

# Influence of Post-Exercise Activity on Rates of Muscle and Blood Lactate Disappearance in the Thoroughbred Horse

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## Summary

*To study the effect of post-exercise activity on the disappearance of lactate from muscle and blood, 5 Thoroughbred geldings were galloped at 12 m/sec on a treadmill with a 5° incline for 2 min. Exercise was followed by 70 min standing (S), 70 min walking (W) or 30 min trotting and 40 min walking (T). Biopsies of the middle gluteal and venous blood samples were taken before and after exercise and during recovery and analyzed for muscle and blood lactate. Mean muscle and blood lactate at the end of exercise was  $113.1 \pm 21.6$  (SD) mmol/kg dry muscle (d.m.) and  $15.5 \pm 3.7$  mmol/l, respectively. Half-times for muscle lactate were  $27.6 \pm 8.3$  min for (S) and  $15.2 \pm 5.8$  min for (T). For blood, half-times were  $26.8 \pm 5.2$ ,  $16.8 \pm 4.3$  and  $12.2 \pm 3.9$  min for (S), (W) and (T), respectively. Exponential rate constants for blood lactate disappearance were linearly related to treadmill speed. There was an apparent inverse relationship between rate constants for muscle and blood disappearance. From (S) to (T), the horses showing the greatest change in blood lactate disappearance showed a disproportional increase in muscle and vice versa. Possible explanations for the differences between horses are discussed.*

*Index terms:* Anaerobic metabolism, muscle biopsy, exercise physiology.

## Introduction

Maximal exercise results in the production and accumulation of large amounts of lactate in muscle, which in turn increases blood levels. This is particularly marked in the horse where contents in muscle of 220 mmol/kg dry muscle (d.m.) and concentrations in blood in excess of 30 mmol/l have been observed (Snow *et al.*, 1985).

It has been postulated that lactate, which is a small and therefore easily diffusible anion, may be translocated rapidly between the water compartments of the body. Lactate produced within a muscle during exercise may be utilized within the active muscle, or following diffusion, exported via the blood to other organs, principally the heart or liver, or taken up by other muscles. As early as 1933, Margaria *et al.*, demonstrated that the fall in blood lactate concentration during recovery was essentially mono-exponential following the first 5–10 min of recovery. More recently, Freund and Zou-

lounian (1981) using a two compartmental model observed that the concentration of arterial blood lactate increases during the initial phase of recovery and that its subsequent decrease could be accurately described by an equation incorporating two exponential functions. The same equation can also be used to describe the kinetics of venous lactate recovery in the horse following maximal exercise of 800 to 2000 m (Harris *et al.*, 1987). In this study it was also found that the exponential rate constants were independent of the duration of the exercise and the lactate level attained.

In man there have been several studies demonstrating a higher rate of disappearance of lactate from blood, when an active as opposed to passive mode of recovery has been adopted (Gisolfi *et al.*, 1966; Evans and Cureton, 1983). Other workers have investigated the effects of different intensities of post-exercise activity, related to the subjects  $\text{VO}_2\text{max}$ , on the rate of blood lactate disappearance (Hermansen and Stensvold, 1972; Dodd *et al.*, 1984). The present study was undertaken in order to examine in more detail lactate recovery kinetics and also to investigate the effect of different intensities of post-exercise activity on recovery in the horse. The relationship of muscle to plasma lactate during recovery was also examined.

### *Materials and Methods*

*Experimental protocol.* Five trained Thoroughbred geldings (SM, 5 yr; KJ, 4 yr; HR, 4 yr; JW, 13 yr and SL, 3 yr) were used in this study. All horses completed three experimental sessions (except KJ), at least one week being allowed between sessions, in a period of seven weeks. In each case the experimental session began with 4 min walking ( $5^\circ$  incline, 1.60 m/sec), 4 min trotting ( $5^\circ$ , 3.2 m/sec) and acceleration to 12 m/sec (40 sec) which was maintained for 2 min ( $5^\circ$  incline). Following exercise, the three recovery protocols used were as follows:

(S) standing recovery : 70 min standing in stocks.

(W) walking recovery : 70 min walking ( $0^\circ$  incline, 1.60 m/sec)

(T) trotting recovery : 30 min trotting ( $0^\circ$  incline, 3.2 m/sec) and 40 min walking ( $0^\circ$  incline, 1.60 m/sec).

