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# The effects of a glycogen loading regimen on acid-base status and blood lactate concentration before and after a fixed period of high intensity exercise in man

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**Summary.** Six healthy male subjects exercised after an overnight fast for a fixed 3 min period at a workload equivalent to 100% of their maximal oxygen uptake ($\dot{V}_{O_2max}$) on 3 separate occasions. The first test took place after subjects had consumed a mixed diet (43±3% carbohydrate (CHO), 41±5% fat and 16±3% protein) for 3 days, and was followed 2 h later by prolonged cycling to exhaustion at 77±3% $\dot{V}_{O_2max}$ to deplete muscle glycogen stores. Following this, subjects consumed a low CHO diet (4±1% CHO, 63±5% fat and 33±6% protein) for the remainder of the day and for the subsequent 2 days; on the morning of the next day a second high intensity test took place. Finally subjects followed a 3 day high CHO diet (73±7% CHO, 17±6% fat and 10±1% protein) before their last test. Acid-base status and selected metabolites were measured on arterialised-venous blood at rest prior to exercise and at intervals for 15 min following exercise. Prior to exercise, plasma pH and blood lactate concentration were higher ($p<0.05$) after the high CHO diet when compared with the low CHO diet. Pre-exercise plasma bicarbonate, blood $PCO_2$ and blood base excess were all higher after the high ($p<0.001$, $p<0.01$, $p<0.01$ respectively) and normal ($p<0.05$, $p<0.05$, $p<0.05$ respectively) CHO diets when compared with the low CHO diet. During the post-exercise period there were no differences in plasma pH or blood base excess between the three experimental situations; plasma bicarbonate was higher ($p<0.05$) at 2 min post-exercise after the high CHO diet when compared with the low CHO diet; blood $PCO_2$ was higher throughout the post-exercise period after the high CHO diet when compared with the low CHO diet and at 2 min post-exercise was higher after the normal CHO diet than after the low CHO diet ($p<0.5$). The post-exercise blood lactate concentration after the high CHO diet was at all times higher than the corresponding values recorded after the normal CHO diet and until 15 min post-exercise was significantly higher than the values recorded after the low CHO diet. The present experiment further substantiates the view that a pattern of dietary and exercise manipulation can significantly influence the acid-base status of the blood and by doing so may influence high intensity exercise performance.

**Key words:** High intensity exercise — Dietary manipulation — Acid-base status — Blood lactate

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## Introduction

In a previous study we reported that high intensity exercise performance can be significantly altered by a pattern of exercise and dietary manipulation intended to alter muscle glycogen availability (Maughan and Poole 1981). However the mechanism by which dietary modifications may have influenced exercise performance was not apparent. Subsequent to this, we have shown (Greenhaff et al. 1987) that a similar pattern of exercise and dietary manipulation can significantly influence the acid-base status of the blood prior to exercise. Changes in acid-base status have been shown to influence performance during high intensity exercise by altering glycolytic flux, $H^+$ efflux from muscle or buffering capacity (Sutton et al. 1981; Hultman et al. 1985). We therefore concluded that the diet-induced changes in acid-base status observed during the above experiment (Greenhaff et al. 1987) may have influenced exer-

cise performance in a similar manner. However, as exercise in all of these studies was continued to the point of exhaustion the variation in endurance performance makes difficult the comparison of post-exercise acid-base status and blood lactate values. The present experiment was therefore undertaken wherein subjects exercised for a fixed 3 min period allowing a clearer interpretation of the metabolic responses to high intensity exercise under different dietary conditions.

## Materials and methods

Six healthy male subjects gave their written consent to take part in the present study which was approved by the local Ethics Committee. All were physically active but none was highly trained as is evident from the maximal oxygen uptake ($\dot{V}_{O_{2max}}$) value which they achieved during a discontinuous stepwise test on an electrically braked cycle ergometer ($49.6 \pm 6.2$ ml·kg$^{-1}$·min$^{-1}$, mean ± SD). Their other physical characteristics were: age $32 \pm 8$ years; height $177 \pm 2$ cm; body weight $70.6 \pm 5.8$ kg.

The experimental design represented diagrammatically in Fig. 1 is identical to that previously reported (Greenhaff et al. 1987) with the exception that each high intensity exercise test was terminated after 3 min of exercise. Three high intensity exercise tests took place at a pedal frequency of 60 rpm and at a workload equivalent to 100% $\dot{V}_{O_{2max}}$; all tests were completed within a space of 10 days. The first was performed after a 3 day period during which subjects consumed and recorded their normal dietary intake. This test was followed (approximately 2 h later) by prolonged cycling to exhaustion at $77 \pm 3\%$ $\dot{V}_{O_{2max}}$ in order to deplete muscle glycogen stores. Subjects then consumed a low CHO diet for 3 days followed by another 3 min high intensity exercise test. Subjects finally underwent a 3 day high CHO diet and on the morning of the following day performed their final 3 min exercise test. All tests were performed in the morning after an overnight fast. No alcohol was consumed during the study period.

