Enhanced endurance in trained cyclists during moderate intensity exercise following 2 weeks adaptation to a high fat diet

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Abstract. These studies investigated the effects of 2 weeks of either a high-fat (HIGH-FAT: 70% fat, 7% CHO) or a high-carbohydrate (HIGH-CHO: 74% CHO, 12% fat) diet on exercise performance in trained cyclists (n=5) during consecutive periods of cycle exercise including a Wingate test of muscle power, cycle exercise to exhaustion at 85% of peak power output [90% maximal oxygen uptake (VO₂max)], high-intensity exercise (HIE)] and 50% of peak power output [60% VO₂max, moderate intensity exercise (MIE)]. Exercise time to exhaustion during HIE was not significantly different between trials, nor were the rates of muscle glycogen utilization during HIE different between trials, although starting muscle glycogen content was lower [81 g (SEM 3.9) vs 120 g (SEM 3.8) mmol kg⁻¹ wet mass, P<0.01] after the HIGH-FAT diet. Despite a lower muscle glycogen content at the onset of MIE [32 g (SEM 7) vs 73 g (SEM 6) mmol kg⁻¹ wet mass, HIGH-FAT vs HIGH-CHO, P<0.01], exercise time to exhaustion during subsequent MIE was significantly longer after the HIGH-FAT diet [79.7 g (SEM 7.6) vs 42.5 g (SEM 6.8) min, HIGH-FAT vs HIGH-CHO, P<0.01]. Enhanced endurance during MIE after the HIGH-FAT diet was associated with a lower respiratory exchange ratio [0.87 (SEM 0.03) vs 0.92 (SEM 0.02), P<0.05], and a decreased rate of carbohydrate oxidation [1.41 (SEM 0.70) vs 2.23 (SEM 0.40) g CHO min⁻¹, P<0.05]. These results would suggest that 2 weeks of adaptation to a high-fat diet would result in an enhanced resistance to fatigue and a significant sparing of endogenous carbohydrate during low to moderate intensity exercise in a relatively glycogen-depleted state and unimpaired performance during high intensity exercise.

Key words: High-fat diet – Carbohydrate – Fat metabolism – Exercise performance – Fatigue

Introduction

There is evidence that adaptation to a high carbohydrate diet prior to exercise (Bergstrom et al. 1967; Saltin and Karlsson 1971) and carbohydrate ingestion during exercise (Coggan and Coyle 1991) enhances sub-maximal endurance performance (of more than 2 h duration) during exercise at a moderately-high intensity (more than 70% maximal oxygen consumption, VO₂max). Moreover, short-term exposure to diets which are very low in carbohydrate has been shown to impair resistance to fatigue during exercise of both high and low intensity (Bergstrom et al. 1967; Christensen and Hansen 1939; Gübo et al. 1979; Maughan and Poole 1981). As a result, endurance athletes are routinely advised to ingest high carbohydrate diets (more than 70% by energy) during training, and to ingest carbohydrate during competition.

In practice, however, it has been found that the habitual diets of endurance-trained athletes suggest that the nutrient composition of these diets is seldom in excess of 55% carbohydrate by energy (Coetzee et al. 1993; van Erp-Baar et al. 1989). Moreover, carbohydrate loading prior to exercise does not result in a sparing of endogenous carbohydrate stores during exercise, but only "spares fat" (Bosch et al. 1993). Similarly, it has been found that it does not spare muscle glycogen stores (Bosch et al. 1994; Coyle et al. 1986) and carbohydrate ingestion during exercise only slows the rate of liver glycogen breakdown (Bosch et al. 1994).

