

## II. Exercising

### Post-Race Blood Biochemistry in Thoroughbreds

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#### Summary

*Blood samples were collected from Thoroughbred racehorses within 10 minutes after races over distances of 1000 to 2402 m held at meetings at Newmarket, England, and Sha Tin, Hong Kong. The alterations in the blood concentration of each constituent measured were of a similar magnitude at the two meetings. Following racing, marked increases in haematocrit and circulating concentrations of glucose, glycerol, lactate, pyruvate, uric acid and cortisol occurred. A moderate increase in total plasma protein, sodium and phosphate, with no change or a slight elevation in potassium and no change in chloride or urea concentrations were observed. The large increase in blood lactate concentration resulted in changes in the acid-base status of the horses, with a venous pH below 6.900 being recorded in some horses. A consistent and generally small increase in both creatine kinase and aspartate aminotransferase activities occurred. There appeared to be no relationship between distance raced and degree of change seen.*

#### Introduction

The effects of different intensities and duration of exercise on the concentrations of a number of blood constituents have been determined during and following various devised tests (Milne 1974; Lindholm and Saltin, 1974; Persson and Ullberg, 1974; Snow and MacKenzie, 1977; Snow *et al.*, 1982) and competitive endurance rides and eventing (Lucke and Hall, 1980; Rose *et al.*, 1977, 1980). However, only limited studies have been carried out on the alterations in a number of important blood constituents following racing of Thoroughbreds (Streter 1959; Keenan 1979) and Standardbreds (trotters) (Krzywaneck 1974). Such studies are important because they indicate the animal's response under actual competition and include the changes associated with pre-race excitement. In addition to these investigations leading to an understanding of the demands of racing, thus aiding the planning of training schedules, measurements in a

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large number of animals may indicate blood constituents that could be usefully analyzed as indicators of ability.

The aim of the present study was to determine the changes in a number of blood constituents as soon as possible after racing over varying distances. This investigation was carried out at two race meetings, one in England and one in Hong Kong.

### *Materials and Methods*

*Study 1: England.* This was carried out at a two-day race meeting at Newmarket held on the July course. Weather conditions were fine and mild (temperature 23°C). Over the two days, blood samples were collected from 45 horses between 3 to 14 minutes ( $7.2 \pm 3.2$ ) after the completion of the race. Samples were collected in stalls next to the dismounting yard which adjoined the exit from the race-track. The distances raced and number of horses sampled were 1201 m (6f, 19 horses), 1401 m (7f, 15 horses), 1602 m (8f, 3 horses), 2002 m (10f, 4 horses) and 2402 m (12f, 4 horses). As resting blood samples could not be collected prior to racing, 17 of the horses that were sampled and that were stabled in the Newmarket area were bled 48 or 72 hours following their race and prior to afternoon stable duties and feeding.

*Study 1: Hong Kong.* This was carried out at a race meeting at the Sha Tin race-course on a fine warm day (20°C and 80% humidity). Blood samples were collected from a total of 45 horses raced over 1000 m (23), 1400 m (8), 1800 m (7) and 2200 m (7). Samples were taken between 2 and 8 minutes after racing (time of sampling was not recorded for all animals; however, the mean time was estimated to be 5 minutes). On the afternoon prior to the race day, resting blood samples were collected from 24 horses competing the next day; 19 of these also had post-race samples taken.

At both race meetings samples were taken from those finishing throughout the field. However, winners could not be sampled because of the dope testing requirements, and place getters were usually sampled after 6 minutes because of the necessity to go to the place winners enclosure first.

### *Sample collection and analyses*

Venous blood samples were collected from the jugular vein into 20 ml syringes, and aliquots of blood were placed into tubes containing the appropriate anticoagulant. The samples for lactate and pyruvate determination were deproteinized using a cold solution of 10% trichloroacetic acid and 4.5% concentrated hydrochloric acid. Samples of 2 ml for blood gas analysis were collected and stored anaerobically on ice. All samples were stored on ice until the plasma or supernatant could be removed and stored at -20°C for later analysis. Blood gas analyses and haematocrit determinations were carried out within several hours of collection and the routine plasma biochemistry was carried out within four days.