All dietary intake over the experimental period was weighed on digital electronic balances (readable to 2 g) and was recorded in log books provided. Subjects were supplied with a copy of food composition tables (McCance and Widdowson 1960) and were requested to maintain CHO intake as low or as high as possible during the appropriate period of dietary manipulation. Table 1 illustrates that daily energy intake fell below normal during the low CHO diet; this decline is probably attributable to the unpalatable nature of the food consumed. However, as requested, CHO intake was increased to approximately 75% of total energy intake during the high CHO diet and dropped to less than 5% of total energy intake during the low CHO diet. As a consequence of this, daily dietary acid intake was lower than normal ($p < 0.05$) during the high CHO diet and higher than normal ($p < 0.01$) during the low CHO diet (Table 1).

Prior to exercise each subject immersed one hand in water at a temperature of 42° C for ten minutes. Immediately after this, a 21 g venous cannula was placed in a superficial vein on the dorsal surface of the hand. This enabled arterialised venous blood samples to be withdrawn anaerobically prior to exercise and at 2, 4, 6, 10 and 15 min post exercise. This technique for sampling arterialised-venous blood has been validated by Forster et al. (1972). The subject's hand was kept in the hot water throughout the post-exercise period and the cannula was kept patent between samples by flushing with a small volume of isotonic saline. Each sample (5 ml) was collected into a heparinised syringe; part (2.5 ml) of this was immediately transferred to a tube containing $K_3$EDTA (1.5 mg·ml$^{-1}$) and the syringe was capped and stored in iced water until analysed (within 2 h) for pH and $PCO_2$ using a Radiometer BMS 3 Mk 2 (Copenhagen, Denmark) blood gas analyser. Plasma bicarbonate concentrations were derived using the method of Siggaard Andersen (1963). Duplicate aliquots (100 µl) of blood from the $K_3$EDTA tube were immediately deproteinised in 1 ml of ice cold 0.33 mol·l$^{-1}$ perchloric acid and used for the measurement of glucose, lactate, alanine and 3-hydroxybutyrate (3-OH butyrate) (Maughan 1982). Preliminary statistical analysis was by analysis of variance, with further analysis by Student's *t*-test for paired data where appropriate. Statistical significance was declared at $p < 0.05$. Values in the text and Tables are shown as mean ± SD; in the Figures mean ± SEM is used for the sake of clarity.

## Results

The acid-base status of the blood at rest immediately prior to exercise after each 3 day period of dietary manipulation is shown in Table 2. Plasma pH was significantly higher after the high CHO diet when compared with the low CHO diet

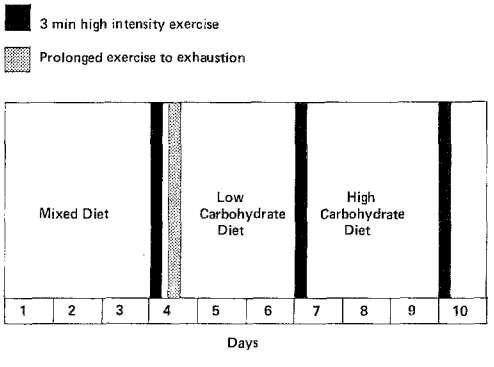


**Fig. 1.** The experimental design. See text for details

**Table 1.** Composition of daily dietary intake at each stage of the study. Values are mean ± SD ($n = 6$) calculated from records kept by subjects for the whole of the experimental period

| | Normal CHO | Low CHO | High CHO | |
|---|---|---|---|---|
| Total intake (MJ) | 12.0 ± 3.1 | 8.6 ± 1.0 | 12.0 ± 2.8 | a, c |
| CHO (%) | 43 ± 3 | 4 ± 1 | 73 ± 7 | a, b, c |
| Fat (%) | 41 ± 5 | 63 ± 5 | 17 ± 6 | a, b, c |
| Protein (%) | 16 ± 3 | 33 ± 6 | 10 ± 1 | a, b, c |
| Acid (m eq) | −12.4 ± 25.5 | 116.3 ± 35.9 | −43.1 ± 45.8 | a, b, c |

a, b, c denote significant differences between means ($p < 0.05$; a: N vs L; b: N vs H; c: L vs H)

