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Diet-induced metabolic acidosis and the performance of high intensity exercise in man

P. L. Greenhaff, M. Gleeson, and R. J. Maughan

Department of Environmental & Occupational Medicine, University Medical School, Aberdeen AB9 2ZD, Scotland

Summary. The influence of four isolated periods of dietary manipulation upon high intensity exercise capacity was investigated in six healthy male subjects. Subjects consumed their 'normal' (N) diet (45±2% carbohydrate (CHO), 41±3% fat, 14±3% protein) for four days after which they exercised to voluntary exhaustion at a workload equivalent to 100% $\dot{V}_{O_{2max}}$. Three further four-day periods of dietary manipulation took place; these were assigned in a randomised manner and each was followed by a high intensity exercise test. The dietary treatments were: a low CHO (3±1%), high fat (71±5%), high protein (26±3%) diet (HFHP); a high CHO (73±2%), low fat (12±2%), normal protein (15±1%) diet (HCLF); and a normal CHO (47±3%), low fat (27±2%), high protein (26±2%) diet (LFHP). Acid-base status and blood lactate concentration were measured on arterialised-venous blood at rest prior to dietary manipulation on each day of the different diets, immediately prior to exercise and at 2, 4, 6, 10 and 15 min post-exercise. Other metabolite concentrations were measured in the blood samples obtained prior to dietary manipulation and immediately prior to exercise. Exercise time to exhaustion after the HFHP diet (179±63 s) was shorter when compared with the N (210±65 s; $p<0.01$) and HCLF (219±69 s; $p<0.05$) diets. Exercise time after the LFHP diet (188±63 s) was also reduced when compared with the HCLF diet ($p<0.05$) but not significantly when compared with the N diet. Immediately prior to exercise after the HFHP diet plasma pH, bicarbonate, blood PCO_2 and base excess levels were lower when compared with the N diet ($p<0.05$, $p<0.001$, $p<0.001$, $p<0.001$ respectively), the HCLF diet ($p<0.05$, $p<0.001$, $p<0.01$, $p<0.001$ respectively) and the LFHP diet ($p<0.05$, $p<0.01$, $p<0.05$, $p<0.001$ respectively). Levels of plasma bicarbonate and blood base excess were also lower after the LFHP diet when compared with the N ($p<0.05$) and HCLF ($p<0.01$, $p<0.001$ respectively) diets. Immediately prior to exercise, plasma free fatty acids (FFA; $p<0.001$, $p<0.01$, $p<0.05$), blood 3-hydroxybutyrate (3-OHB; $p<0.05$, $p<0.05$, $p<0.05$) and plasma urea ($p<0.001$, $p<0.001$, $p<0.05$) were all higher after the HFHP diet when compared with the N, the HCLF and the LFHP diets respectively; plasma total protein was higher when compared with the N diet ($p<0.05$). The results of the present experiment suggest that dietary composition influences acid-base balance by affecting the plasma buffer base and circulating non-volatile weak acids and by doing so may influence the time taken to reach exhaustion during high intensity exercise.

Key words: High intensity exercise — Metabolic acidosis — Lactate — Diet

Introduction

We have previously demonstrated that a pattern of dietary and exercise manipulation intended to alter muscle glycogen content can significantly influence resting blood acid-base status (Greenhaff et al. 1987a). We suggested that such changes in acid-base status may influence exercise time to exhaustion during subsequent high intensity exercise tests. Experiments on subjects who performed a fixed 3 min period of high intensity exercise having undergone a similar pattern of exercise and dietary manipulation demonstrated that

Offprint requests to: R. J. Maughan

the metabolic responses after a fixed period of high intensity exercise could be significantly influenced by such a regimen (Greenhaff et al. 1988). It is now clear that dietary manipulation alone i.e. without major alterations in muscle glycogen content, will bring about similar changes in pre-exercise acid-base status and high intensity exercise performance to those we reported earlier (Greenhaff et al. 1987b). We postulated that the observed changes in resting acid-base balance were probably associated with an alteration in dietary protein intake and that this, rather than the dietary carbohydrate (CHO) intake may influence exercise capacity. The present experiment was undertaken in order to determine more precisely the dietary components that were responsible for the reported alterations in acid-base balance and exercise performance.

