

Diet composition and the performance of high-intensity exercise

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The crucial role of muscle glycogen as a fuel during prolonged exercise is well established, and the effects of acute changes in dietary carbohydrate intake on muscle glycogen content and on endurance capacity are equally well known. More recently, it has been recognized that diet can also affect the performance of high-intensity exercise of short (2–7 min) duration. If the muscle glycogen content is lowered by prolonged (1–1.5 h) exhausting cycle exercise, and is subsequently kept low for 3–4 days by consumption of a diet deficient in carbohydrate (<5% of total energy intake), there is a dramatic (~10–30%) reduction in exercise capacity during cycling sustainable for about 5 min. The same effect is observed if exercise is preceded by 3–4 days on a carbohydrate-restricted diet or by a 24 h total fast without prior depletion of the muscle glycogen. Consumption of a diet high in carbohydrate (70% of total energy intake from carbohydrate) for 3–4 days before exercise improves exercise capacity during high-intensity exercise, although this effect is less consistent. The blood lactate concentration is always lower at the point of fatigue after a diet low in carbohydrate and higher after a diet high in carbohydrate than after a normal diet. Even when the duration of the exercise task is kept constant, the blood lactate concentration is higher after exercise on a diet high in carbohydrate than on a diet low in carbohydrate. Consumption of a low-carbohydrate isoenergetic diet is achieved by an increased intake of protein and fat. A high-protein diet, particularly when combined with a low carbohydrate intake, results in metabolic acidosis, which ensues within 24 h and persists for at least 4 days. This appears to be the result of an increase in the circulating concentrations of strong organic acids, particularly free fatty acids and 3-hydroxybutyrate, together with an increase in the total plasma protein concentration. This acidosis, rather than any decrease in the muscle glycogen content, may be responsible for the reduced exercise capacity in high-intensity exercise; this may be due to a reduced rate of efflux of lactate and hydrogen ions from the working muscles. Alternatively, the accumulation of acetyl groups in the carbohydrate-deprived state may reduce substrate flux through the pyruvate dehydrogenase complex, thus reducing aerobic energy supply and accelerating the onset of fatigue.

Keywords: Ammonia, dietary carbohydrate, glycolysis, muscle glycogen.

Introduction

The realization that the performance of muscular exercise is influenced by the preceding diet is not new. Christensen and Hansen (1939) showed that endurance capacity in prolonged work was enhanced if a diet

high in carbohydrate was consumed in the days prior to exercise, and was reduced by consumption of a low-carbohydrate diet. The mechanism underlying this observation did not become apparent until the use of the needle biopsy technique to investigate muscle metabolism came into more widespread use in the 1960s. Bergstrom and Hultman (1966) showed that the glycogen content of the exercising muscles is dramatically reduced during prolonged strenuous cycle

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exercise. Hermansen *et al.* (1967) reported that during cycling exercise at a work rate corresponding to approximately 75% of maximum oxygen uptake ($\dot{V}O_2$ max), marked depletion of the glycogen stores of the quadriceps muscles occurred at exhaustion. Bergstrom and Hultman (1966) had already shown that consumption of a diet rich in carbohydrate for a few days after exercise-induced glycogen depletion resulted in a rapid resynthesis of the glycogen stores; after 2–3 days on the high-carbohydrate diet, muscle glycogen content, in those muscles which had been exercised, was about 2–3 times greater than the resting value. If a low-carbohydrate diet is consumed in the days after exercise-induced glycogen depletion, the muscle glycogen content remains low for several days (Ahlborg *et al.*, 1967). Thus by using a combination of exercise and dietary modification to manipulate the glycogen content of the muscles, Bergstrom *et al.* (1967) were able to show a close relationship between the pre-exercise muscle glycogen content and the endurance time during cycle exercise at about 70–75% $\dot{V}O_2$ max. The obvious conclusion from these studies was that the availability of carbohydrate in the form of liver and muscle glycogen stores represents a limiting factor in this type of exercise, and although there are some qualifications, this remains generally true. A second conclusion is that performance is strongly influenced by the pre-exercise diet and exercise regimen.

By contrast, the cause of fatigue in high-intensity exercise of short duration (2–7 min) is not clearly understood and many different mechanisms have been proposed. Here, fatigue is defined as the inability to maintain the expected or required power output. Intense exercise results in the rapid and almost complete depletion of the intramuscular content of creatine phosphate, with the adenosine triphosphate (ATP) content being relatively well maintained. However, exercise can continue beyond the point at which no further change in these metabolites occurs. Karlsson and Saltin (1970) suggested that depletion of ATP or creatine phosphate did not limit exercise of 2–7 min duration. Both Hultman and Bergstrom (1973) and Katz *et al.* (1986) concluded that the availability of creatine phosphate could limit high-intensity exercise of short duration. Sahlin and Katz (1988) have proposed that accumulation of adenosine diphosphate (ADP) and adenosine 5'-phosphate (AMP) within the muscle as a consequence of an inadequate rate of ADP rephosphorylation will lead to deamination of AMP to form inosine monophosphate (IMP) and ammonia and that these changes are implicated in the fatigue process.