The following determinations were carried out: haematocrit, total plasma protein, pH,  $pCO_2$ ,  $pO_2$ ,  $HCO_3^-$ , base excess, electrolytes ( $Na^+$ ,  $K^+$ ,  $Cl^-$  and  $PO_4^{2-}$ ), urea, glucose, glycerol, uric acid, lactate, pyruvate, cortisol, bilirubin, aspartate aminotransferase (AST) and creatine kinase (CK). Conventional laboratory techniques were used for the determinations, although for some constituents these differed between the two laboratories carrying out analyses of constituents routinely determined. Enzyme assays were carried out at 37°C. At Newmarket, the blood gas analyses were carried out at

37°C and a haemoglobin of 150 g/l, whilst at Hong Kong, measurements were carried out at 38.9°C after setting for the true haemoglobin concentration. Technical problems with the O<sub>2</sub> electrode precluded the determination of pO<sub>2</sub> at Hong Kong.

A full haematological examination was carried out and this has been reported elsewhere (Snow *et al.*, 1983a). In addition, the change in the composition of the circulating free fatty acid pool was determined in some animals (Snow *et al.*, 1983b).

Where possible, statistical analyses were carried out using either a paired or unpaired Student's 't' test. Because of the variation in post-race sampling times comparison between individuals was not possible.

### Results

The winning speeds and the results of blood gas determinations are shown in Tables 1a and 1b. The results of haematocrit, total plasma protein, enzyme, cortisol and bilirubin determinations are shown in Tables 2a, 2b, 4a and 4b.

There was a marked change from resting values for pH, pCO<sub>2</sub>, pO<sub>2</sub>, HCO<sub>3</sub><sup>-</sup> and base excess with racing, and the magnitude of change was similar over the various distances. Considerable variation for these measurements was seen between animals even in one race. The lowest venous pHs recorded at Newmarket and Hong Kong were 6.894 (1201 m, post 10 min.) and 6.874 (1000 m, post 8 min.), whilst the highest were 7.162 (1201 m, post 6 min.) and 7.145 (1400 m, post 3 min.), respectively. The pH values were significantly lower in the horses raced at Newmarket than in the horses raced in Hong Kong ( $p < 0.001$ ), and the difference would be even greater if both values were adjusted for an assumed post-race body temperature of 40°C (Table 1a, 1b).

As shown in Tables 2a and 2b there was an approximate increase in haematocrit of 50%, total plasma protein concentration of 17% and plasma sodium concentration of 10%. A smaller increase was seen in plasma potassium concentration when post-race values were compared to the resting group. When the paired groups were compared before and after racing, a significant increase ( $p < 0.02$ ) was only found for the Newmarket horses.

With the exception of plasma urea, there was a very marked increase in the metabolites measured which was similar over all distances (Table 3a, 3b). The concentrations of most of these metabolites were found to vary widely following racing. Lactate concentrations ranged from 16.4 to 35.0 mmol/l and from 18.1 to 38.5 mmol/l at Newmarket and Hong Kong, respectively. The range of plasma glycerol concentrations was even greater, ranging from 0.277 to 1.66 mmol/l and from 0.380 to 1.61 mmol/l at Newmarket and Hong Kong, respectively. No resting values are given for the uric acid concentrations at Newmarket as many animals had values below the sensitivity of the assays ( $< 50 \mu\text{mol/l}$ ). In general the highest post-race uric acid concentrations were found in those sampled the longest time after finishing.

A marked increase in cortisol concentrations is evident from Tables 4a and 4b, with a tendency for greater concentrations over the longer distances at Newmarket but not Hong Kong. A moderate increase in both CK and AST activities was generally found post race. In the paired horses the increase was highly significant ( $p < 0.001$ ). In the Hong Kong horses, where the resting samples were obtained on the day prior to racing, there was an increase in the activity of both enzymes by more than 20% in 15 of the 19 horses.

TABLE 1. Various acid-base and blood gas values following racing (mean  $\pm$  SD).  
(a) Newmarket: measured at 37°C and Hb 150 g/l.