Materials and methods

Subjects. Six healthy male subjects gave their written consent to take part in the present experiment which was approved by the local Ethics Committee. Their physical characteristics were: age 28±5 years (mean±SD); height 176±5 cm; pre-experimental body weight 72.1±6.2 kg; post-experimental body weight 71.6±7.1 kg; pre-experimental $\dot{V}_{O_{2max}}$ 45.9±6.0 $ml \cdot kg^{-1} \cdot min^{-1}$, post-experimental $\dot{V}_{O_{2max}}$ 47.5±6.0 $ml \cdot kg^{-1} \cdot min^{-1}$.

Experimental protocol. All subjects exercised to voluntary exhaustion at a workload equivalent to 100% $\dot{V}_{O_{2max}}$ on four separate occasions each separated by a period of two weeks. Each exercise test was performed on an electrically braked cycle ergometer after an overnight fast and was preceded by a four-day period during which each subject's diet was manipulated. For the four days prior to the first exercise test all subjects refrained from alcohol and weighed and recorded their 'normal' (N) dietary intake on digital electronic balances provided (readable to 2 g). The records kept by subjects were used to assess their normal energy intake and dietary composition using a computerised version of the food tables of McCance and Widdowson (1960). The results were used to design a further three dietary regimens (Table 1) which were consumed for the four days prior to the remaining three high intensity exercise tests. All diets were isoenergetic in nature, and apart from the N diet all diets were assigned in a randomised manner and were prepared and consumed within the department. No caffeine or alcohol containing drinks were allowed on the study but water and low energy drinks were available ad libitum during the period of each diet. Exercise was restricted to a minimum for the four days prior to each exercise test and subjects were asked to refrain from any strenuous physical activity for the week preceding and for the whole of the seven-week experimental period. Table 1 illustrates the energy content and composition of each of the four dietary treatments. In an effort to determine whether the previously reported opposing changes in acid-base status and endurance performance after a low CHO, high fat, high protein diet when compared with a high CHO, low fat, low protein diet (Greenhaff et al. 1987b) were the result of a variation in dietary protein intake, the following 3 diets were administered: a low CHO, high fat, high protein (HFHP) diet similar in composition to the previously reported low CHO diet (Greenhaff et al. 1987a, 1987b); a high CHO, low fat, normal protein (HCLF) diet similar in composition to the previously reported high CHO diet (Greenhaff et al. 1987a, 1987b) with the exception that during the present study daily protein intake was maintained as close to that in the N diet as possible; finally a diet was administered wherein daily protein intake was matched with that of the HFHP diet but during this treatment daily CHO intake was maintained as close to that in the N diet as possible by adjusting the fat content (LFHP).

On each day of dietary manipulation and on the day of each exercise test subjects reported to the laboratory having fasted overnight. On these occasions and at 2, 4, 6, 10, and 15 min post-exercise, 5 ml of arterialised-venous blood was obtained from a 21 g venous cannula inserted into a superficial vein on the dorsal surface of a heated hand (Forster et al. 1972). The cannula was kept patent between samples on the day of the exercise test by flushing with isotonic saline.

Analytical procedures. Blood was collected anaerobically into a heparinised syringe and 2.5 ml was placed immediately into a tube containing K_3EDTA ($1.5 \ mg \cdot ml^{-1}$); the syringe containing the remaining blood was sealed and stored on ice until analysed in duplicate (within 2 h) for plasma pH and blood PCO_2 using a Radiometer BMS 3 Mk2 (Copenhagen, Denmark) blood gas analyser. Plasma bicarbonate and base excess values were calculated from the results using the method of Siggaard Andersen (1963). Duplicate aliquots (100 µl) of blood from the K_3EDTA tube were immediately deproteinised in 1 ml of ice cold 0.33 $mol \cdot l^{-1}$ perchloric acid and used for the measurement of glucose, lactate, pyruvate, alanine and 3-hydroxybutyrate (3-OHB) using the method of Maughan (1982). Blood from the K_3EDTA tube was also used for the determination of packed cell volume (conventional micro-haematocrit method) and haemoglobin (colorimetric method, Sigma Chemical Company Ltd., Dorset, UK) values. Plasma was obtained from the remaining blood in the K_3EDTA tube by centrifugation and was used for the measurement of free fatty acids (FFA; Noma et al. 1973), urea (enzymatic colorimetric method, Boehringer Corporation Ltd., Lewes, UK) total protein (colorometric method, Sigma Chemical Company Ltd., Dorset, UK) and glutamine (fluorimetric modification of the method described by Lund 1985). Plasma sodium was mea-