A randomized design was used to allocate recovery sessions to alleviate any effects of training which could have occurred during the study.

*Blood Sampling.* Venous blood samples (20 ml) were obtained via a catheter inserted into the jugular vein under local anesthesia of the skin and secured by sutures prior to exercise. Samples were taken before exercise, at the end of 2 min at 12 m/sec and at 2-min intervals up to 20 min recovery, at 5-min intervals to 40 min recovery and at 10-min intervals to 70 min recovery (Fig. 1). Between sampling times the catheter was flushed with saline to maintain patency. Prior to sampling, 20 ml of saline and blood were withdrawn and discarded. Samples were immediately divided as follows: 2–3 ml into pre-weighed tubes containing 5 ml 1 mol/l perchloric acid (PCA) and 10 ml into a tube containing Li-Heparin. All samples were kept on ice. Blood samples for pH measurement were drawn immediately after the 20 ml sample, into 2-ml syringes with the deadspace filled with Na-Heparin (5000 u/ml). Bubbles were removed from the sample which was capped and placed in iced water. Samples were collected on (S) and (T) sessions only.

*Muscle Sampling.* An area of skin approximately  $10 \times 3$  cm over the left or right middle gluteal was shaved. Following local anesthesia of the skin, 5 incisions, 1 cm

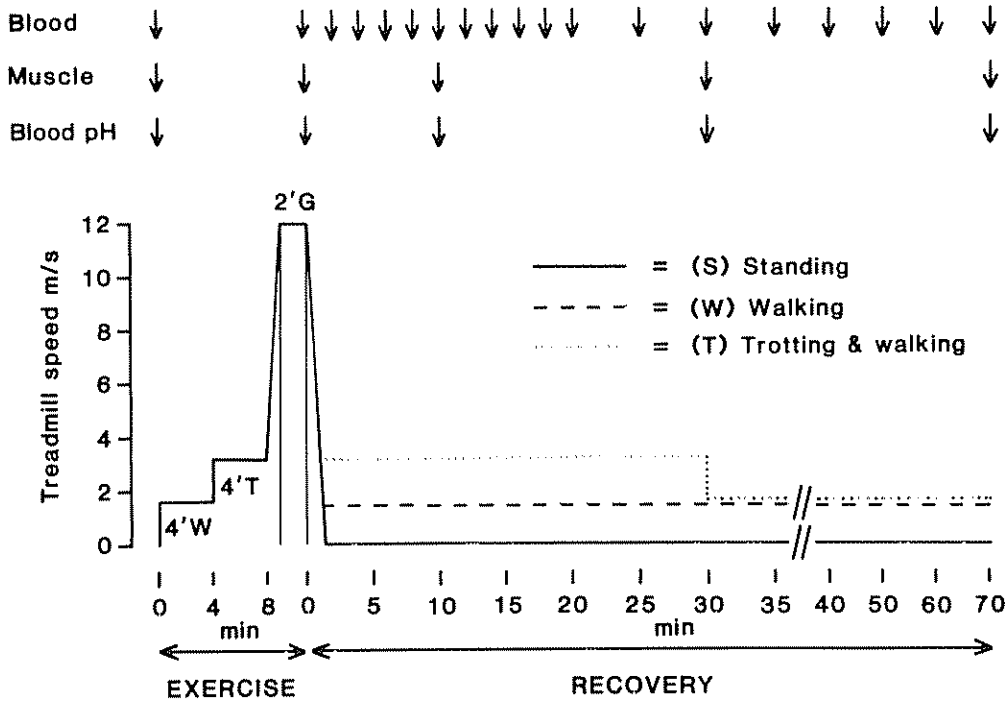


FIGURE 1. Exercise and recovery protocols. Muscle biopsies and samples for blood gas analysis were collected on (S) and (T) sessions only.

long and approximately  $1\frac{1}{2}$  cm apart, were made with a sterile surgical blade. Muscle samples, obtained with a 5 mm Bergström-Stille biopsy needle, were taken at rest, following 2 min at 12 m/sec and at 10, 30 and 70 min recovery on (S) and (T) sessions only. Samples were frozen and stored in liquid nitrogen.