Under euglycaemic conditions, the rate at which ingested carbohydrate can be utilized by the working muscle has appeared to be limited to 1 g min⁻¹ (Hawley et al. 1994). Hence, despite the ingestion or infusion of carbohydrate, it has been shown that fatigue develops as the rate of fat oxidation during prolonged exercise continues to rise with increasing exercise duration to compensate for declining muscle glycogen utilization (Bosch et al. 1993; Coggan and Coyle 1987; Hawley et al. 1994). A number of attempts have therefore been made to...
increase the availability of free fatty acids (FFA) for oxidation during exercise, in order to decrease the rates of muscle glycogen depletion (Costill et al. 1977; Hargreaves et al. 1991; Jenkins et al. 1988; Rennie et al. 1976). However, the procedures involved in artificially increasing plasma FFA concentrations are too invasive to be of any practical value to athletes. Accordingly, others have examined whether chronic exercise (greater than or equal to 4 weeks) to a high fat diet might increase the capacity of the muscle to oxidize fat and thereby, spare muscle glycogen utilization during exercise (Conlee et al. 1990; Miller et al. 1984; Phinney et al. 1983; Simi et al. 1991). Studies have shown that adaptation to a high-fat diet in rats (Conlee et al. 1990; Miller et al. 1984; Simi et al. 1991) and dogs (Hammet et al. 1977; Kronfeld et al. 1973) causes a marked improvement in endurance during exercise of moderate-to-high intensity lasting from 60 to 90 min (or approximately 120 min when rats were carbohydrate-fed after fat adaptation, when compared to exercise after adaptation to a high carbohydrate diet only).

Similar studies in humans ingesting a high (more than 60% by energy) fat diet for more than 2 weeks have demonstrated that submaximal endurance performance (less than 70% maximum) is unaffected in exercise lasting between 2.5 to 3 h despite apparently much lower rates of carbohydrate oxidation (Phinney et al. 1983; Pruett 1976). However, the gaseous fuels of fuel utilization in the study by Phinney et al. (1983) cannot be reliably determined because the extremely low (less than 2% by energy) carbohydrate content of the diets induced a marked ketonemia. Significant rates of ketone body production and oxidation may lower the respiratory exchange ratio (Schartz and Ravussin 1980), therefore, underestimating carbohydrate oxidation, and thus, may preclude the use of gas exchange to calculate the relative rates of fat and carbohydrate oxidation.

Therefore, the aim of the present study was to determine whether adaptation to a high-fat diet, sufficient to alter muscle carbohydrate concentrations and rates of carbohydrate and fat oxidation during exercise, but not sufficient to induce ketosis, influences the patterns of fuel utilization and performance during high- and low-intensity submaximal exercise in endurance-trained human athletes.

Methods

Subjects. Five endurance-trained male cyclists volunteered for the study. All the subjects were cycling between 100 and 120 km each week, and had a mean VO_{2max} of 7.2 l/min (SEM 0.4 l/min). Individual VO_{2max} values and other subject characteristics are given in Table 1. Each subject was familiarized with the nature of the study and informed, written consent was obtained prior to the trials. All experimental procedures were approved by the Ethics and Research Committee of the Faculty of Medicine of the University of Cape Town Medical School.

Measurement of VO_{2max} and peak sustained power output. The VO_{2max} and peak sustained power output (PPO) were determined during a progressive exercise test performed on a Godart electronically braked cycle ergometer (Bihoven, Holland) modified with toe clips and racing handle bars. Each subject began cycling at an exercise intensity of 3.3 W kg^{-1} body mass and this was increased by 5 W after the first 15 s. Thereafter, the exercise intensity was increased by 25 W every 15 s until the subjects felt exhausted. Exhaution was defined as more than a 10% reduction in pedalling frequency and/or an inability to maintain the same exercise intensity. The PPO was determined according to the procedure of Knappe et al. (1985) as modified by Hawley and Noakes (1992).

PPO (r)x second intensity completed (W) + [r (150°) x 25 W]

where r is the number of seconds completed in the final exercise intensity.

During the PPO estimations, expired gas was sampled continuously for the determination of oxygen consumption (VO_{2}) and carbon dioxide production (VCO_{2}). The subjects inspired air through a Hans Rudolph 2700 one-way valve (Vacumed, Ventura, Calif.) connected to a Minihart dry gas meter. Expired gas was passed via a condensation coil through a 154 baffled mixing chamber and an Amico 50554 O_2 and CO_2 analysers (Thermo Instruments, Pittsburgh, Pa.). Prior to each test, analyser were calibrated using room air and analytical grade gases of known composition (16% O_2, 5% CO_2 and 79% N_2). The gas meter was calibrated with a 5-l syringe. Instrument outputs were processed by an on-line IBM personal computer which calculated inspired ventilation (V_i, in litres per minute), VO_{2} and VCO_{2} using conventional equations (Costill et al. 1983). Metabolic rates of VO_{2} were used to adjust the exercise intensities in the subsequent performance trials described below.