	Resting (11) <sup>a</sup>	1201 m (18)	1401 m (15)	1602 m (3)	2002 m (4)	2402 m (4)
pH <sup>b</sup>	7.403 $\pm$ 0.031	7.013 $\pm$ 0.065	6.968 $\pm$ 0.010	6.990 $\pm$ 0.038	6.984 $\pm$ 0.036	6.998 $\pm$ 0.010
pCO <sub>2</sub> (mm Hg)	48.7 $\pm$ 4.0	26.4 $\pm$ 3.9 <sup>xxx</sup>	25.5 $\pm$ 1.0 <sup>xxx</sup>	22.0 $\pm$ 1.4 <sup>xxx</sup>	25.1 $\pm$ 2.5 <sup>xxx</sup>	24.3 $\pm$ 0.6 <sup>xxx</sup>
pO <sub>2</sub> (mm Hg)	40.4 $\pm$ 5.9	60.6 $\pm$ 7.7 <sup>xxx</sup>	65.8 $\pm$ 2.5 <sup>xxx</sup>	69.2 $\pm$ 1.9 <sup>xxx</sup>	66.6 $\pm$ 4.3 <sup>xxx</sup>	64.3 $\pm$ 4.8 <sup>xxx</sup>
HCO <sub>3</sub> <sup>-</sup> (mmol/l)	30.0 $\pm$ 2.4	6.7 $\pm$ 1.6 <sup>xxx</sup>	5.8 $\pm$ 0.3 <sup>xxx</sup>	5.5 $\pm$ 0.7 <sup>xxx</sup>	6.0 $\pm$ 0.9 <sup>xxx</sup>	5.9 $\pm$ 0.2 <sup>xxx</sup>
Base excess (mmol/l)	5.1 $\pm$ 2.4	-24.1 $\pm$ 3.0 <sup>xxx</sup>	-26.2 $\pm$ 0.5 <sup>xxx</sup>	-25.1 $\pm$ 1.3 <sup>xxx</sup>	-25.5 $\pm$ 1.8 <sup>xxx</sup>	-25.1 $\pm$ 0.5 <sup>xxx</sup>
Speed (m/s)		15.9	15.8	15.8	15.5	15.4

(b) Hong Kong: measured at 37.9°C and calibrated for the Hb concentration.

	Resting (24)	1000 m (22)	1400 m (8)	1800 m (7)	2200 m (17)
pH <sup>c</sup>	7.353 $\pm$ 0.040	7.035 $\pm$ 0.030 <sup>xxx</sup>	7.027 $\pm$ 0.054 <sup>xxx</sup>	7.022 $\pm$ 0.062 <sup>xxx</sup>	7.034 $\pm$ 0.052 <sup>xxx</sup>
pCO <sub>2</sub> (mm Hg)	52.6 $\pm$ 2.1	32.8 $\pm$ 4.6 <sup>xxx</sup>	38.8 $\pm$ 4.2 <sup>xxx</sup>	37.8 $\pm$ 5.1 <sup>xxx</sup>	33.0 $\pm$ 2.7 <sup>xxx</sup>
HCO <sub>3</sub> <sup>-</sup> (mmol/l)	29.3 $\pm$ 0.9	9.8 $\pm$ 0.9 <sup>xxx</sup>	10.1 $\pm$ 3.1 <sup>xxx</sup>	9.8 $\pm$ 1.4 <sup>xxx</sup>	8.8 $\pm$ 1.3 <sup>xxx</sup>
Base excess (mmol/l)	3.4 $\pm$ 0.8	-19.3 $\pm$ 2.2 <sup>xxx</sup>	-19.2 $\pm$ 3.2 <sup>xxx</sup>	-19.6 $\pm$ 2.4 <sup>xxx</sup>	-20.1 $\pm$ 2.0 <sup>xxx</sup>
Speed (m/s)		16.7	16.3	16.2	15.1

<sup>x</sup> p < 0.05; <sup>xx</sup> p < 0.01; <sup>xxx</sup> p < 0.001.

<sup>a</sup>Group of horses sampled at Newmarket on a different occasion from other resting horses.

<sup>b</sup>Approximately 0.037 units should be subtracted to adjust for temperature of 40°C.

<sup>c</sup>Approximately 0.025 units should be subtracted to adjust for temperature of 40°C.

TABLE 2. Haematocrit, total plasma protein and electrolyte changes following racing (mean  $\pm$  SD).  
(a) Newmarket