**Biochemical analysis.** Approximately 40 mg of muscle tissue was freeze-dried, dissected free of connective tissue and blood to yield 10 mg of powder which was extracted in 0.5 mol/l PCA. Lactate and hexosemonophosphate (HMP) were determined on the neutralized PCA extracts (Harris *et al.*, 1974). Muscle metabolite values were corrected for blood contamination (Harris *et al.*, 1987). Lactate was determined enzymatically in the PCA extracts of whole blood and plasma (Hohorst, 1963). Glycogen was determined in a 0.5–4.0 mg sample of muscle powder as previously described (Snow *et al.*, 1985). All measurements were made on a Vitatron MPS filter photometer. Analysis of blood pH was performed in duplicate, within 2 hours of collection on a Corning 166 micro pH/blood gas analyzer.

**Statistics.** Levels of significance were determined using Student's t-test for paired and unpaired data. Data from KJ were excluded from statistical comparisons as they were incomplete. Results are presented as mean  $\pm$  SD.

## Results

**Muscle lactate.** Changes in muscle lactate with exercise and recovery are shown in Table 1. The resting mean value for the four horses on both (S) and (T) sessions was

$10.1 \pm 4.2$  mmol/kg d.m. During recovery muscle lactate contents showed essentially a mono-exponential decrease from an immediate post-exercise mean for (S) and (T) sessions of  $113.1 \pm 29.6$  mmol/kg d.m. Half-times for muscle lactate disappearance, calculated from the linear portion of semi-logarithmic plots of lactate recovery, were significantly lower ( $P < 0.02$ ) with (T) ( $15.2 \pm 5.8$  min) compared to (S) ( $27.6 \pm 8.3$  min). This represents an almost two-fold increase in the rate of muscle lactate disappearance with trotting.

**Blood lactate.** Changes in blood lactate with exercise and recovery for the four horses used are shown in Fig. 2. Mean immediate post-exercise blood lactate level for all sessions ( $n = 12$ ) was  $16.2 \pm 3.9$  mmol/l. Mean peak post-exercise blood lactate was not significantly different with (S), (W) or (T), values being  $21.2 \pm 3.0$ ,  $22.6 \pm 5.6$  and  $20.1 \pm 4.7$  mmol/l, respectively. There was a trend towards shorter times to peak with (T) ( $7.0 \pm 1.2$  min) compared to (W) ( $8.0 \pm 2.3$  min) compared to (S) ( $10.0 \pm 3.3$  min). The half-times for blood lactate disappearance with (T), (W) and (S) were  $12.2 \pm 3.9$ ,  $16.9 \pm 4.3$  and  $26.8 \pm 5.2$  min ( $T < W < S$ ,  $P < 0.01$ ). Rate constants for blood lactate disappearance were correlated to post-exercise work rate expressed in terms of treadmill speed (Fig. 3). The greatest response in blood lactate disappearance to increased post-exercise work rate was shown by SL, whilst the other 4 horses showed an almost equal response. The increase in muscle lactate with exercise was significantly ( $P < 0.01$ ) correlated to peak lactate concentration observed in blood after 5–10 min recovery (Fig. 4).

**Muscle to plasma lactate gradients.** Mean muscle to plasma lactate gradients calculated from the ratio of intracellular lactate concentration in muscle (mmol/l intracellular water, assuming a content of 3 liters of water per 4 kg of wet muscle) to the lactate concentration in plasma water (mmol/l) at rest, after exercise and during recovery, are shown in Table 2.

**Blood pH.** Venous blood pH at rest was  $7.417 \pm 0.021$  and decreased to  $7.000 \pm 0.092$  following 2 min exercise at 12 m/sec. During (T) recovery pH was significantly higher at 10 min ( $P < 0.05$ ), 30 min ( $P < 0.01$ ) and 70 min ( $P < 0.01$ ) compared to (S) as shown in Table 3.

**Glycogen and HMP.** Changes in glycogen and HMP are shown in Table 1.

### *Discussion*

In contrast to previous field studies where running speed inevitably varied with the distance covered, the horses in this study were exercised at a constant rate of 12 m/sec for 2 min. The total distance covered was 1440 m. Accumulation of lactate in the muscle at the end of the run was of the predicted order for a maximal field gallop over the same distance (Fig. 5), as was the accumulation of lactate in the blood. The changes in muscle lactate shown in Fig. 5 are taken from three previous studies in which horses were galloped maximally over 620, 800 and 2000 m (Snow *et al.*, 1985; Harris *et al.*, 1987). These findings indicate that in terms of anaerobic stress the exercise model used was comparable to a maximal gallop in the field over the same distance. This was despite the absence of the added weight of a rider, though this would have been partly compensated for by the 5° incline of the treadmill.