Dietary manipulations. Prior to the performance trials, subjects underwent two randomly-assigned, 2-week periods of either a high fat diet (70% fat by energy, HIGH FAT) or an equal energy, high carbohydrate diet (50% by energy, HIGH CHO), separated by a minimum of 2 weeks of ad libitum or normal diet. The HIGH FAT consisted of 67.3 (SEM 1.8)% fat, 14.1 (SEM 2.1)% carbohydrate and 25.5 (SEM 2.3)% protein by energy. The HIGH CHO comprised 73.6 (SEM 5.0)% carbohydrate, and 12.0 (SEM 6.7)% fat, and 13.5 (SEM 6.0)% protein by energy. To aid adherence to dietary manipulations, the appropriate foods were purchased for the subjects and palatable menus were provided.

During the 2 weeks of dietary manipulation preceding the exercise performance trials, the subjects were asked to maintain regular training, but to refrain from training on the day preceding the performance trials. In general, the dietary manipulation was well-tolerated, although subjects reported individual and day-to-day variation in their ability to maintain the amount of training and its intensity. All performance tests were conducted in the morning, after an overnight fast.

Experimental trials. Upon arrival at the laboratory, the subjects were weighed and skinfold thicknesses were measured at the determination of body composition (Durnin and Womersley 1974).
Effects of dietary manipulation on maximal muscle power

There was no effect of nutrient composition of the diet on muscle power. Estimated \( V_{\text{max}} \) and \( P_{\text{max}} \) during 5+ cycle exercise periods at workloads ranging from 20 to 70 N, and maximal power output during a 30 s Wingate test were similar after 2 weeks of either HIGH FAT or HIGH CHO, under conditions of either dietary fat or dietary carbohydrate adaptation (Table 2).

Table 2. Basal muscle volume (\( V_{\text{m}} \)) and force of contraction (\( F_c \)) values and maximal power output measured during the 30 s Wingate test in response to 2 weeks of either high-fat (HIGH FAT) or high-carbohydrate (HIGH CHO) feeding in trained cyclists

<table>
<thead>
<tr>
<th>Test</th>
<th>( V_{\text{m}} ) (L)</th>
<th>( F_c ) (Nm)</th>
<th>Power output (W)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIGH FAT</td>
<td>124</td>
<td>36.5</td>
<td>422</td>
</tr>
<tr>
<td>HIGH CHO</td>
<td>126</td>
<td>37.7</td>
<td>424</td>
</tr>
</tbody>
</table>

In contrast, the strength muscle glycogen content was significantly lower in the HIGH-FAT trial than in the HIGH-COM trial.

Table 3. Effects of dietary manipulation on 2-min Wingate test and oxygen uptake, lactate concentrations, and performance test

<table>
<thead>
<tr>
<th>Test</th>
<th>( V_{\text{m}} ) (L)</th>
<th>( F_c ) (Nm)</th>
<th>Oxygen uptake (\mu mol\cdot min^{-1})</th>
<th>Lactate concentration (\mu mol\cdot L^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIGH FAT</td>
<td>n=5</td>
<td>n=5</td>
<td>462.2</td>
<td>128.9</td>
</tr>
<tr>
<td>HIGH CHO</td>
<td>n=5</td>
<td>n=5</td>
<td>463.9</td>
<td>127.7</td>
</tr>
</tbody>
</table>

Respiratory exchange ratio

<table>
<thead>
<tr>
<th>Test</th>
<th>( V_{\text{m}} ) (L)</th>
<th>( F_c ) (Nm)</th>
<th>Oxygen uptake (\mu mol\cdot min^{-1})</th>
<th>Lactate concentration (\mu mol\cdot L^{-1})</th>
</tr>
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<td>n=5</td>
<td>n=5</td>
<td>463.9</td>
<td>127.7</td>
</tr>
</tbody>
</table>
Fig. 1. Muscle glycogen content before and after cycling exercise at 85% of peak power output following 2 weeks of dietary manipulation, ingesting a HIGH-FAT (6) or HIGH-CHO (2) diet. Initial glycogen content was significantly lower after 2 weeks of a HIGH-FAT diet (P<0.01) when compared to 2 weeks of adaptation to a HIGH-CHO diet, although the rate of muscle glycogen depletion with exercise was not different among trials.