The high rates of anaerobic glycolysis which occur during such intense exercise result in a marked acidosis within the active muscle fibres. Circumstantial evidence suggests that this acidosis is intimately associated

with the fatigue process and may indeed be its cause (Dennig *et al.*, 1931). The primary substrate for glycolysis in intense exercise is the intramuscular glycogen store, and the main product is lactic acid, which is completely ionized to form lactate and free hydrogen ions (H^+). Lactate is often considered to be responsible for fatigue, but the accumulated H^+ ions are the more likely candidate for the fatigue-inducing agent, either by inhibition of phosphofructokinase, leading to a reduction in the rate of energy production by anaerobic glycolysis (Hermansen, 1981; Sahlin, 1983), or to a more direct effect on the contractile process itself (Donaldson *et al.*, 1978; Fabiato and Fabiato, 1978; Spriet *et al.*, 1987; Godt and Nosek, 1989). There is also some evidence that the lactate anion and inorganic phosphate may be involved directly in the fatigue process through their effects on the interaction between the contractile proteins (for recent reviews of peripheral fatigue mechanisms, see Enoka and Stuart, 1992; Fitts and Metzger, 1993). Most of the information relating to the possible mechanisms of fatigue in intense exercise is derived from *in vitro* studies, and the relevance of these results to the intact organism is debatable.

Dietary influences on the performance of high-intensity exercise

Although the rate of glycogen degradation during exercise increases exponentially with respect to exercise intensity, the duration is necessarily short when the intensity is high. Saltin and Karlsson (1971) and Hermansen (1981) concluded that the availability of muscle glycogen does not normally limit endurance capacity at work rates in excess of 90% $\dot{V}O_2$ max. The normal muscle glycogen content is about 100 mmol glucosyl units kg^{-1} wet weight (w.w.) (Jacobs, 1981). During exercise at 100% $\dot{V}O_2$ max, the glycogen depletion rate is about 11 mmol glucosyl units kg^{-1} w.w. min^{-1} (Sutton *et al.*, 1981). Endurance time at this work rate is generally in the order of 3–6 min, so even accepting that these figures are subject to considerable variation, there should be adequate glycogen available. Some researchers (e.g. Greenhaff *et al.*, 1988c) have measured muscle glycogen concentration at the point of fatigue during intense cycling exercise of short duration and have reported values in excess of 50 mmol glucosyl units kg^{-1} w.w.: this value is well above the K_m for phosphorylase and suggests that substrate availability should not be limiting. Similar findings have been reported when subjects have performed repeated sprints with short recovery periods. Gaitanos *et al.* (1993) found that muscle glycogen content fell from 317 mmol glucosyl units kg^{-1} dry weight (d.w.) to 201 mmol kg^{-1} d.w. after 10 maximal cycle ergometer

sprints of 6 s duration, with a 30 s rest between sprints (assuming that water content of the muscle tissue was 75% of the total wet weight, these values equate to 79 and 50 mmol glucosyl units kg^{-1} w.w., respectively). These values relate to the whole muscle, and further information on depletion rates in the different muscle fibre types is required. It is of course possible that with low pre-exercise muscle glycogen stores, high-intensity exercise performance could be limited by glycogen depletion in the fast-twitch (Type II) fibres. With normal pre-exercise glycogen levels, the glycogen content of Type II fibres during repeated sprints decreases sooner and to a greater extent than that of Type I fibres (Gollnick *et al.*, 1973). However, in the study of Gollnick *et al.* (1973), over 60% of the initial pre-exercise muscle glycogen content was still left in the Type II fibres at exhaustion.

Perhaps for this reason, the influence of dietary factors which are known to alter endurance capacity in prolonged exercise has largely been neglected during high-intensity exercise of short duration. There are suggestions in the literature that the patterns of exercise and dietary modification used to manipulate muscle glycogen content might have an effect on high-intensity exercise. Saltin and Hermansen (1967) found that even during the early stages of prolonged exercise, the respiratory exchange ratio and blood lactate concentration were lower if the exercise had been preceded by prolonged exhausting exercise and a 3 day low-carbohydrate diet compared with a control group. Rennie and Johnson (1974) found that in runners who had followed a glycogen supercompensation regimen, the blood lactate concentration was higher than during exercise of the same intensity performed under normal dietary conditions. It is also apparent that the rate of muscle glycogen degradation during moderate exercise is increased when the pre-exercise muscle glycogen content is high (Hargreaves *et al.*, 1995). Kelman *et al.* (1975) reported that blood lactate was lower than normal after a low-carbohydrate diet and higher than normal after a high-carbohydrate diet, after exercise at each of four 5 min bouts at intensities varying from 30 to 95% $\dot{V}\text{O}_2$ max (Fig. 1). Although exercise capacity was not measured in this study, the clear implication was that, if the work had been continued to the point of exhaustion, the exercise time would have been reduced after the low-carbohydrate diet and increased after the high-carbohydrate diet.