Table 1. Composition of daily dietary intake on each dietary treatment. Carbohydrate, fat and protein intake are expressed as percentages of total energy intake. Values are mean±SD

Normal	Total energy intake (MJ) 10.30±2.17	Carbohydrate (%) 45±2	Fat (%) 41±3	Protein (%) 14±3
Low carbohydrate High fat High protein	10.97±2.70	3±1	71±5	26±3
High carbohydrate Low fat Normal protein	11.14±2.42	73±2	12±2	15±1
Normal carbohydrate Low fat High protein	11.19±2.47	47±3	27±2	26±2

sured by flame photometry (410 C clinical flame photometer, Ciba-Corning, Halstead, UK). Plasma chloride was measured using a titrimetric chloride meter (EEL 920, Ciba-Corning, Halstead, UK). Statistical analysis was by analysis of variance followed by Student's paired *t*-test where appropriate. Values in the text and Tables are shown as mean ± SD; in the figures, mean ± SE is used for clarity.

Results

Exercise time

Exercise time to exhaustion after the HFHP diet (179 ± 63 s) was less for all subjects when compared with the value recorded after the N (210 ± 65 s; $p < 0.01$) and the HCLF (219 ± 69 s; $p < 0.05$) diets (Fig. 1). Exercise time after the LFHP diet (188 ± 63 s) was also reduced when compared with the value recorded after the HCLF diet ($p < 0.05$) but not significantly when compared with the value recorded after the N diet ($p = 0.14$).

Pre-exercise acid-base status

Initial measurements made after an overnight fast before dietary manipulation began showed there were no significant differences in acid-base status on the four study periods (Figs. 2-5). However on the remaining days prior to exercise it is clear that the dietary manipulation had a significant effect

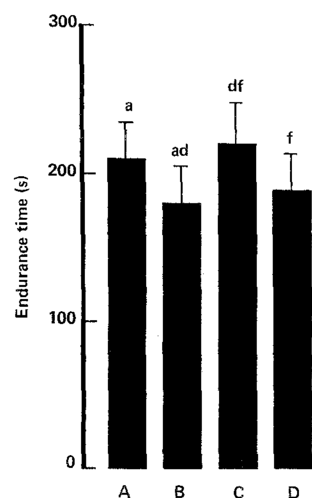

Fig. 1. Exercise time to exhaustion (mean ± SE) after a 'normal' diet (*A*), a low carbohydrate, high fat, high protein diet (*B*), a high carbohydrate, low fat, normal protein (*C*) and a normal carbohydrate, low fat, high protein diet (*D*). Significant differences ($p < 0.05$) are as follows: a = *A* vs *B*; b = *A* vs *C*; c = *A* vs *D*; d = *B* vs *C*; e = *B* vs *D*; f = *C* vs *D*

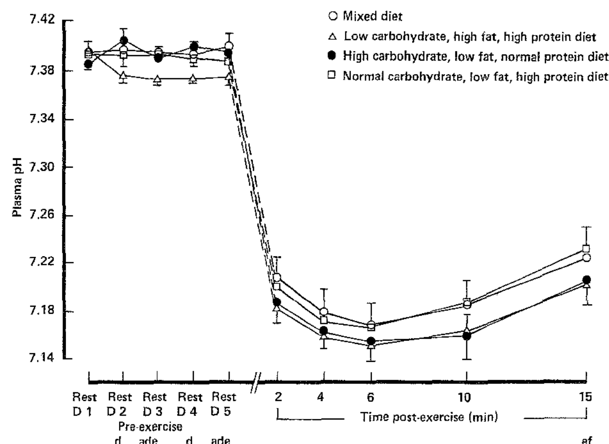

Fig. 2. Plasma pH (mean ± SE) at rest on each day of dietary manipulation and after exhaustive exercise on a 'normal' diet (*A*), a low carbohydrate, high fat, high protein diet (*B*), a high carbohydrate, low fat, normal protein (*C*) and a normal carbohydrate, low fat, high protein diet (*D*). Significant differences ($p < 0.05$) are as follows: a = *A* vs *B*; b = *A* vs *C*; c = *A* vs *D*; d = *B* vs *C*; e = *B* vs *D*; f = *C* vs *D*