The HIGH-CHO trial (68±3 SEM 3.9) vs 120±6 (SEM 3.8) mmol·kg⁻¹·h⁻¹ wet mass, P<0.01, Fig. 1), and remained lower at exhaustion. At exhaustion, in the HIGH-CHO trial, muscle glycogen content was the same as the starting muscle glycogen content in the HIGH-FAT trial (Fig. 1). The overall rates of muscle glycogen utilization during HIE in the HIGH-CHO and HIGH-FAT trials were similar (4.9 (SEM 1.1) vs 5.3 (SEM 0.9) mmol·kg⁻¹·min⁻¹, respectively).

Effects of dietary manipulation on metabolism and endurance during subsequent moderate intensity exercise

Despite the much lower starting leg muscle glycogen content in the subjects on the HIGH-FAT trial than in the subjects on the HIGH-CHO diet (Fig. 1), the mean exercise time to exhaustion during moderate intensity exercise (MIE) was nearly two-fold greater after HIGH-FAT than after HIGH-CHO (79±7 (SEM 7.6) min vs 42±5 (SEM 6.4) min, P<0.01, Table 4). This improvement in endurance was associated with a lower V̇O₂ and R, as well as lower calculated rates of carbohydrate oxidation after HIGH-FAT. While approximately 2.2±3 (SEM 0.14) g of carbohydrate were oxidized each minute during steady-state MIE during the HIGH-CHO trial, only 1.4±1 (SEM 0.6) g of carbohydrates were oxidized per minute during the HIGH-FAT trial.

Blood metabolite concentrations during the successive periods of HIE and MIE

Plasma glucose concentrations during the HIGH-FAT and HIGH-CHO trials were not significantly different. At no time during either the HIE or MIE periods did the mean plasma glucose concentration fall below 3.9 mmol·l⁻¹ (Fig. 2).

There were also no differences in the change in blood lactate concentration during HIE and MIE between the HIGH-FAT and HIGH-CHO trials. Blood lactate concentrations in both HIE trials increased to over 10 mmol·l⁻¹ at exhaustion (Fig. 2). However, the rate of reduction in blood lactate concentrations during the 20-min rest following HIE was greater after HIGH-FAT than after HIGH-CHO (P<0.01).

Serum FFA and glycerol concentrations during successive steady-state HIE and MIE to exhaustion are given in Fig. 3. As expected, initial serum FFA concentrations were significantly higher after HIGH-FAT than after HIGH-CHO (P<0.01). Serum FFA concentrations immediately post-HIE were also greater in the

Table 4. Effects of dietary manipulation on submaximal heart rate, oxygen uptake, minute ventilation, substrate utilization, respiratory exchange ratio and performance time to exhaustion during moderate-intensity cycling at 50% of peak power output

<table>
<thead>
<tr>
<th></th>
<th>HIGH-FAT (n=5)</th>
<th>HIGH-CHO (n=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to exhaustion (min)</td>
<td>79.7±7.6</td>
<td>42.5±6.8</td>
</tr>
<tr>
<td>Heart rate (beats·min⁻¹)</td>
<td>142±7</td>
<td>143±8</td>
</tr>
<tr>
<td>Oxygen uptake (l·min⁻¹)</td>
<td>2.28±0.06</td>
<td>2.38±0.05</td>
</tr>
<tr>
<td>Ventilation (l·min⁻¹)</td>
<td>30.4±1.5</td>
<td>59.2±0.8</td>
</tr>
<tr>
<td>CHO oxidation (g·min⁻¹)</td>
<td>1.4±0.7</td>
<td>2.23±0.40</td>
</tr>
<tr>
<td>Fat oxidation (g·min⁻¹)</td>
<td>0.60±0.12</td>
<td>0.32±0.07</td>
</tr>
<tr>
<td>Respiratory exchange ratio</td>
<td>0.87±0.03</td>
<td>0.92±0.02</td>
</tr>
</tbody>
</table>

* P<0.05, ** P<0.01

Fig. 2. Changes in plasma glucose concentrations during high intensity and moderate intensity exercise were not significantly different between the HIGH-FAT and HIGH-CHO trial. Blood lactate concentrations rose to over 10 mmol·l⁻¹ at exhaustion following high-intensity exercise, and were significantly lower during the 20-min rest which separated the high-intensity exercise from the moderate-intensity exercise (P<0.01). V̇O₂max—maximal oxygen uptake.
Blood β-hydroxybutyrate concentrations between trials and with time were also similar (Fig. 4). The subjects did not become ketotic in either trial.