	Resting (17)	1201 m (19)	1401 m (15)	1602 m (4)	2002 m (4)	2401 m (4)
Haematocrit (l/l)	0.42 $\pm$ 0.05	0.59 $\pm$ 0.03 <sup>xxx</sup>	0.59 $\pm$ 0.04 <sup>xxx</sup>	0.63 $\pm$ 0.04 <sup>x</sup>	0.64 $\pm$ 0.02 <sup>xxx</sup>	0.62 $\pm$ 0.05 <sup>xxx</sup>
Total plasma protein (g/l)	62.3 $\pm$ 3.7	71.6 $\pm$ 4.1 <sup>xxx</sup>	75.5 $\pm$ 3.2 <sup>xxx</sup>	80.7 $\pm$ 5.7	78.5 $\pm$ 5.8 <sup>xxx</sup>	77 $\pm$ 5.8 <sup>xxx</sup>
Sodium (mmol/l)	136 $\pm$ 3.8	147 $\pm$ 3.5 <sup>xxx</sup>	144 $\pm$ 3.5 <sup>xxx</sup>	149 $\pm$ 1.0	141 $\pm$ 2.9	147 $\pm$ 2.8
Potassium (mmol/l)	3.6 $\pm$ 0.5	4.0 $\pm$ 0.4	4.1 $\pm$ 0.4 <sup>xx</sup>	4.5 $\pm$ 0.4	3.6 $\pm$ 0.2	4.3 $\pm$ 0.8
Phosphate (mmol/l)	1.23 $\pm$ 0.12	1.64 $\pm$ 0.18 <sup>xxx</sup>	1.44 $\pm$ 0.15 <sup>xxx</sup>	1.38 $\pm$ 0.06	1.45 $\pm$ 0.12	1.45 $\pm$ 0.06

(b) Hong Kong

	Resting (24)	1000 m (23)	1400 m (8)	1800m (7)	2200m (7)
Haematocrit (l/l)	0.43 $\pm$ 0.03	0.64 $\pm$ 0.02 <sup>xxx</sup>	0.64 $\pm$ 0.04 <sup>xxx</sup>	0.66 $\pm$ 0.02 <sup>xxx</sup>	0.64 $\pm$ 0.01 <sup>xxx</sup>
Total plasma protein (g/l)	62.5 $\pm$ 3.6	73.0 $\pm$ 4.9 <sup>xxx</sup>	78.0 $\pm$ 6.7 <sup>xxx</sup>	75.0 $\pm$ 4.8 <sup>xxx</sup>	69.0 $\pm$ 6.9 <sup>xxx</sup>
Sodium (mmol/l)	138 $\pm$ 0.8	148 $\pm$ 2.1 <sup>xxx</sup>	149 $\pm$ 3.3 <sup>xxx</sup>	148 $\pm$ 4.3 <sup>xxx</sup>	146 $\pm$ 3.1 <sup>xxx</sup>
Potassium (mmol/l)	3.8 $\pm$ 0.2	4.2 $\pm$ 0.4 <sup>xxx</sup>	4.1 $\pm$ 0.4 <sup>xx</sup>	3.9 $\pm$ 0.3	4.0 $\pm$ 0.2 <sup>x</sup>
Chloride (mmol/l)	96 $\pm$ 2.8	96 $\pm$ 2.7	95 $\pm$ 1.6	96 $\pm$ 1.7	96 $\pm$ 3.1

<sup>x</sup> p < 0.05; <sup>xx</sup> p < 0.01; <sup>xxx</sup> p < 0.001.

TABLE 3. Plasma glucose, glycerol, urea, uric acid, blood lactate and pyruvate concentrations following racing (mean  $\pm$  SD).  
(a) Newmarket

	Resting (17)	1201 m (19)	1401 m (15)	1602 m (3)	2002 m (4)	2402 m (4)
Glucose (mmol/l)	5.6 $\pm$ 0.5	9.3 $\pm$ 0.7 <sup>xxx</sup>	9.2 $\pm$ 1.4 <sup>xxx</sup>	10.1 $\pm$ 3.4 <sup>xxx</sup>	8.9 $\pm$ 0.8 <sup>xxx</sup>	9.4 $\pm$ 0.6 <sup>xxx</sup>
Glycerol ( $\mu$ mol/l)	20 $\pm$ 6	760 $\pm$ 256 <sup>xxx</sup>	763 $\pm$ 323 <sup>xxx</sup>	750 $\pm$ 353 <sup>xxx</sup>	993 $\pm$ 486 <sup>xxx</sup>	697 $\pm$ 248 <sup>xxx</sup>
Lactate (mmol/l)	0.3 $\pm$ 0.1	26.6 $\pm$ 4.3 <sup>xxx</sup>	25.5 $\pm$ 2.7 <sup>xxx</sup>	29.6 $\pm$ 2.1 <sup>xxx</sup>	25.0 $\pm$ 3.4 <sup>xxx</sup>	27.3 $\pm$ 2.4 <sup>xxx</sup>
Pyruvate ( $\mu$ mol/l)	40 $\pm$ 10	330 $\pm$ 60 <sup>xxx</sup>	300 $\pm$ 50 <sup>xxx</sup>	—	320 $\pm$ 30 <sup>xxx</sup>	320 $\pm$ 80 <sup>xxx</sup>
Urea (mmol/l)	4.8 $\pm$ 1.0	5.1 $\pm$ 0.8	5.2 $\pm$ 1.4	5.7 $\pm$ 0.8	6.5 $\pm$ 0.9	5.8 $\pm$ 0.9
Uric acid ( $\mu$ mol/l)	—	90 $\pm$ 30	100 $\pm$ 30	100 $\pm$ 52	90 $\pm$ 10	88 $\pm$ 72