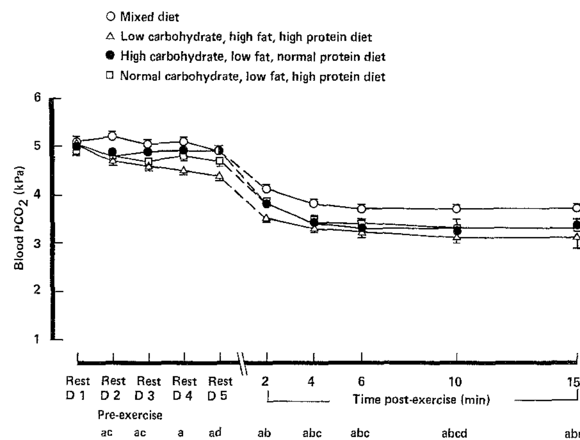

Fig. 3. Blood PCO_2 (mean ± SE) at rest on each day of dietary manipulation and after exhaustive exercise on a 'normal' diet (*A*), a low carbohydrate, high fat, high protein diet (*B*), a high carbohydrate, low fat, normal protein (*C*) and a normal carbohydrate, low fat, high protein diet (*D*). Significant differences ($p < 0.05$) are as follows: a = *A* vs *B*; b = *A* vs *C*; c = *A* vs *D*; d = *B* vs *C*; e = *B* vs *D*; f = *C* vs *D*

upon acid-base balance. On days 2 and 4 of the HFHP diet plasma pH was lower than on the HCLF diet; on day 3 and immediately prior to exercise levels were lower than on the three other dietary treatments (Fig. 2). Blood PCO_2 was lower than N on days 2 and 3 of the HFHP and LFHP diets and levels continued to fall on the remaining days of the HFHP diet resulting in blood PCO_2 being lower than on both the N and HCLF diets immediately prior to exercise (Fig. 3). There was a dramatic reduction in plasma bicarbonate levels

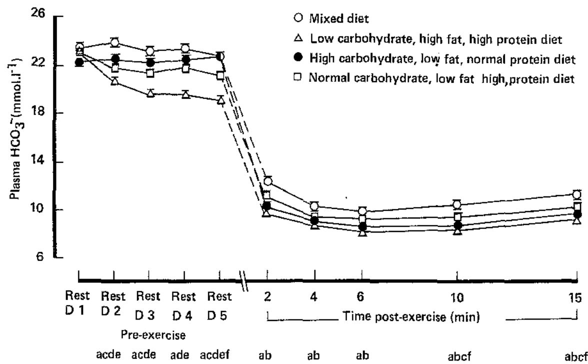

Fig. 4. Plasma bicarbonate (mean ± SE) at rest on each day of dietary manipulation and after exhaustive exercise on a 'normal' diet (*A*), a low carbohydrate, high fat, high protein diet (*B*), a high carbohydrate, low fat, normal protein (*C*) and a normal carbohydrate, low fat, high protein diet (*D*). Significant differences ($p<0.05$) are as follows: a = *A* vs *B*; b = *A* vs *C*; c = *A* vs *D*; d = *B* vs *C*; e = *B* vs *D*; f = *C* vs *D*

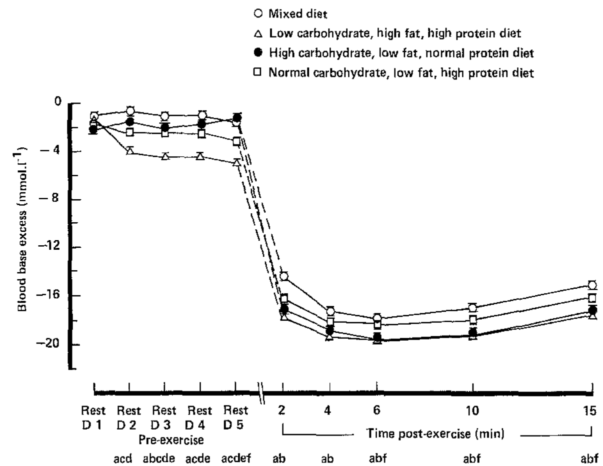

Fig. 5. Blood base excess (mean ± SE) at rest on each day of dietary manipulation and after exhaustive exercise on a 'normal' diet (*A*), a low carbohydrate, high fat, high protein diet (*B*), a high carbohydrate, low fat, normal protein (*C*) and a normal carbohydrate, low fat, high protein diet (*D*). Significant differences ($p<0.05$) are as follows: a = *A* vs *B*; b = *A* vs *C*; c = *A* vs *D*; d = *B* vs *C*; e = *B* vs *D*; f = *C* vs *D*