Discussion

The first important finding of this study was that the exercise performance of the subjects adapted to 2 weeks of a high-fat diet was not significantly impaired during supra-maximal and high intensity exercise (Wingeard et al. 1980). Secondly, endurance performance was significantly enhanced during subsequent prolonged exercise at approximately 65% VO_{2max} (Table 4). This improvement in endurance occurred, despite a mean starting muscle glycogen content which was two-fold lower than in the carbohydrate-adapted trial (Fig. 1).

Hence, chronic adaptation to a high-fat, low-carbohydrate diet appears to be fundamentally different to an acute lowering of body glycogen stores which has been found to impair performance during exercise of both high and low intensity (Bargstrom et al. 1967; Christensen and Hansen 1979; Gubel et al. 1979; Maughan and Poole 1981). This potential to adapt to a high-fat, low-carbohydrate diet is consistent with studies that have shown that rats adapted to high-fat diets over 4 to 5 weeks had increased endurance performance during steady-state submaximal exercise lasting approximately 1 h (Miller et al. 1984; Simi et al. 1991), or more than 2 h (after carbohydrate loading following fat adaptation (Conlee et al. 1990).

It has been suggested that possible mechanisms which may be responsible for the attenuation of carbohydrate oxidation following chronic ingestion of a high-fat diet, and the associated improvement in endurance performance may include an increased storage of triglyceride in the muscle (Conlee et al. 1990), an increased activity of carnitine-palmitoyl transferase (Fish-er et al. 1983), and an increased activity of 3-hydroxy-
acyl-coenzyme A dehydrogenase, relative to an increase in citrate synthase activity in the skeletal muscle mitochondria (Miller et al. 1984; Simi et al. 1991). These adaptations to a chronic exposure to high-fat or low CHO feeding may "retro" the working muscle mitochondria and increase their capacity for fat oxidation. Moreover, chronic exposure to a high-fat, low-carbohydrate diet has been shown to decrease muscle hexoki-
nase activity (Fishler et al. 1980), to reduce glucose uptake by the muscle and to increase tissue insulin resistance (Beck-Nielsen et al. 1978), all of which would be expected to attenuate the rate of endogenous carbohydrate oxidation.

The improvements in endurance performance associated with dietary fat adaptation in the present study contrast with the findings of Phinney et al. (1983). These investigators have shown that exercise performed at 45% VO_{2max} lasting between 2 and 2.5 h, will be similar in both fat-adapted and carbohydrate-adapted conditions. Differences between our studies and those

Fig. 3. Serum free fatty acid (FFA) concentrations were higher after tests of muscle power and prior to the high intensity exercise period in the HIGH-FAT trial compared to the HIGH-CHO trial (P<0.05). In addition, FFA concentrations remained higher in the postexercise recovery period when subjects were fat-adapted (P<0.05). Similarly, serum glycerol concentrations were higher during high-intensity exercise during the HIGH-FAT trial, than during the HIGH-CHO trial (P<0.05) and lower in recovery when the subjects were fat-adapted (P<0.05). VO_{2max}, maximal oxygen uptake

Fig. 4. Blood β-hydroxybutyrate (β-OH-butyrate) concentrations were no different between trials and did not in-tace significantly with exercise or in the postexercise recovery period. VO_{2max}, maximal oxygen uptake

subjects during the HIGH-FAT trial (P<0.05). How-
ever, there were no differences between trials in serum FFA concentrations during subsequent MIE. Serum glycerol concentrations were higher throughout HIE in the HIGH-FAT than in the HIGH-CHO trial (P<0.01, Fig. 3). However, the rates of se-
rum glycerol accumulation in the two HIE trials were not different. Serum glycerol concentrations also rose similarly during MIE in both the HIGH-FAT and HIGH-CHO trials. The only difference between trials in serum glycerol concentrations was during the recover-
ery period following HIE. Serum glycerol concentra-
tions were lower in the HIGH-FAT trial than the HIGH-CHO at 10 min postexercise (P<0.05).