## (b) Hong Kong

	Resting (24)	1000 m (23)	1400 m (8)	1800 m (7)	2200 m (7)
Glucose (mmol/l)	6.1 $\pm$ 0.4	11.7 $\pm$ 1.9 <sup>xxx</sup>	11.4 $\pm$ 1.8 <sup>xxx</sup>	11.2 $\pm$ 1.9 <sup>xxx</sup>	11.7 $\pm$ 2.2 <sup>xxx</sup>
Glycerol ( $\mu$ mol/l)	18 $\pm$ 7	795 $\pm$ 292 <sup>xxx</sup>	780 $\pm$ 235 <sup>xxx</sup>	727 $\pm$ 243 <sup>xxx</sup>	674 $\pm$ 158 <sup>xxx</sup>
Lactate (mmol/l)	0.5 $\pm$ 0.1	23.4 $\pm$ 5.4 <sup>xxx</sup>	26.5 $\pm$ 1.8 <sup>xxx</sup>	27.5 $\pm$ 6.0 <sup>xxx</sup>	22.1 $\pm$ 3.6 <sup>xxx</sup>
Pyruvate ( $\mu$ mol/l)	60 $\pm$ 10	270 $\pm$ 80 <sup>xxx</sup>	300 $\pm$ 60 <sup>xxx</sup>	310 $\pm$ 60 <sup>xxx</sup>	300 $\pm$ 60 <sup>xxx</sup>
Urea (mmol/l)	2.2 $\pm$ 0.4	2.4 $\pm$ 0.3	2.1 $\pm$ 0.4	2.0 $\pm$ 0.3	2.5 $\pm$ 0.4
Uric acid ( $\mu$ mol/l)	30 $\pm$ 12	119 $\pm$ 48 <sup>xxx</sup>	124 $\pm$ 24 <sup>xxx</sup>	113 $\pm$ 24 <sup>xxx</sup>	95 $\pm$ 20 <sup>xxx</sup>

xxx =  $p < 0.001$ .

TABLE 4. Plasma cortisol and bilirubin concentrations and aspartate aminotransferase (AST) and creatine kinase (CK) activities following racing (mean  $\pm$  SD).

## (a) Newmarket

	Restung (17)	1201 m (19)	1401 m (15)	1602 m (3)	2002 m (4)	2402 m (4)
Cortisol (nmol/l)	79 $\pm$ 39	265 $\pm$ 84 <sup>xxx</sup>	367 $\pm$ 24 <sup>xxx</sup>	267 $\pm$ 24 <sup>xxx</sup>	350 $\pm$ 82 <sup>xxx</sup>	443 $\pm$ 155 <sup>xxx</sup>
CK (IU/l)	132 $\pm$ 15	207 $\pm$ 86 <sup>xxx</sup>	160 $\pm$ 66 <sup>xx</sup>	181 $\pm$ 58	176 $\pm$ 47 <sup>xx</sup>	181 $\pm$ 36 <sup>xxx</sup>
AST (IU/l)	342 $\pm$ 128	424 $\pm$ 120 <sup>xxx</sup>	388 $\pm$ 67	372 $\pm$ 30	337 $\pm$ 78	372 $\pm$ 23

## (b) Hong Kong

	Resting (24)	1000 m (23)	1400 m (8)	1800 m (7)	2200 m (7)
Cortisol (nmol/l)	102 $\pm$ 24	275 $\pm$ 55 <sup>xxx</sup>	284 $\pm$ 55 <sup>xxx</sup>	298 $\pm$ 66 <sup>xxx</sup>	251 $\pm$ 19 <sup>xxx</sup>
CK (IU/l)	65 $\pm$ 10	94 $\pm$ 81 <sup>x</sup>	88 $\pm$ 82	118 $\pm$ 119 <sup>xx</sup>	59 $\pm$ 12
AST (IU/l)	230 $\pm$ 119	278 $\pm$ 86	378 $\pm$ 163 <sup>xxx</sup>	335 $\pm$ 101 <sup>x</sup>	306 $\pm$ 133 <sup>x</sup>
Bilirubin	65 $\pm$ 11	62 $\pm$ 21	65 $\pm$ 19	64 $\pm$ 11	70 $\pm$ 11

<sup>x</sup> =  $p < 0.05$ ; <sup>xx</sup> =  $p < 0.01$ ; <sup>xxx</sup> =  $p < 0.001$ .