on the HFHP diet resulting in levels being significantly lower than the other three dietary treatments for each of the four mornings prior to exercise (Fig. 4). Bicarbonate levels were lower than on the N diet after the LFHP diet on days 2, 3 and 5 (pre-exercise); immediately prior to exercise levels were also lower than on the HCLF diet. Blood base excess (Fig. 5) showed changes similar to those seen for bicarbonate. This resulted in base excess being lower immediately prior to exercise on the HFHP diet when compared with the other three dietary treatments and lower than on the N and HCLF diets after the LFHP diet.

Pre-exercise changes in blood and plasma constituents

With the exception of blood pyruvate, haemoglobin (Hb) and packed cell volume (PCV) it is clear that each of the four periods of dietary manipulation had some influence upon the concentration of the blood and plasma constituents (Table 2). Immediately prior to exercise plasma FFA, blood 3-OHB and plasma urea were higher while blood glucose, lactate and alanine were lower after the HFHP diet when compared with the other three treatments. Plasma glutamine and total protein

Table 2. Selected circulating concentrations of blood and plasma constituents (mmol·l^{-1} unless stated) measured prior to exercise after four periods of dietary manipulation. Values are presented as mean ± SD. Significant differences ($p<0.05$): a = A vs B; b = A vs C; c = A vs D; d = B vs C; e = B vs D; f = C vs D

	(A) Normal	(B) Low CHO High fat High protein	(C) High CHO Low fat Normal protein	(D) Normal CHO Low fat High protein	Significant differences
Glucose	5.9 ± 0.3	4.8 ± 0.3	5.7 ± 0.3	5.5 ± 0.3	a, b, c, d, e, f
Lactate	0.6 ± 0.2	0.3 ± 0.1	0.7 ± 0.2	0.5 ± 0.1	a, d, e
Pyruvate	0.08 ± 0.01	0.08 ± 0.01	0.08 ± 0.01	0.07 ± 0.03	NS
Alanine	0.31 ± 0.05	0.22 ± 0.03	0.28 ± 0.05	0.26 ± 0.03	a, d
FFA	0.28 ± 0.08	0.46 ± 0.09	0.26 ± 0.12	0.30 ± 0.12	a, d, e
3-OHB	0.36 ± 0.06	0.94 ± 0.39	0.38 ± 0.12	0.36 ± 0.07	a, d, e
Glutamine	0.52 ± 0.11	0.71 ± 0.11	0.71 ± 0.07	0.83 ± 0.06	a, b, c, f
Urea	5.0 ± 1.2	8.6 ± 1.0	5.6 ± 0.1	6.8 ± 0.8	a, c, d, e, f
Total protein (g·l^{-1})	72 ± 5	87 ± 1	68 ± 9	75 ± 7	a
Hb (g·dl^{-1})	14.5 ± 0.7	15.0 ± 0.8	14.7 ± 1.2	15.0 ± 0.8	NS
PCV	41.9 ± 1.7	43.1 ± 1.6	42.1 ± 1.9	42.4 ± 1.8	NS

were higher after the HFHP diet when compared with the N diet. Plasma glutamine and plasma urea were higher and the levels of blood glucose were lower after the LFHP diet when compared with the N and the HCLF diets. After the HCLF diet blood glucose and plasma glutamine were higher than after the N diet.