In a study by Maughan and Poole (1981), six recreationally active male subjects rode to exhaustion on a cycle ergometer at a work rate equivalent to 105% $\dot{V}\text{O}_2$ max on three occasions. Each test was carried out in the morning after an overnight fast. The first was preceded by a period on a normal mixed diet in which $43 \pm 9\%$ (mean \pm S.D.) of the total energy intake was

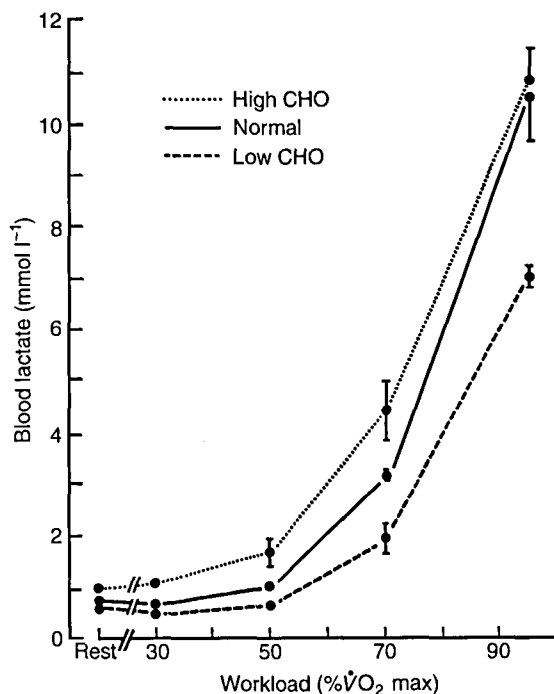


Figure 1 Blood lactate response to an incremental exercise test carried out after a normal diet, a low-carbohydrate diet and a high-carbohydrate diet. See text for further details. Reproduced with permission from Kelman *et al.* (1975).

derived from carbohydrate. Later the same day, the subjects exercised to exhaustion at $75 \pm 4\%$ $\dot{V}\text{O}_2$ max, the mean exercise time being 65 ± 4 min. For the rest of this day and the following 2 days, the subjects consumed a low-carbohydrate diet, in which carbohydrate accounted for $3 \pm 2\%$ of total energy intake, and then performed a second high-intensity exercise test. The subjects then ate a high-carbohydrate ($84 \pm 6\%$) diet for 3 days before the final high-intensity exercise test. Exercise time on the first test after the normal diet was 4.87 ± 1.07 min. After the low-carbohydrate diet, this was reduced to 3.32 ± 0.93 min ($P < 0.005$). After the high-carbohydrate diet, endurance time was longer than on the normal diet (6.65 ± 1.39 min; $P < 0.05$).

Measurements of the blood lactate concentration showed that the peak blood lactate concentration observed after exercise on the low-carbohydrate diet (8.6 ± 1.6 mmol l^{-1}) was lower ($P < 0.01$) than on the normal diet (11.7 ± 1.2 mmol l^{-1}). Following the high-carbohydrate diet, peak blood lactate (12.9 ± 1.4 mmol l^{-1}) was higher ($P < 0.05$) than on the normal diet. Although the differences in exercise duration make interpretation of these results difficult, it is apparent that the reduced endurance time is accompanied by a lower blood lactate concentration and the increased endurance time on the high-carbohydrate diet results in

Table 1 Endurance times (min) during high-intensity cycle after periods on normal, low-carbohydrate and high-carbohydrate diets^a

Endurance time (min)			n	PE	Reference
Normal	Low-CHO	High-CHO			
4.87 ± 1.07	3.32 ± 0.93	6.65 ± 1.39	6	Yes	Maughan and Poole (1981)
5.13 ± 2.00	3.68 ± 0.97	5.75 ± 3.12	11	Yes	Greenhaff <i>et al.</i> (1987a)
3.95 ± 0.48	3.33 ± 0.38	3.80 ± 0.27	7	No	Greenhaff <i>et al.</i> (1987b)
3.50 ± 1.08	2.98 ± 1.05	3.65 ± 1.15	6	No	Greenhaff <i>et al.</i> (1988a)

^a Results obtained from four separate studies. The number of subjects (*n*) in each study is shown; also shown is whether or not the low-carbohydrate experimental period was preceded by prolonged exhausting exercise (PE). Values are means ± s.d. CHO, carbohydrate.

an increased blood lactate concentration. Clearly, the relationship between blood lactate concentration and the subjective sensation of fatigue is not a simple one under these conditions.