Blood β-hydroxybutyrate concentrations between trials and with time were also similar (Fig. 4). The subjects did not become ketotic in either trial.
of Phinney et al. (1983) may be attributed, in part, to the HIE that preceded the MIE in this study. The mean starting muscle glycogen content after prior HIE in the fat-adapted state in the present study was lower than that found even at the end of MIE in the study by Phinney et al. (1983). Thus, dietary fat adaptation may be more likely to have an effect when body glycogen stores are low.

Another difference between our studies and those of Phinney et al. (1983) is that their 5-week fat adaptation diet was ketogenic, whereas our 2-week fat adaptation diet did not raise circulating β-hydroxybutyrate concentrations (Fig. 4). Blood β-hydroxybutyrate concentrations were threefold higher in the fat-adapted condition in the study by Phinney et al. (1983) than in the present study.

A third difference between our studies and those of Phinney et al. (1983) is in the rates of CHO oxidation during prolonged, moderate exercise. Even after dietary fat adaptation, rates of CHO oxidation in the present study were still over fivefold higher than in subjects exposed to a ketogenic, high-fat diet for a period of 5 weeks ([4.1 g CHO·min⁻¹ vs 0.25 g CHO·min⁻¹; Phinney et al. 1983]. This may have been due, in part, to HIE which preceded MIE. At exhaustion after HIE, mean blood lactate concentrations exceeded 14 mmol·l⁻¹, and were still greater than 5 mmol·l⁻¹ just prior to starting MIE. Moreover, the blood lactate concentrations during the 20 min rest between HIE and MIE was significantly lower during the HIGH-FAT trial than in the HIGH-CHO trial. Thus, the differences in the rates of lactate disappearance would suggest that in a relatively glycogen-depleted state, lactate may have been oxidized and thus, contributed to the overall rate of carbohydrate oxidation under the HIGH-FAT conditions.

However, it is also possible that the overall rates of carbohydrate oxidation were underestimated in the study by Phinney et al. (1983). The respiratory quotient associated with both ketogenesis without concomitant oxidation and glycogenolysis from amino acids with the retention of glucose have been stated to be 0 and 0.36, respectively by Schatz and Ravussin (1980). Significant rates of ketogenesis, ketone body oxidation and glycoenergy may explain why the calculated rate of blood glucose oxidation after fat adaptation, based on 13C-glucose enrichment of blood and breath CO₂ of 5.1 mg CHO·kg⁻¹·min⁻¹ exceeded the total carbohydrate oxidation (0.36 vs 0.25 g CHO·min⁻¹; Phinney et al. 1983) estimated from gas exchange data.

Phinney et al. (1983) have suggested that the extreme conservation of carbohydrate oxidation during exercise would limit the intensity of exercise which could be performed by the subjects. This suggestion is not supported by the results of the present study, in which HIE performance was not significantly different in the carbohydrate versus the fat-adapted state. In the present study, blood glucose concentrations were maintained throughout HIE and MIE, subjects were not ketogenic, and R indicated that carbohydrate oxidation accounted for over 50% of the overall substrate oxidized during MIE (Fig. 2).

One of the limitations in the present study was that the HIGH FAT was also relatively higher in protein content, when compared to the equal energy HIGH CHO. The de-amination of excess protein may have increased the availability of carbohydrate from the diet, and therefore, would have diluted the effect of the HIGH FAT, apparently low carbohydrate diet.

Thus, the results of the present study would suggest that 2 weeks of adaptation to a high-fat diet is sufficient to alter endogenous carbohydrate stores and relative rates of substrate oxidation, in the absence of marked ketosis. Submaximal exercise performance is enhanced under these conditions, especially when preceded by HIE, which partially depletes muscle glycogen stores. These results do not suggest that fatigue is dissociated from muscle glycogen depletion, but rather, that fatigue in the glycogen-depleted state may be delayed in subjects with mitochondrial adaptations favours an increased capacity for fat oxidation. Further work, however, is required to see if such adaptations improve endurance under conditions of more competitive, higher intensity (more than 70% of maximum), prolonged exercise, without the complications of prior exercise, or in ultra-endurance sport, in which both liver glycogen and muscle glycogen stores may become depleted.

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References