\*One horse that finished second had post race CK and AST activities of 1071 and 2214 IU/l, respectively, and 72 hours later these were still elevated at 1494 and 1306 IU/l, respectively. The other horse had post-race CK and AST activities of 519 and 763 IU/l, respectively, and, 48 hours later, of 660 and 715 IU/l, respectively.

### *Discussion*

In the initial planning of these studies it was intended that all blood samples would be collected at a uniform time following the race so that comparisons between individual horses could be made. However, due to practical problems, this was not feasible and therefore comparisons between individuals are not possible as the concentrations of some of the blood constituents measured can change considerably within the first ten minutes following maximal exercise. This applies especially to glycerol, uric acid,  $pO_2$  and  $pCO_2$ . These findings should therefore be considered only as indicative of the magnitude and range of concentrations that may be expected following racing. The similarity of findings between Newmarket and Hong Kong indicates that a similar effort was being exerted by both groups of horses. The slower times at Newmarket over the shorter distances are not a reflection of a poorer quality of horses but are attributable to the uphill nature of the course. The similarity in changes over the different distances raced is not surprising as the greatest difference in time of maximal galloping was approximately one minute, with the greatest effort being demanded over the last 200 metres.

Blood gas and acid/base changes have not been previously reported after Thoroughbred racing. The decreases seen in blood pH are considerable and greater overall than those reported following Standardbred trotting races over distances of 1900 to 2500 m (Krzywanek 1974). The resting values for  $pCO_2$  and derived  $HCO_3^-$  and base excess recorded at both Newmarket and Hong Kong are higher than those generally reported as normal for horses (Bergsten 1974; Milne 1974). The reason for these high values is unknown, but they may be due to a training effect seen with these horses or to possible dietary manipulation, or differences in technology.

The marked alterations seen in venous acid/base balance and  $pCO_2$  and  $pO_2$  reflect the severe metabolic demands of racing over relatively short distances, requiring an extremely high turnover of ATP to maintain muscle contraction. As the rate of oxygen utilization by muscle cannot meet all the requirements for ATP, anaerobic metabolism, i.e. the breakdown of creatine phosphate and, more importantly, the formation of lactate, occurs. The very high concentrations of blood lactate reflect the extent of anaerobic metabolism, and it is the efflux of lactate together with hydrogen ions into the blood stream that is responsible for the altered acid/base status. The blood lactate concentrations in this study are of similar magnitude to those previously reported by Keenan (1979) following Thoroughbred racing, and greater than those seen following trotting races (Krzywanek 1974). The lower blood lactate concentrations seen after trotting are probably due to the slower (11.0–12.2 m/s) and more even speed throughout a trotting race. Although the horses were not sampled at identical times during the first 15 minutes following maximal exertion, only small changes in blood pH and lactate occur as the efflux of lactate from muscle continues. Krzywanek (1974) reported that exhausted

\*In the Newmarket results two horses were excluded from the calculation of mean activities.

horses had a considerably greater increase in lactate and decrease in blood pH compared to non-exhausted horses after racing. In this present study no attempt was made to carry out such an evaluation or to relate lactate levels to performance.

Despite the high production of CO<sub>2</sub> from metabolism during exercise, a marked decrease in venous pCO<sub>2</sub> was seen following racing. A similar finding has previously been reported by Snow and MacKenzie (1977) for horses following strenuous exercise, and it is considered to be due to hyperventilation following racing.