We have since repeated this experimental design on a number of occasions. We have used physically active but not highly trained subjects in all of these studies (typically, their $\dot{V}O_2$ max values ranged from 40 to 60 ml kg⁻¹ min⁻¹), and a consistent pattern of results has emerged (Table 1). If a low-carbohydrate diet, in which less than 10% of total energy is derived from carbohydrate, is fed for 3–4 days before high-intensity (95–105% $\dot{V}O_2$ max) cycle exercise, the endurance time is significantly reduced. This is true whether or not a prolonged exhausting exercise bout intended to deplete the muscle glycogen content precedes the low-carbohydrate diet. Feeding a high-carbohydrate diet, in which about 65–75% of total energy is derived from carbohydrate, for 3–4 days before exercise at the same intensity generally results in an increased exercise performance, but this effect is less consistent; because of inter-subject variability, it may not be statistically significant. In the initial study (Maughan and Poole, 1981), the dietary variations were more extreme than in the later studies in this series. In the first study, carbohydrate accounted for 84% of total energy intake on the high-carbohydrate diet; in the later studies, it generally accounted for no more than 65–75% of energy intake.

In view of the small differences that normally separate winning from losing in sport, the balance of evidence would seem to support the use of diets high in carbohydrate where events lasting a few minutes are to be performed. What is clear is that beginning such an event with a muscle glycogen store that is low, as a result of prior exercise and inadequate glycogen repletion, will almost certainly impair performance.

In the absence of strenuous exercise, consumption of a low-carbohydrate diet for 3–4 days will not in itself result in a significant fall in the muscle glycogen con-

tent (Hultman, 1967; Jansson and Kaijser, 1982). The finding that exercise time is reduced after a low-carbohydrate diet even when this is not preceded by prolonged exhausting exercise, therefore makes it unlikely that the reduction in exercise tolerance is a consequence of a reduced carbohydrate availability. Two possible explanations for the effect of these diets on the capacity to perform high-intensity exercise are as follows: (1) an effect on the body's acid–base status; (2) an accumulation of acetyl groups after feeding a high-fat, low-carbohydrate diet may reduce flux through the pyruvate dehydrogenase (PDH) enzyme complex at the onset of exercise, leading to reduced oxidative ATP formation and an accelerated onset of fatigue. It is widely accepted that the accumulation of acetyl CoA and NADH, which results from fat oxidation, can reduce the catalytic activity of PDH, resulting in the inhibition of carbohydrate oxidation. Further work on the effect of diet on muscle metabolism during high-intensity exercise is required to determine if this possible mechanism is an important factor in influencing time to exhaustion. The remainder of this paper concentrates on the available evidence that dietary-induced modification of whole-body acid–base balance is an important factor in the development of fatigue during high-intensity exercise.

Effects of changes in acid–base status on exercise performance

Artificially induced changes in acid–base balance can affect the time for which high-intensity exercise can be sustained. Jones *et al.* (1977) showed that oral administration of NH₄Cl over 3 h before a bout of high-intensity exercise of ~5 min duration resulted in metabolic acidosis and a reduction in endurance capacity in well-trained cyclists. The administration of NaHCO₃ resulted in the development of metabolic alkalosis and increased endurance capacity. Similar

results have been reported since, and it is clear that the prior administration of bicarbonate can be effective in improving performance where fatigue results within a few minutes, but there are some inconsistencies in the literature.

Several investigators have reported a decrease in perceived exertion (Robertson *et al.*, 1986; Swank and Robertson, 1989) or an increase in performance (Sutton *et al.*, 1981; Costill *et al.*, 1984; Maughan *et al.*, 1986; Bouissou *et al.*, 1988; Goldfinch *et al.*, 1988) during high-intensity exercise after bicarbonate administration. Others, however, have shown no benefit of an induced metabolic alkalosis on perceived exertion (Poulos *et al.*, 1974) or performance (Kindermann *et al.*, 1977; Parry-Billings and MacLaren, 1986; Kelso *et al.*, 1987; Horswill *et al.*, 1988; Brien and McKenzie, 1989; Kowalchuk *et al.*, 1989). In one study designed to simulate athletic competition, trained non-elite (best 800 m time about 2 min 5 s) middle-distance runners performed a simulated 800 m race. In the alkalotic condition, subjects ran almost 3 s faster than in the placebo or control trials (Wilkes *et al.*, 1983). A more recent report has indicated similar improvements (3–4 s) over a distance of 1500 m among runners who completed simulated races in about 4 min 15 s (Bird *et al.*, 1995).