In the earlier 800 and 2000 m field studies (Harris *et al.*, 1987) in which the distances were covered at mean running speeds of 14.3 and 13.4 m/sec, respectively, peak blood

TABLE 1. Glycogen (mmol glucosyl units/kg d.m.), hexosemonophosphate (HMP) and lactate (mmol kg d.m.) at rest, at the end of 2 min exercise and at 10, 30 and 70 min recovery with (S) and (T)

Recovery time (min)	(S)			(T)		
	Glycogen	HMP	Lactate	Glycogen	HMP	Lactate
Pre-exercise	500 ± 72	3.80 ± 1.32	11.61 ± 5.45	566 ± 41	3.96 ± 0.87	8.66 ± 2.33
0	418 ± 59	24.18 ± 2.47	114.15 ± 33.35	443 ± 78	27.48 ± 2.71	112.13 ± 31.64
10	392 ± 55	14.79 ± 4.46	84.75 ± 18.58	454 ± 63	18.41 ± 4.64	67.91 ± 10.75
30	425 ± 79	6.35 ± 1.18	40.37 ± 16.51	475 ± 79	7.68 ± 1.38	29.80 ± 18.46
70	431 ± 76	5.15 ± 1.06	16.01 ± 3.24	488 ± 68	6.19 ± 1.65	9.22 ± 4.93

lactates of  $20.6 \pm 3.6$  and  $30.9 \pm 5.3$  mmol/l were recorded. Half-times for blood lactate disappearance during recovery, where horses were walked, were  $15.2 \pm 5.9$  and  $21.1 \pm 7.7$  min, respectively. These times compare to a value of  $16.9 \pm 4.3$  recorded in the present study with walking recovery and with mean peak-post-exercise blood lactate concentrations of  $21.3 \pm 4.7$  mmol/l. Compared to man, half-times for

