high-fat diet, time to exhaustion at 62% VO_{2}max was significantly longer (6028 min versus 4357 min). However, 43 min seems a very poor performance for an intensity of 62% VO_{2}max in well-trained (80 ± 8) VO_{2}max was significantly improved in both groups. The improvement by 11% in both diet groups, while also time to exhaustion at 82% VO_{2}max was increased from 65 min in the high-fat group. The authors therefore concluded that competitive endurance athletes can perform intense interval training (respiratory exchange ratio) were observed between the diets, are suboptimal. A period of adaptation to a high-fat diet followed by acute carbohydrate feeding might theoretically induce the enzymatic adaptations in the muscle while pre-exercise glycogen stores remain. If the high glycogen levels would be accompanied by a slightly lower rate of glycogenolysis an improvement in exercise capacity would be expected. Indeed, in rats it was shown that, after 3–8 weeks of adaptation to a high-fat diet (0–25% carbohydrate) followed by three days of carbohydrate feeding (70% carbohydrate), muscle and liver glycogen stores were restored to very high levels [Lapachet et al. 1996].

In humans, Helge et al. [Helge et al. 1996] studied trained cyclists who, after 7 weeks of adaptation to a high-fat diet (62% fat, 21% carbohydrate), changed to a high-carbohydrate diet (65% carbohydrate, 20% fat) for one week. A control group followed a high-carbohydrate diet for 8 weeks. Although exercise time to exhaustion increased from week 7 to 8 in the group that received a high-fat diet followed by the high-carbohydrate diet, performance was less compared to the group that received the high-carbohydrate diet for 8 weeks (Fig. 4). Because switching to a high-carbohydrate diet after 7 weeks of a high-fat diet did not reverse the negative effects, these authors concluded that the negative effects of 7 weeks high-fat diet on performance are not simply due to a lack of carbohydrate as a fuel, but rather due to suboptimal adaptations to the training.

Burke et al. [Burke et al. 2002] investigated a group of trained cyclists and fed them a high-fat diet for a relatively short period (5 days) followed by a day of carbohydrate loading on day 6. On day 7, substrate oxidation during exercise was measured followed by a test in endurance. The results show increased fat oxidation and a trend towards performance improvement with the high-fat diet but this did not reach statistical significance. Burke et al. [Burke et al. 2002] showed that a high-fat, low-carbohydrate diet resulted in increased fat oxidation rates during exercise even after a day on a high-carbohydrate diet. The one-day with high carbohydrate intake replenished glycogen stores in both conditions and muscle glycogen concentrations were identical. Yet there were large differences in substrate utilization between the two diets. Respiratory exchange ratio (RER) changed from 0.90 to 0.82 after 5 days on a high-fat diet. After consuming a high-carbohydrate diet for one day RER was still lower compared to baseline values (0.87). Since these changes were not caused by alterations in substrate availability, they are likely to be related to metabolic adaptations in the muscle.

However, despite changes in substrate utilization there was no demonstrable effect on performance.

Some have argued that performance tests used in most studies have been inadequate to investigate the effects of high-fat diets because such diets are more likely to have effects during very prolonged ultra-endurance performance. To investigate this question, Carey et al. [Carey et al. 2001] studied seven competitive athletes who were fed a high-fat or a high-carbohydrate diet for 6 days followed by one day of carbo-loading. Performance was

**Figure 4:** Effect of a high-fat or high-carbohydrate (CHO) diet on endurance capacity in athletes who were engaged in a training program. Data from Helge et al., 1998a, 1996.

**High-fat diets followed by carbo-loading**

Although chronic high-fat diets induce persistent enzymatic adaptations in skeletal muscle which favour fat oxidation, the effects on performance may not be visible because muscle glycogen levels

![Endurance capacity](image-url)
measured in a one hour time trial after 4 hours of cycling at 65% VO2max. Again, fat oxidation was increased but no effect on performance could be found.

**Health effects of high-fat diets**

From a health perspective, eating large amounts of fat has been associated with the development of obesity and cardiovascular disease. If this is also true for athletes who are physically active and have a greater capacity to oxidize fat, has yet to be determined. It has recently been demonstrated that diet composition does not affect the short-term changes in blood lipids and lipoproteins, normally associated with a high-fat diet, in endurance athletes who are in training.

Exposure to high-fat diets has also been associated with insulin resistance. Again, there is little information about trained individuals and it is not clear whether the same relationship can also be found in trained athletes. In a recent study by Staudacher et al. (Staudacher et al. 2001) it was concluded that despite marked changes in the pattern of substrate oxidation during submaximal exercise, short-term adaptation to a high-fat diet did not alter whole-body glucose tolerance or an index of insulin sensitivity in highly-trained individuals. However, the duration of this study was relative short with 6 days and the long-term effects of high-fat diets in athletes are currently unknown.

Because there is limited information about the negative effects of high-fat diets for athletes and the effects of these diets on performance are unclear, we suggest that caution should be exercised when recommending high-fat diets to athletes.

**Summary of high-fat diets**

In conclusion, chronic diets can have marked effects on metabolism. These effects seem only partly related to the effects of diets on substrate availability. Adaptations at the muscular level that result in changes in substrate utilization in response to a diet may occur already after 5 days. The potential benefits of an adaptation period to a high-fat diet followed by a period of carbohydrate loading are not clear, but the vast majority of studies reports no effect on performance. Therefore there is currently very little or no evidence to support the use of high-fat diets and long-term health effects of such diets in athletes are unknown.

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