The reason for these conflicting results is not clear, but they could be due to variations in the intensity and duration of the exercise tests used, the nature of the exercise task, the dose of sodium bicarbonate administered, or the time delay between bicarbonate administration and the beginning of the exercise test (i.e. the degree of metabolic alkalosis induced).

These effects appear to be due in large part to an effect of induced extracellular alkalosis on the rate of H^+ efflux from the muscle. Bicarbonate is a natural buffer in the blood plasma and is normally present in a concentration of about 25 mmol l^{-1} . Ingestion of sodium bicarbonate in a dose of 0.3 g kg^{-1} body mass over a few hours raises the blood bicarbonate concentration by about 10 mmol l^{-1} and raises blood pH by about 0.1 units. It has been shown in studies on isolated animal muscle that changes in the pH or buffering capacity of the incubation medium can significantly alter the rate of efflux of lactate and H^+ from muscle (Hirsche *et al.*, 1975; Mainwood and Worsley-Brown, 1975). As well as affecting the extracellular acid-base balance, oral administration of NH_4Cl will significantly reduce the buffering capacity of skeletal muscle, although the intracellular pH at rest is unchanged (Hultman *et al.*, 1985). In the acidotic state, therefore, the muscle buffers are less able to cope with the H^+ produced during intense exercise; additionally, the rate of efflux of H^+ from the muscle is reduced, leading to a faster rate of intracellular H^+ accumulation. When

acidosis contributes to fatigue, this will reduce the individual's exercise tolerance.

There is also evidence from animal studies that bicarbonate administration can result in a reduced loss of ATP and accumulation of IMP when the exercise intensity is constant, and this reduced loss of adenine nucleotides from the muscle may delay fatigue (Greenhaff *et al.*, 1991).

Effects of diet on acid-base status

The effects on extracellular acid-base status of feeding diets which are either high or low in carbohydrate have been measured a number of times. These studies revealed that feeding a diet which is low in carbohydrate (<10% of total energy derived from carbohydrate) results in a metabolic acidosis, based on measurements made on arterialized venous blood samples. Feeding a high-carbohydrate diet (65–75% carbohydrate) tends towards the development of metabolic alkalosis (Greenhaff *et al.*, 1987a,b, 1988a,b). In these experiments, the low-carbohydrate diet was high in fat and high in protein, whereas the high-carbohydrate diet was low in fat and low in protein. In addition, it is not always possible to ensure a constant energy intake on each of the diets: low-carbohydrate diets tend to be unpalatable, and most subjects, if given a free choice, reduce their total energy intake on this diet. These factors make it difficult to establish which aspect of the diet is responsible for the observed changes in acid-base status. The results of a typical experiment, in which subjects prepared their own food in accordance with instructions given, are shown in Table 2. In later experiments in this series, all food was provided to subjects in the laboratory to ensure better control.

From the tables of McCance and Widdowson (1960), it is possible to estimate the dietary acid-base intake, and the results of an analysis of the diets reported in Table 2 are shown in Fig. 2. This appears to show

Table 2 Analysis of dietary records kept by subjects ($n = 11$) for 3 day periods on a normal diet, a low-carbohydrate diet and a high-carbohydrate diet (from Greenhaff *et al.*, 1987a)

	Normal	Low-CHO	High-CHO
Total energy intake (MJ)	12.7 \pm 4.1	9.9 \pm 2.6	13.9 \pm 4.2
CHO (%)	46.2 \pm 6.7	10.1 \pm 6.8	65.6 \pm 9.8
Fat (%)	39.4 \pm 6.6	64.5 \pm 5.9	24.7 \pm 10.3
Protein (%)	14.1 \pm 3.7	25.3 \pm 4.1	9.4 \pm 1.8

Values are means \pm s.d. CHO, carbohydrate.

that the dietary acid intake largely reflects the amount of protein consumed.

To address this question further, we attempted to identify more precisely which dietary components were responsible for the observed effects on acid-base balance (Greenhaff *et al.*, 1988a). Six subjects were studied on four separate occasions, each separated by 2 weeks. For the first 4 day dietary period, the subjects weighed and recorded their normal food intake. The