Pre-exercise electrolyte changes

Plasma sodium and chloride concentrations measured at rest prior to the N diet were 142±8 $mmol \cdot l^{-1}$ and 101±1 $mmol \cdot l^{-1}$ respectively, prior to the HFHP diet they were 135±7 $mmol \cdot l^{-1}$ and 102±3 $mmol \cdot l^{-1}$ respectively, prior to the HCLF diet they were 137±6 $mmol \cdot l^{-1}$ and 101±3 $mmol \cdot l^{-1}$ respectively and prior to the LFHP diet they were 134±9 $mmol \cdot l^{-1}$ and 101±3 $mmol \cdot l^{-1}$ respectively. The four periods of dietary manipulation had little influence upon plasma sodium and chloride concentrations. At rest immediately prior to exercise (d5) after the N diet levels were 148±4 $mmol \cdot l^{-1}$ and 101±4 $mmol \cdot l^{-1}$ respectively, after the HFHP diet levels were 142±8 $mmol \cdot l^{-1}$ and 100±6 $mmol \cdot l^{-1}$ respectively, after the HCLF diet levels were 134±7 $mmol \cdot l^{-1}$ and 100±2 $mmol \cdot l^{-1}$ respectively and after the LFHP diet levels were 135±11 $mmol \cdot l^{-1}$ and 100±2 $mmol \cdot l^{-1}$ respectively. Plasma potassium was not measured due to the presence of K_3EDTA. However unpublished data from our laboratory indicate that dietary manipulation similar to that of the present study has no effect on plasma potassium levels.

Post-exercise acid-base status and blood lactate concentration

For the whole of the post-exercise period, blood PCO_2, base excess and plasma bicarbonate levels were lower after the HFHP diet when compared with the N diet (Figs. 3, 5, and 4 respectively). At 10 and 15 min post-exercise blood PCO_2 and plasma bicarbonate levels were lower after the LFHP diet when compared with the N diet. At the same times plasma bicarbonate and blood base excess values were lower after the HCLF diet when compared with the LFHP diet. There was no difference in post-exercise plasma pH until 15 min post-exercise when levels were significantly lower after the HCLF and HFHP diets when compared with the LFHP diet (Fig. 2). Table 3 illustrates the peak post-exercise decline in plasma pH, blood PCO_2, plasma bicarbonate and blood base excess on each of the four dietary treatments. The peak decline in blood PCO_2, plasma bicarbonate and blood base excess was greater after the HCLF diet than after the other three dietary treatments. In addition the peak decline in plasma bicarbonate and blood base excess was smaller after the HFHP diet when compared with the N diet. At 4 min post-exercise blood lactate concentration was lower after the HFHP diet when compared with the N and HCLF diets; at 10 min post-exercise it was lower only when compared with the HCLF diet (Fig. 6).

Discussion

The traditional understanding of acid-base balance centres around the relationship between pH, PCO_2 and bicarbonate concentration. Acid-base disturbances of respiratory origin are recognised by changes in PCO_2. The change in plasma bicarbonate is often used as an indicator of metabolic disturbances, but as bicarbonate is influenced also by PCO_2 changes this is not the most accurate measure. Changes in base excess or buffer base are classically considered to be the most accurate method of determining acid-base distur-

Table 3. The peak post-exercise decline in plasma pH, blood PCO_2, plasma bicarbonate (HCO_3^-) and blood base excess on each of the four dietary treatments. Values are presented as mean±SD. Significant differences ($p<0.05$): a=A vs B; b=A vs C; c=A vs D; d=B vs C; e=B vs D; f=C vs D

	(A) Normal	(B) Low CHO High fat High protein	(C) High CHO Low fat Normal protein	(D) Normal CHO Low fat High protein	Significant differences
pH	0.232±0.047	0.221±0.036	0.242±0.055	0.219±0.61	NS
PCO_2(kPa)	1.27 ±0.18	1.22 ±0.26	1.65 ±0.22	1.33 ±0.13	b, d, f
HCO_3^-($mmol \cdot l^{-1}$)	13.0 ±1.7	10.8 ±1.7	14.4 ±1.2	11.9 ±1.4	a, b, d, f
Base excess ($mmol \cdot l^{-1}$)	16.5 ±2.6	14.8 ±2.3	18.8 ±2.5	15.3 ±2.8	a, b, d, f