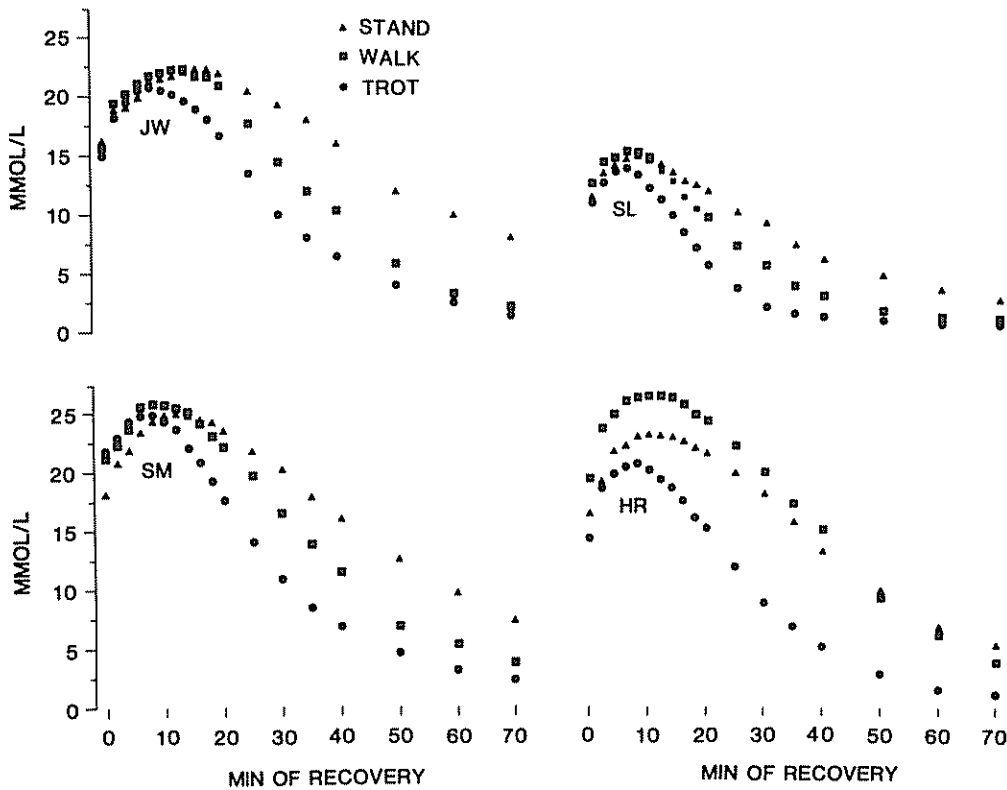


FIGURE 2. Venous blood lactate contents of 4 horses at the end of exercise and during 70 min recovery with (S), (W) and (T)

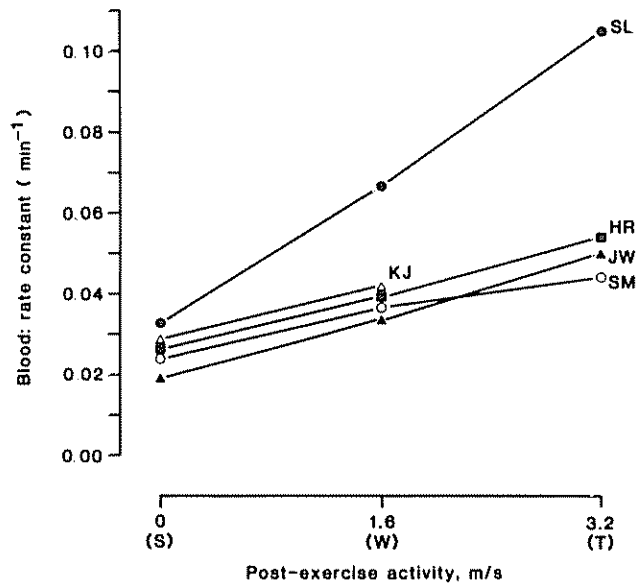


FIGURE 3. Exponential rate constant for the disappearance of venous blood lactate during recovery in relation to activity, expressed as treadmill speed, for 5 horses (SM, HR, SL, JW and KJ)

disappearance of lactate from muscle and blood tend to be somewhat longer in the horse. Half-times for lactate disappearance from the vastus lateralis of 2.5 and 9.5 min have been observed following intense isometric contraction (Harris *et al.*, 1981) and 6–11 min exhaustive bicycle exercise (Sahlin *et al.*, 1976), respectively. Immediate post-exercise muscle lactate contents in either case were of the order of 100 mmol/kg d.m. The range in half-times observed in the human studies can probably be explained on the basis that in the isometric exercise model only one major muscle group was used and that the blood perfusing the muscle during recovery would have had a lactate concentration little removed from that at rest. In the dynamic exercise study the perfusing blood would have had a much higher lactate concentration. Half-times for blood lactate disappearance in trained subjects following intermittent exercise calculated from data of Dodd *et al.* (1984) were 15.0 min with passive recovery, and 9.8 min with active recovery in which subjects continued to exercise at 35%  $\dot{V}O_{2max}$ . The effect of activity during recovery was similar to that seen in the present study with the half-time of blood lactate disappearance with trotting being about half of that when recovery was passive, i.e.  $12.2 \pm 3.9$  compared to  $26.8 \pm 5.2$  min.

The process of lactate release from muscle is still not fully understood. In the study of Dodd *et al.* (1984) the failure to obtain an early peak in blood lactate during active recovery was seen as evidence that lactate is not freely diffusible from the muscle. This appeared to agree with studies of Jordfeldt *et al.* (1978) in which increase in the translocation rate of lactate from muscle to blood was linear with intracellular concentrations up to 4 mmol lactate/kg wet muscle (about 16 mmol/kg d.m.) after which the rate of efflux was constant. This suggests that lactate transport from the muscle is carrier mediated, and that the carrier is saturated even at low concentrations of lactate. If this