**Table 2.** Acid-base status of arterialised venous blood samples obtained at rest prior to exercise. Values are mean ± SD ($n = 6$)

| | Normal CHO | Low CHO | High CHO | |
|---|---|---|---|---|
| Plasma pH | 7.418 ± 0.014 | 7.408 ± 0.020 | 7.427 ± 0.010 | c |
| Plasma bicarbonate ($mmol \cdot l^{-1}$) | 25.2 ± 2.0 | 21.2 ± 0.7 | 25.4 ± 1.0 | a, c |
| Blood base excess ($mmol \cdot l^{-1}$) | +0.8 ± 1.9 | −2.6 ± 0.6 | +1.4 ± 1.0 | a, c |
| Blood $PCO_2$ (kPa) | 5.2 ± 0.4 | 4.5 ± 0.3 | 5.2 ± 0.2 | a, c |

a, b, c, denote significant differences between means ($p < 0.05$: a: N vs L; b: N vs H; c: L vs H)

**Table 3.** Circulating metabolite concentrations ($mmol \cdot l^{-1}$) at rest prior to exercise after each stage of dietary manipulation. Values are mean ± SD ($n = 6$)

| | Normal CHO | Low CHO | High CHO | |
|---|---|---|---|---|
| Glucose | 4.0 ± 0.2 | 3.5 ± 0.2 | 4.4 ± 0.4 | a, c |
| Lactate | 1.0 ± 0.3 | 0.9 ± 0.3 | 1.3 ± 0.5 | c |
| Alanine | 0.23 ± 0.03 | 0.18 ± 0.05 | 0.28 ± 0.06 | c |
| 3-OH butyrate | 0.34 ± 0.31 | 0.76 ± 0.11 | 0.25 ± 0.13 | a, c |

a, b, c, denote significant differences between means ($p < 0.05$: a: N vs L; b: N vs H; c: L vs H)

($p < 0.05$); no significant difference was found when comparing either of the above two conditions with the normal CHO diet. Blood $PCO_2$, plasma bicarbonate and blood base excess were all lower after the low CHO diet when compared with either the normal ($p < 0.05$, $p < 0.05$, $p < 0.05$ respectively) or high ($p < 0.01$, $p < 0.001$, $p < 0.001$ respectively) CHO diets, but there were no significant differences between the normal and high CHO diets.

The influence that the pattern of dietary manipulation had upon some of the circulating metabolites at rest prior to exercise is illustrated in Table 3. The most notable features were that blood glucose concentration was lower after the low CHO diet when compared with either the normal ($p < 0.01$) or high ($p < 0.01$) CHO diets; blood lactate and alanine concentrations were higher after the high CHO diet when compared with the low ($p < 0.05$, $p < 0.05$ respectively) CHO diet. Blood 3-OH butyrate was higher after the low CHO diet than after the normal ($p < 0.05$) or high ($p < 0.01$) CHO diets.

Throughout the post-exercise period no significant difference in plasma pH or blood base excess could be seen when comparing values after each of the 3 periods of dietary manipulation (Fig. 2, Fig. 3 respectively). As previously reported (Greenhaff et al. 1987) the greatest fall in

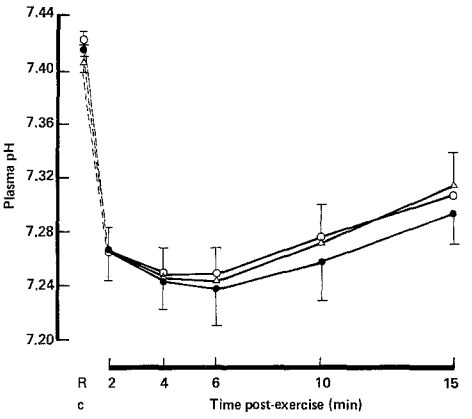


**Fig. 2.** Plasma pH (mean ± SE) before and after 3 min of high intensity exercise on normal (●), low (△) and high (○) carbohydrate diets. Significant differences ($p < 0.05$) are as follows: $a$ = normal vs low; $b$ = normal vs high; $c$ = low vs high

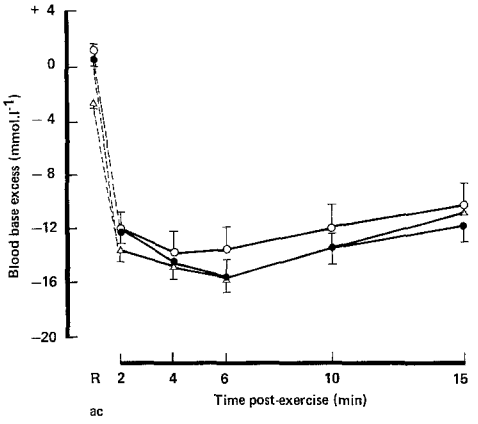


**Fig. 3.** Blood base excess (mean ± SE) before and after 3 min of high intensity exercise on normal (●), low (△) and high (○) carbohydrate diets. Significant differences ($p < 0.05$) are as follows: $a$ = normal vs low; $b$ = normal vs high; $c$ = low vs high

plasma pH and blood base excess always occurred at either 4 or 6 min post exercise.