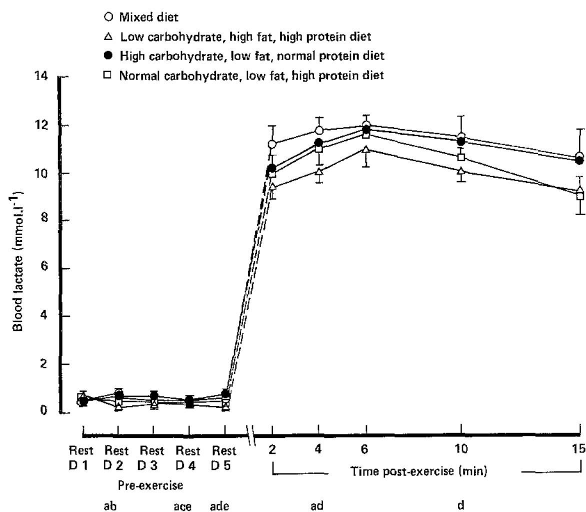

Fig. 6. Blood lactate concentration (mean ± SE) at rest on each day of dietary manipulation and after exhaustive exercise on a 'normal' diet (*A*), a low carbohydrate, high fat, high protein diet (*B*), a high carbohydrate, low fat, normal protein (*C*) and a normal carbohydrate, low fat, high protein diet (*D*). Significant differences ($p < 0.05$) are as follows: a = *A* vs *B*; b = *A* vs *C*; c = *A* vs *D*; d = *B* vs *C*; e = *B* vs *D*; f = *C* vs *D*

bances of metabolic origin, indicating the difference between the sum of all the strong cations and anions in plasma (Siggaard-Andersen 1974). Thus PCO_2 and buffer base operate as two independent variables determining acid-base status. More recently Stewart (1981, 1983) has demonstrated that the buffer base has two discrete component parts: these are the completely dissociated inorganic ions e.g. sodium, potassium and chloride and the organic ions e.g. lactate, FFA and ketoacids. He gave the name strong ion difference (SID) to the sum of all the strong cations minus the sum of all the strong anions. Moreover, Stewart indicated also that in addition to PCO_2 and SID there is a third independent variable, namely the total concentration of non volatile weak acids which in plasma are predominantly in the form of proteins. It is the variation in PCO_2, SID and non volatile weak acids, independent of one another, that will determine the changes in the concentration of other dependent variables e.g. H^+ (pH) and bicarbonate.

Pre-exercise acid-base balance

We have previously demonstrated that manipulation of dietary composition can significantly influence pre-exercise acid-base status and may thereby influence exercise duration during high intensity exercise (Greenhaff et al. 1987b). During the present experiment it is clear that for the four days prior to exercise subjects on the HFHP diet were in an acidotic state (Figs. 2–5). Base excess (Fig. 5), the conventional indicator of non-respiratory acidosis, was significantly decreased below normal. The measurements made of pre-exercise plasma sodium and chloride concentrations demonstrate that there were no differences between the four dietary treatments in the levels of the two major plasma strong inorganic ions. However total plasma proteins, plasma FFA and blood 3-OHB were all elevated during the HFHP diet (Table 2). It is therefore highly probable that the pre-exercise metabolic acidosis of the HFHP diet was brought about by an increase in the organic component of the SID (FFA, 3-OHB) in conjunction with an increase in non volatile weak acids (plasma proteins). The suggestion that the dietary component influencing acid-base status may be protein is supported by the recent work of Rossing et al. (1986) and McAuliffe et al. (1986). They demonstrated that raising or lowering the plasma protein concentration can bring about a plasma acidosis or alkalosis respectively. Changes in acid-base status brought about by an alteration in plasma protein concentration have received little consideration in determining the origin of acid-base disturbances (McAuliffe et al. 1986; Rossing et al. 1986). The augmented oxidation of dietary protein associated with the high protein diets of the present study will have resulted in a significant increase in sulphate and H^+ production (Newsholme and Leech 1983) adding further to the individual's acid load. The very mild acidosis during the LFHP diet could very likely be the result of an increase in dietary protein degradation; the absence of an increase in blood 3-OHB, plasma FFA and plasma protein levels on this diet is in agreement (Table 2). Fig. 3 demonstrates that over the four days prior to exercise on the HFHP diet, respiratory compensation (respiratory alkalosis) had taken place, thereby lowering the third independent variable PCO_2 and minimising the decline in pre-exercise pH (Fig. 2) brought about by the increased acid load of this diet.