The increases in haematocrit, total plasma protein, sodium, potassium and phosphate concentrations are very similar to those reported by Keenan (1979). The marked increase in haematocrit is mainly due to the influx of red blood cells into the circulation as a result of splenic contraction, and also to a decrease in plasma volume, as indicated by the increase in total plasma protein concentration. The decrease in plasma volume is a result of fluid shifts from the vascular compartment, including losses from the lungs and in the form of sweat. The alterations in plasma sodium, potassium and chloride were not similar and did not match the increase in total plasma protein concentration. The difference in change seen with sodium and chloride may be explained by their concentrations in sweat (Kerr and Snow, 1982). Sodium concentration in sweat is only slightly higher than that in plasma, whilst sweat chloride concentration is almost twice that of plasma. Changes in plasma potassium are a result of both loss in sweat and influx from contracting muscle. If there was no efflux from muscle to plasma, a considerable decrease in total circulating potassium would occur, as sweat potassium concentration is of the order of 40–60 mmol/l (Kerr and Snow, 1982). Therefore the finding of a significant increase in plasma potassium in the paired group at Newmarket, but not in Hong Kong, may be due to a greater sweat production under the more humid conditions on the day in Hong Kong than in Newmarket.

The increase in plasma phosphate following racing was also found by Keenan (1979) and is considered to be due to the escape from muscle of phosphate formed during the breakdown of high-energy phosphates. The continual rise of plasma uric acid following racing is similar to that seen following racing (Keenan 1979) and endurance exercise (Snow *et al.*, 1982). Snow *et al.* (1982) suggested that this was due to an efflux from muscle as a result of complete metabolism of some of the purine nucleotides being rapidly cycled during exercise.

The increases in glucose and glycerol following exercise are similar to those reported by Snow and MacKenzie (1977) following maximal exercise, and they probably reflect the marked increase in sympathetic activity stimulating hepatic glycogenolysis and adipose lipolysis. However, as high lactate concentrations are considered to inhibit lipolysis (Boyd *et al.*, 1974), a complete explanation for the extremely high plasma glycerol concentrations found in horses following racing is still outstanding. Recently Snow *et al.* (1983b) have suggested that FFAs are utilized even during short periods of maximal exertion.

The increase in plasma cortisol concentration in this present study is of the same magnitude as that reported following the first of three gallops in a repetition gallop test (Snow and MacKenzie, 1977). The significant increase in CK and AST following exercise was too great to be attributable to a decrease in plasma volume. The increase probably represents an efflux of these enzymes from muscle groups being maximally utilized. This efflux probably represents a transient change in permeability of the sarcolemma rather than any permanent damage to the muscle fibres. Nimmo and Snow (1982) have

suggested that in the horse mitochondrial membrane permeability changes are associated with maximal exercise. Whether the extent of the increase in plasma enzymes is a reflection of the state of fitness of the animal, the degree of exertion, or the inherent susceptibility of the muscle fibres still has to be ascertained. It was of interest to find that the horse at Newmarket with the highest post-race CK and AST levels had a history of elevated plasma enzyme activities during its racing career in the absence of any clinical problems. As suggested elsewhere (Kerr and Snow, 1983), elevated plasma enzyme activities following exercise do not necessarily suggest lack of fitness, or exhaustion.

The present findings should be considered as a preliminary investigation describing the extent of alterations in a number of blood constituents. Further studies are needed in comparing the alterations found in top-class as well as mediocre performers on the race-course.

### **Acknowledgements**

This study would not have been possible without approval from the Jockey Club and Royal Hong Kong Jockey Club, and the excellent co-operation from officials at the race-courses, the respective security services and the numerous trainers who allowed their horses to be sampled. Mr D. Simpson, D. Powell, K. Watkins and E. Collins kindly assisted in the collection of blood samples. Part of this study was supported by a grant from the Horserace Betting Levy Board. A Carnegie Trust for the Universities of Scotland Travel Grant permitted D.H.S. to carry out the studies in Hong Kong.

### **References**

- Bergsten, G. (1974). Blood pressure, cardiac output, and blood-gas tension in the horse at rest and during exercise. *Acta Vet. Scand.* **15**, Suppl. 48.
- Boyd, A. E., Giamber, S. R., Mager, M. and Lebovitz, H. E. (1974). Lactate inhibition of lipolysis in exercising man. *Metabolism* **23**, 531–542.
- Keenan, D. M. (1979). Changes of blood metabolites in horses after racing with particular reference to uric acid. *Aust. Vet. J.* **55**, 54–57.
- Kerr, M. G. and Snow, D. H. (1982). The composition of equine sweat during adrenaline infusion, prolonged heat exposure and exercise. *Am. J. Vet. Res.* (in press).
- Kerr, M. G. and Snow, D. H. (1983). Plasma enzyme activities in endurance horses. In: *Equine Exercise Physiology*. Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge. pp. 432–440.
- Krzywanek, H. (1974). Lactic acid concentration and pH values in trotters after racing. *J. S. Afr. Vet. Ass.* **45**, 355–360.
- Lindholm, A. and Saltin, B. (1974). The physiological and biochemical response of Standardbred horses to exercise of varying speed and duration. *Acta Vet. Scand.* **15**, 1–15.
- Lucke, J. N. and Hall, G. N. (1980). Further studies on the metabolic effects of long distance riding. Golden Horseshoe Ride 1979. *Equine Vet. J.* **12**, 189–192.
- Milne, D. W. (1974). Blood gases, acid–base balance and electrolyte and enzyme changes in exercising horses. *J. S. Afr. Vet. Ass.* **45**, 345–354.