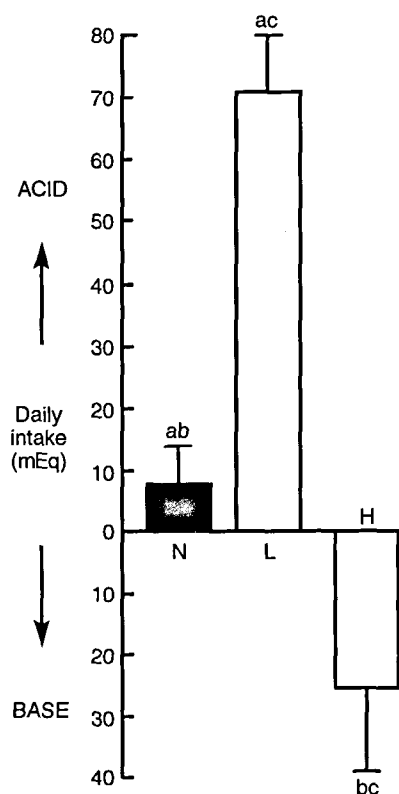


Figure 2 Net dietary acid-base intake (mean \pm S.E.M.) for subjects eating diets with normal (N), low (L) and high (H) carbohydrate contents. Significant differences ($P < 0.05$) are indicated as follows: a = normal vs low-carbohydrate; b = normal vs high-carbohydrate; c = low-carbohydrate vs high-carbohydrate. See text for further details. Reproduced with permission from Greenhaff *et al.* (1987a).

results were used to design a further three dietary regimens, each of which was fed to the subjects for a 4 day period. All diets were isoenergetic, and apart from the initial period on a normal diet, were administered in a randomized manner. The experimental diets were (1) a low-carbohydrate, high-fat, high-protein (HFHP) diet, (2) a high-carbohydrate, low-fat, normal protein (HCLF) diet, and (3) a normal carbohydrate, low-fat, high-protein (LFHP) diet (Table 3).

Arterialized venous blood samples were obtained from a superficial hand vein at rest as described by Forster *et al.* (1972) from overnight-fasted subjects prior to each diet period and each day thereafter for measurement of acid-base status (Table 4) and circulating metabolite concentrations. It was clear that metabolic acidosis was induced on the HFHP diet, with a mild acidosis on the LFHP diet. By the last day of measurement, plasma free fatty acid ($P < 0.001$, $P < 0.01$, $P < 0.05$) and blood 3-hydroxybutyrate concentrations ($P < 0.05$, $P < 0.05$, $P < 0.05$) were higher after the HFHP diet when compared with the normal, the HCLF and the LFHP diets respectively; plasma total protein concentration was higher when compared with the normal diet ($P < 0.05$).

After each of the 4 day dietary periods, the subjects performed a high-intensity exercise test at 100% $\dot{V}O_2$ max. Exercise time to exhaustion after the HFHP diet (2.98 ± 1.05 min) was shorter when compared with the normal (3.50 ± 1.08 min; $P < 0.01$) and HCLF (3.65 ± 1.15 min; $P < 0.05$) diets. Exercise time after the LFHP diet (3.13 ± 1.05 min) was also reduced when compared with the HCLF diet ($P < 0.05$), but was not significantly different from the normal diet.

Changes in blood base excess are commonly used as the major index of whole-body acid-base status in states of metabolic acidosis or alkalosis, and relate to differences in the plasma content of strong anions and cations (Siggaard-Andersen, 1974). An alternative approach (Stewart, 1981, 1983) has focused on the components of the buffer base; the term 'strong ion differences' (SID), analogous to the traditional concept of the anion gap, relates to the difference between the sum of all strong cations minus the sum of all strong

Table 3 Composition of daily intake for each dietary treatment (from Greenhaff *et al.*, 1988a)^a

	Total energy intake (MJ)	Carbohydrate (%)	Fat (%)	Protein (%)
Normal diet	10.30 \pm 2.17	45 \pm 2	41 \pm 3	14 \pm 3
Low-CHO, high-fat, high-protein diet	10.97 \pm 2.70	3 \pm 1	71 \pm 5	26 \pm 3
High-CHO, low-fat, normal protein diet	11.14 \pm 2.42	73 \pm 9	12 \pm 2	15 \pm 1
Normal carbohydrate, low-fat, high-protein diet	11.19 \pm 2.47	47 \pm 3	27 \pm 2	26 \pm 2

^a Carbohydrate, fat and protein intake are expressed as percentages of total energy intake. Values are means \pm S.D. CHO, carbohydrate.

Table 4 Exercise time to exhaustion and resting pre-exercise blood pH, bicarbonate (HCO_3^-), PCO_2 and base excess (from Greenhaff *et al.*, 1988a)

Treatment	Exercise time (min)	pH	HCO_3^- (mmol l^{-1})	PCO_2 (kPa)	Base excess (mmol l^{-1})
N	3.50 ± 1.05	7.401 ± 0.024	22.8 ± 0.9	4.9 ± 0.3	-1.4 ± 0.8
HFHP	2.98 ± 1.05	7.378 ± 0.007	19.0 ± 1.2	4.4 ± 0.4	-4.9 ± 1.0
HCLF	3.65 ± 1.15	7.396 ± 0.019	22.8 ± 0.7	4.9 ± 0.4	-1.3 ± 0.7
LFHP	3.13 ± 1.05	7.388 ± 0.016	21.1 ± 1.0	4.7 ± 0.4	-3.0 ± 0.8
	a, d, f	a, d, e	a, c, d, e, f	a, d	a, c, d, e, f