Exercise performance and acid-base balance

The changes in pre-exercise acid-base balance of the present study are more pronounced than those we have previously reported (Greenhaff et al. 1987a, b; 1988). This is probably a consequence of the more controlled nature of the present ex-

periment whereby each of the three extreme diets was prepared and consumed within the department. As on previous occasions (Greenhaff et al. 1987 a, b) we believe that the acidosis produced by the consumption of a HFHP diet could have been one of the causes of the reduced exercise time recorded by subjects after this type of diet. In addition the less severe pre-exercise acidosis recorded during the LFHP diet may have also been responsible for the reduced exercise time after this diet. In the animal model extracellular pH can influence lactate and H^+ efflux from muscle by changing the transmembrane pH gradient (Mainwood and Worsley-Brown 1975; Mainwood et al. 1987; Hirche et al. 1975). In electrically stimulated human muscle, however, lactate efflux after 75 s of stimulation is not affected by extracellular acidosis induced prior to exercise by the ingestion of NH_4Cl (Hultman et al. 1985). How these findings relate to the present experimental situation is not clear. As exercise was continued for varying times to the point of exhaustion it is difficult to interpret the meaning of the post-exercise measurements. The peak post-exercise decline in blood PCO_2, plasma bicarbonate and blood base excess (Table 3) and the post-exercise increase in blood lactate concentration may have been smaller after the HFHP diet because muscle glycolysis or H^+ removal were inhibited during exercise as a result of the pre-exercise acidosis. However, these differences may merely be a consequence of the variations in exercise time to exhaustion; the post-exercise acid-base and blood lactate changes after the HCLF diet would suggest that this is the case.

Exercise performance and substrate availability

It is known that reduced muscle glycogen availability prior to intense exercise (Jacobs 1981; Saltin and Hermansen 1967) or selective glycogen depletion of muscle fibres during exercise (Piehl 1974) may be causally related to fatigue during exercise lasting less than 5 min. During the present study in an attempt to overcome the possible influence of pre-exercise glycogen availability on performance, a two-week recovery period was allowed between each dietary treatment. A prolonged period of reduced dietary CHO intake may lower muscle glycogen availability; Hultman (1967) reported that muscle glycogen content was not influenced unless dietary CHO was reduced for longer than 6 days. Recent work from our laboratory has shown that a four day low CHO, high fat, high protein diet very similar in composition to the HFHP diet of the present experiment, has no effect on muscle glycogen content if physical activity is restricted during the dietary period. Moreover, Maughan and Williams (1981) demonstrated that muscle glycogen content was unaltered after a 24 h fast; after a 82 h fast during which physical activity was maintained at a low level muscle glycogen content was not greatly reduced (Maughan, unpublished work). It is probable therefore that a reduction in pre-exercise muscle glycogen availability was not the reason for the reduced exercise time after the HFHP diet of the present experiment and certainly cannot explain the reduction in exercise time after the LFHP diet when compared with the HCLF diet. The pattern of glycogen depletion of muscle fibre types during cycle ergometer exercise at greater than 90% $\dot{V}_{O_2 max}$ is not influenced by the initial glycogen content of the muscle (Piehl 1974). Therefore it would appear unlikely that selective glycogen depletion of the different fibre types could explain the variation in exercise performance of the present experiment. Muscle glycogen availability has been shown to influence the pattern of substrate oxidation and relative importance of substrates taken up by muscle from blood during dynamic exercise at 84% $\dot{V}_{O_2 max}$ in man (Gollnick et al. 1981) and during supramaximal electrical stimulation in the rat hindlimb (Richter and Galbo 1986). However the effects were only apparent when large changes in muscle glycogen content were induced and may therefore not be relevant to the present study. Jansson and Kaijser (1982) demonstrated that a 5 d high fat, low CHO diet, which did not dramatically reduce muscle glycogen content, increased the relative contribution of fat to oxidative metabolism and decreased CHO utilisation during exercise at 65% $\dot{V}_{O_2 max}$. Although the dietary induced changes in circulating metabolites during the HFHP diet of the present study are in agreement with the results of Jansson and Kaijser (1982) it is not yet known if their findings are applicable to more intense exercise where the relative contribution of oxidative metabolism to energy provision is reduced. It is difficult to see how this concept could explain the reduction in exercise time after the LFHP diet when compared with the HCLF diet.

The present study confirms our previous finding that dietary composition alone can significantly influence high intensity exercise performance (Greenhaff et al. 1987b). When a high protein, low fat diet was consumed there was a trend towards high intensity exercise time being re-

duced. When a high protein, high fat diet was consumed exercise time fell significantly below normal.

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