- Nimmo, M. A. and Snow, D. H. (1982). Time course of ultrastructural changes in skeletal muscle after two types of exercise. *J. Appl. Physiol.* **52**, 910–913.
- Persson, S. G. B. and Ullberg, L. E. (1974). Blood volume in relation to exercise tolerance in trotters. *J. S. Afr. Vet. Ass.* **45**, 293–299.
- Rose, R. J., Purdue, R. A. and Hensley, W. (1977). Plasma biochemistry alterations in horses during an endurance ride. *Equine Vet. J.* **9**, 122–126.
- Rose, R. J., Ilkiw, J. E., Arnold, K. S., Backhouse, W. and Sampson, D. (1980). Plasma biochemistry in the horse during 3-day event competition. *Equine Vet. J.* **12**, 132–136.
- Snow, D. H. and MacKenzie, G. (1977). Some metabolic effects of maximal exercise in the horse and adaptations with training. *Equine Vet. J.* **9**, 134–140.
- Snow, D. H., Kerr, M. G., Nimmo, M. A. and Abbott, E. M. (1982). Alterations in blood, sweat, urine and muscle composition during prolonged exercise in the horse. *Vet. Rec.* **110**, 377–384.
- Snow, D. H., Ricketts, S. W. and Mason, D. K. (1983a). The haematological response to racing and training exercise in Thoroughbred horses, with particular reference to the leucocyte response. *Equine Vet. J.* **15**, 149–154.
- Snow, D. H., Fixter, L. M., Kerr, M. G. and Cutmore, C. M. M. (1983b). Alterations in composition of venous plasma FFA pool during prolonged and sprint exercises. In: *Biochemistry of Exercise V*. Knuttgen, H., Vogel, J. A. and Poortmans, J. (eds.). Human Kinetics Publishers, Champaign (in press).
- Streter, F. A. (1959). The effect of systemic training on plasma electrolytes, haematocrit value and blood sugar in Thoroughbred racehorses. *Can. J. Biochem. Physiol.* **37**, 273–283.

# The Effects of Maximal Exercise on Acid–Base Balance and Arterial Blood Gas Tension in Thoroughbred Horses

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## Summary

*Carotid artery blood samples were sequentially collected for acid–base and blood gas measurements from five fit Thoroughbred horses during maximal exercise. Each horse galloped 1.6 km, and samples were collected via a catheter when the horse was at rest, after 0.8 km and 1.6 km of galloping, immediately upon stopping and 5 minutes after exercise. The arterial oxygen tension ( $\text{PaO}_2$ ) and pH decreased progressively during exercise in all horses and four of them developed an obvious hypercapnoea. Each animal became progressively more acidaemic in the time it took to pull it up and in the succeeding 5 minutes. The horses developed a respiratory alkalosis, however, and the  $\text{PaO}_2$  returned to a level higher than that measured at rest. It was concluded that this phenomenon possibly limits performance in racing horses. The reasons for the development of hypoxaemia and hypercapnoea are not clear, although there are several possible causes: 1. the existence of marked ventilation–perfusion inequalities due to arteriovenous shunting; 2. an increase in the blood–alveolar diffusion distance; 3. alveolar hypoventilation. Further work is needed to assess the possible role of each of these factors in affecting performance during maximal exercise.*

## Introduction

Little information is available concerning the response of the equine respiratory system to exercise and the role it might play in limiting performance. On the basis of prediction formulae, Gillespie (1974) concluded that ventilatory factors were unlikely to limit oxygen consumption in horses exercising maximally. It is generally believed that man's ability to exercise is not limited by respiratory considerations, and the evidence for this opinion has been well reviewed (Wasserman *et al.*, 1981). Treadmill studies with horses working at submaximal levels have reinforced Gillespie's opinion and indicated that equidae and humans may be similar as regards the response of the respiratory system to exercise (Bergsten 1974; Bayly 1979; Thomas and Fregin, 1981).