Values are means \pm s.d. a, b, c, d, e and f indicate significant differences between means ($P < 0.05$: a = N vs HFHP; b = N vs HCLF; c = N vs LFHP; d = HFHP vs HCLF; e = HFHP vs LFHP; f = HCLF vs LFHP). See text for further details. N = normal diet; HFHP = low-carbohydrate, high-fat, high-protein diet; HCLF = high-carbohydrate, low-fat, normal protein diet; LFHP = normal carbohydrate, low-fat, high-protein diet.

anions. These include not only the major inorganic ions, but also organic ions including lactate, free fatty acids and ketoacids. In addition, Stewart took account of the plasma concentration of non-volatile weak acids, primarily in the form of proteins. Plasma pH and bicarbonate concentrations are therefore dependent on PCO_2 , SID and the plasma protein concentration, each of which can vary independently.

The dietary manipulation did not change the plasma concentrations of the major strong ions (sodium and chloride), but the free fatty acids, 3-hydroxybutyrate and plasma protein concentrations were all elevated after the HFHP diet. Metabolic acidosis, therefore, appears to be a consequence of an increase in the organic component of the SID, together with an increase in the non-volatile weak acids. Rossing *et al.* (1986) and McAuliffe *et al.* (1986) have demonstrated that increasing or decreasing the plasma protein concentration will induce acidosis or alkalosis, respectively. Where dietary protein intake is increased, increased protein oxidation will also occur, leading to greater H^+ production, further increasing the acid load (News-holme and Leech, 1983). The plasma urea concentration was significantly elevated on both the HFHP and LFHP diets, thus supporting enhanced protein oxidation. Some respiratory compensation was observed on the HFHP diet, leading to a fall in the circulating PCO_2 . It seems clear from these results that it is the protein content of the diet which is primarily responsible for the observed changes in acid-base status: a high-protein diet, particularly when given in conjunction with a low-carbohydrate diet, will result in metabolic acidosis.

In a study designed to examine the effects of dietary changes on muscle metabolism, five subjects performed a 3 min exercise test at 100% $\dot{V}\text{O}_2$ max on two separate occasions (Greenhaff *et al.*, 1988c). Each test was preceded by a 4 day period on a low-carbohydrate (3%), high-protein (24%) diet, or a high-carbohydrate (82%),

low-protein (10%) diet. In addition to the usual blood samples, muscle samples were taken from the M. vastus lateralis before the diet and immediately before and immediately after the exercise test. Muscle glycogen content was unchanged by the high-protein diet, but rose by 23% on the high-carbohydrate diet. During exercise, the decline in muscle glycogen was 50% greater after the high-carbohydrate diet, but post-exercise muscle lactate content was not different between the two dietary conditions. The post-exercise blood lactate concentration was lower after the high-protein diet, indicating either a greater rate of oxidation or a slower rate of lactate efflux from the muscle. Muscle pH was not different between the two dietary conditions before exercise, but the decrease in muscle pH which occurred during the 3 min exercise period was greater (by 104%; $P < 0.05$) on the high-protein diet than on the high-carbohydrate diet; at the end of exercise, muscle pH was significantly lower ($P < 0.05$) after the high-protein diet. The changes in blood acid-base status following exercise were similar for the two exercise sessions, suggesting that the rate of H^+ efflux from the muscle was not influenced by the dietary treatments. These results suggest that the low-carbohydrate, high-protein diet may have resulted in a decrease in the muscle buffering capacity; this is in agreement with the observation of Hultman *et al.* (1985), that acute metabolic acidosis induced by oral NH_4Cl administration results in a reduction in intramuscular buffering capacity and a greater fall in muscle pH during muscle activity.

We have also studied the effects of a fast of short duration on endurance performance during cycle exercise at 100% $\dot{V}\text{O}_2$ max (Gleeson *et al.*, 1988). Six male subjects were tested twice. On the first occasion, exercise was performed 4 h after a standardized meal; on the second occasion, exercise was undertaken after a 24 h fast (although water intake was allowed during this period). Fasting resulted in the development of metabolic acidosis with marked elevation of blood

3-hydroxybutyrate and plasma free fatty acid concentrations. Exercise time in the 24 h fasted condition (3.53 ± 0.45 min) was shorter ($P < 0.01$) than in the post-absorptive state (4.05 ± 0.28 min). Again the differences in exercise times make it difficult to interpret the changes in acid-base variables and circulating metabolite concentrations measured in the post-exercise period, but the effect on performance is clear. An effect on flux through PDH could also explain the results, but the data do not allow this to be confirmed or denied.