At 2 min post-exercise plasma bicarbonate concentration was significantly higher after the high CHO diet when compared with the low CHO diet (Fig. 4); no other significant differences in plasma bicarbonate concentration were seen during the remainder of the post-exercise period.

Blood $PCO_2$ was significantly lower after the low CHO diet when compared with the high CHO diet for the whole of the post-exercise period and at 2 min post-exercise was significantly lower than after the normal CHO diet (Fig. 5).

Throughout the post-exercise period blood lactate concentration after the high CHO diet was significantly higher than the values measured

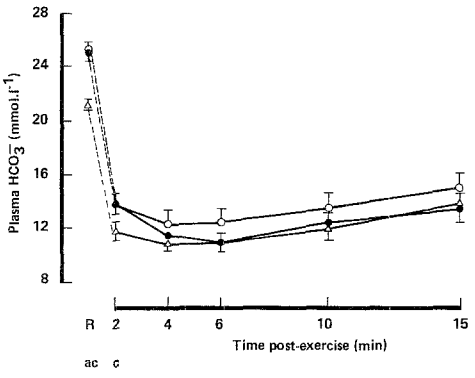


**Fig. 4.** Plasma bicarbonate ($HCO_3^-$, mean ± SE) before and after 3 min of high intensity exercise on normal (●), low (△) and high (○) carbohydrate diets. Significant differences ($p < 0.05$) are as follows: $a$ = normal vs low; $b$ = normal vs high; $c$ = low vs high

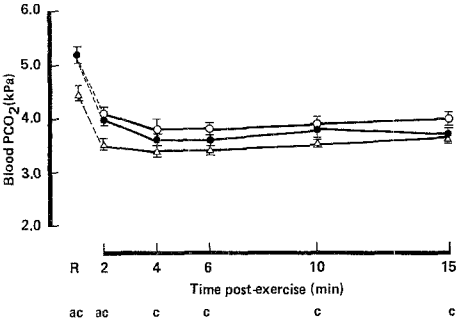


**Fig. 5.** Blood $PCO_2$ (mean ± SE) before and after 3 min of high intensity exercise on normal (●), low (△) and high (○) carbohydrate diets. Significant differences ($p < 0.05$) are as follows: $a$ = normal vs low; $b$ = normal vs high; $c$ = low vs high

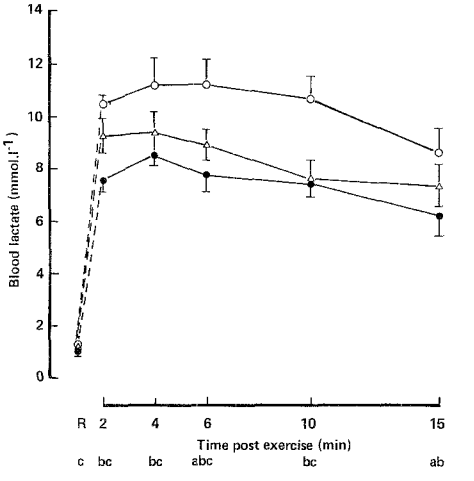


**Fig. 6.** Blood lactate concentration (mean ± SE) before and after 3 min of high intensity exercise on normal (●), low (△) and high (○) carbohydrate diets. Significant differences ($p < 0.05$) are as follows: $a$ = normal vs low; $b$ = normal vs high; $c$ = low vs high

after the normal CHO diet and until 15 min post-exercise was significantly higher than the values measured after the low CHO diet (Fig. 6). No significant differences between the normal and low CHO diets were apparent apart from at 6 and 15 min post-exercise when levels were significantly lower after the normal CHO diet. On all occasions the peak blood lactate concentration was recorded at 4 or 6 min post-exercise; peak concentrations of 8.5 ± 1.1 mmol·l$^{-1}$, 9.4 ± 2.1 mmol·l$^{-1}$ and 11.2 ± 2.4 mmol·l$^{-1}$ were recorded after the normal, low and high CHO diets respectively.

## Discussion

We have previously reported (Greenhaff et al. 1987) that a pattern of exercise and dietary variation can significantly influence the pre-exercise acid-base status of the blood and may thereby influence subsequent exercise performance. The changes in pre-exercise blood acid-base status of the present experiment are similar to our earlier findings during which exercise was continued to the point of exhaustion (Greenhaff et al. 1987).

As exercise was performed for a fixed time period during the present study, interpretation of the post-exercise blood acid-base and metabolite measurements is not obscured by variation in endurance performance. Therefore it is plausible to suggest that the absence of any significant differences in post-exercise plasma pH, plasma bicarbonate and blood base excess values in spite of the differences that existed prior to exercise indicates that muscle $H^+$ production or efflux had been influenced by the pattern of dietary and exercise manipulation. However the similarity in post-exercise blood lactate when comparing the normal and low CHO diets of the present experiment would seem to indicate that $H^+$ production of efflux was not reduced after the low CHO diet when compared to the normal diet and therefore cannot explain the reduction in exercise time to exhaustion which we have previously reported (Greenhaff et al. 1987). This is in agreement with the results of Hultman et al. (1985) who demonstrated that muscle lactate accumulation during control and acidotic conditions did not differ after 75 s of electrical stimulation. They suggested that any decrease in performance during conditions of acidosis can be attributed to a decline in intramuscular buffering capacity and not to a change in muscle $H^+$ production or efflux. One should note that differences in blood $PCO_2$ did

exist for the whole of the post-exercise period. As pH is a function of both bicarbonate and $PCO_2$ levels (Adler et al. 1965) this may also indicate that $H^+$ production or efflux was not influenced by dietary manipulation but that respiratory compensation went some way towards maintaining pH homeostasis during the post-exercise period. However as no respiratory measurements were made during the present experiment it is difficult to comment further on this point.

Assuming that lactate and $H^+$ pass through the muscle membrane at a similar rate (Hultman and Sahlin 1980), it is possible that the higher than normal post-exercise blood lactate values recorded after the high CHO diet of the present experiment may indicate that the variation in pre-exercise acid-base status increased the rate of muscle $H^+$ removal, thus helping to explain the increase in endurance performance after the consumption of a high CHO diet reported by Maughan and Poole (1981). However this may not be the case: Jansson and Kaijser (1982) indicated that a variation in dietary CHO intake could significantly influence muscle CHO utilisation and blood lactate accumulation. The increased blood lactate accumulation after the high CHO diet of the present experiment may therefore be a direct consequence of an increased glycolytic flux. The study of Jansson and Kaijser (1982) involved exercise at an intensity equivalent to 65% $\dot{V}_{O_2max}$ during which oxidative metabolism will have made a near total contribution to energy production. However, during 3 min of exercise at a workload equivalent to 100% $\dot{V}_{O_2max}$ the relative contribution of oxidative metabolism to total energy output will be reduced with anaerobic processes providing about 35% of the total energy needs (Gollnick 1973); whether a change in CHO intake can influence glycolytic flux under these circumstances is not known. The present results provide circumstantial evidence that glycolytic flux may be increased after the high CHO diet. The similarity in post-exercise blood lactate values when comparing the normal and low CHO diets suggests that there may be a critical level of CHO intake, or more speculatively, muscle glycogen, below which this effect does not operate.

Muscle glycogen levels have been causally related to post-exercise blood lactate accumulation (Asmussen et al. 1974). Although muscle glycogen stores were not measured during the present experiment, levels were probably higher than normal after the high CHO diet and lower than normal after the low CHO diet. In agreement with the results of Asmussen et al., the rate of muscle glycolysis may have been increased above normal after the high CHO diet and therefore produced the high post-exercise blood lactate levels of the present experiment. Richter and Galbo (1986) recently showed that higher than normal muscle glycogen levels could increase the rate of glycolysis and as a consequence elevate blood lactate levels above normal values during supramaximum electrical stimulation. However, as this type of electrical stimulation will abolish the normal muscle fibre recruitment pattern and as the experiment was performed using a rat hindlimb preparation the extent to which the results are applicable to man is not known. The existence of a direct relationship between muscle glycogen content and blood lactate accumulation in man has been questioned (Jacobs 1981; Saltin and Hermansen 1967) with similar post-exercise blood lactate concentrations being observed over a wide range of pre-exercise muscle glycogen levels. This latter point is supported by the similarity in post-exercise blood lactate concentrations when comparing the normal and low CHO diets of the present experiment. Whether this similarity can be explained by the uptake of lactate after short term high intensity exercise by skeletal muscle and liver being different under different dietary conditions is not known.

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