These studies provide strong evidence that acute changes in the composition of the diet can influence performance during high-intensity exercise of short duration. It is clear that restriction of dietary carbohydrate intake in the days before exercise impairs performance. Because of the observed effects on exercise performance, and because of the known effects of artificially induced alkalosis and acidosis on performance, it is tempting to speculate that the mechanism of this effect involves an alteration in the body's acid-base balance. To investigate this further, we examined the effects of the acute administration of bicarbonate to reverse the metabolic acidosis which results from a low-carbohydrate diet.

In two unpublished studies, we have been able to show that acute reversal of the metabolic acidosis that results from either fasting or the consumption of a low-carbohydrate, high-protein diet over a period of days does not restore exercise performance. In these studies, either sodium bicarbonate or sodium citrate was administered in a dose of 0.3 g kg^{-1} body mass. This was sufficient to restore normal acid-base status, but time to fatigue at $100\% \dot{V}O_2$ max remained less than on the control trial. Fasting (for 27 h) reduced exercise time from 3.33 ± 0.27 min in the post-absorptive condition to 2.82 ± 0.32 min on the placebo trial; bicarbonate administration had no effect on performance in the post-absorptive state (mean exercise time of 3.33 ± 0.25 min) or after fasting for 27 h (2.70 ± 0.23 min).

These results clearly imply that the metabolic acidosis that results from carbohydrate deprivation or increased protein intake is not a primary factor in the fatigue process. The possibility that the reduced exercise performance in this condition results from an altered contribution of carbohydrate oxidation to ATP resynthesis remains a possibility. A further possibility, which also remains speculative at this stage, is that there may be an effect on central mechanisms associated with the perception of fatigue. The increased free fatty acid concentration observed in the low-carbohydrate conditions would be expected to displace tryptophan from its binding sites on albumin, and might lead to increased tryptophan uptake into the brain (Bailey *et*

al., 1992; Davis *et al.*, 1992). This in turn should increase serotonin (5-HT) synthesis and release, and there are some indications that this may play a role in fatigue (see Davis, 1995, for a review).

Conclusions and practical implications

It is clear from these results that the capacity to perform high-intensity exercise of only a few minutes duration can be influenced by acute changes in the composition of the diet consumed in the days before exercise. Feeding a low-carbohydrate, high-protein diet results in a dramatic reduction in endurance capacity during cycle exercise which can be sustained for about 5 min. Consumption of a high-carbohydrate, low-protein diet for 3–4 days before exercise can increase exercise capacity; this effect is less consistent, but a significant improvement in performance was seen when the most extreme variations in diet composition were used.

Consumption of a low-carbohydrate isoenergetic diet is achieved by increased intake of protein and fat. A high-protein diet, particularly when in combination with a low-carbohydrate intake, results in metabolic acidosis, as assessed by measurements on arterialized blood, which ensues within 24 h and persists for at least 4 days. The same result is observed in response to a fast of short duration (24 h). This appears to be the result of an increase in the circulating concentrations of strong organic acids, particularly free fatty acids and 3-hydroxybutyrate, together with an increase in the total plasma protein concentration. It may be this acidosis, rather than any decrease in the muscle glycogen content, which is responsible for the reduced exercise capacity in high-intensity exercise. This could be due to a reduced buffering capacity within the muscles and in the extracellular fluid, and possibly also to a reduced rate of efflux of lactate and hydrogen ions from the working muscles. There is some preliminary evidence that this is not the case, but further information is required.

From a practical point of view, there are two clear messages. The first is that it may be possible to improve performance during high-intensity exercise of short duration by consuming a high-carbohydrate, low-protein diet for a few days before competition. In endurance events lasting in excess of 1–2 h, this type of dietary manipulation is relatively common. Although the results were somewhat variable in these studies, which involved maximal exercise of 3–6 min duration, there was generally an increase in exercise time relative to the normal diet condition when the high-carbohydrate, low-protein diet was consumed; in the case of many individuals, these improvements were substan-

tial. Athletes should perhaps experiment with these procedures to establish whether they themselves would benefit. Secondly, performance in this type of exercise will be impaired if the pre-competition diet is high in protein and deficient in carbohydrate, or if food intake is restricted in the pre-competition period. It is common practice for many athletes, perhaps especially female track runners, to restrict food intake to reduce body mass. This practice, if continued up to the time of competition, may have a detrimental effect on performance. In the present studies, subjects avoided any unnecessary physical activity during the periods of dietary modification. In athletes who continue to train, even at a reduced level, during the pre-competition period, the importance of an adequate carbohydrate intake to avoid the development of metabolic acidosis will be much greater.

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