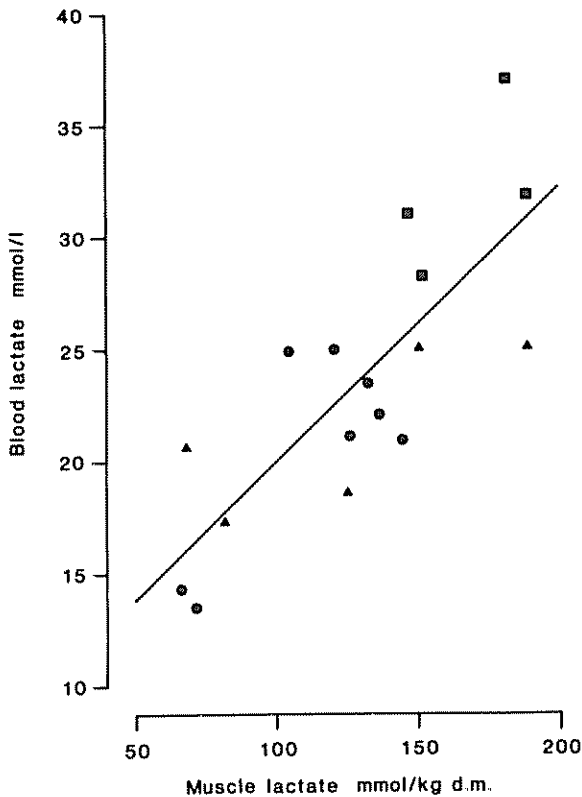


FIGURE 4. Correlation between peak-post-exercise blood lactate concentration and immediate post-exercise muscle lactate contents.  $y = 7.69 + 0.12X$ ,  $r = 0.79$ ,  $P < 0.01$ . ● = data from (S) and (T) recoveries in present study. Also shown are values following maximal gallops of 800 m (▲) and 2000 m (■) in the field (Harris *et al.* 1987).

were true, then differences in blood flow through the muscle bed would be unlikely to affect the rate of efflux, implying that the difference in muscle disappearance rates between (S) and (T) recoveries resulted from differences in the rates of local utilization, i.e. glycogen resynthesis and oxidation. Although there was some evidence of glycogen resynthesis during recovery, this was not statistically significant and did not appear to differ between (S) and (T) recoveries. Also, most of the glycogen increase could be accounted for by disappearance of muscle hexosemonophosphate. Between 0 and 30 min recovery the difference between (S) and (T) recoveries in muscle lactate disappearance amounted to approximately 30 mmol/kg d.m., sufficient for the aerobic synthesis of 495 mmol ATP/kg and support of an ATP turnover rate of 0.3 mmol/kg d.m./sec. Although the increased metabolic cost to the middle gluteal of trotting compared to standing is not known, by comparison to human rates (Harris, 1981) this is a reasonable figure if not somewhat conservative. In other words, local utilization of lactate to meet the increased metabolic cost of trotting could account for the increased rate of disappearance.

TABLE 2 Muscle to plasma lactate gradients at rest, following 2 min exercise and at 10, 30 and 70 min recovery with (S) and (T) recovery.

Recovery: time (min)	(S)	(T)
Pre-exercise		4.73** ± 2.01
0		1.45* ± 0.36
10	0.82** ± 0.07	0.77 ± 0.30
30	0.52** ± 0.12	1.25 ± 1.26
70	0.90 ± 0.86	1.40 ± 0.66

Values differing significantly from unity:

\* $p < 0.05$

\*\* $p < 0.01$

The alternative thesis is that lactate is freely diffusible across the muscle membrane and its release dependent upon the muscle to plasma gradient. This is supported by the earlier mentioned studies of muscle lactate disappearance in man following isometric and dynamic exercise, and by the exponential nature of the decrease in muscle in both man and horse.

Even considering that some of the lactate may be utilized within the muscle in which it was produced, the fate of most is clearly efflux into and redistribution by the blood. Examination of muscle to plasma gradients showed a fall from  $1.45 \pm 0.36$  following exercise to near unity after 10 min with both (S) and (T) recoveries, implying an equilibrium in terms of concentration. However, with standing recovery the gradient showed a further fall to  $0.52 \pm 0.12$  at 30 min which was significantly ( $P < 0.01$ ) lower than unity. By 70 min recovery it had risen to  $0.90 \pm 0.86$ . With (T), recovery gradients actually showed a small increase at 30 and 70 min. Neither, however, were significantly different from unity, though clearly there was an upward trend. The changes in gradients are complex and difficult to interpret, though between the two recovery modes there was a consistent trend.

TABLE 3 Venous blood pH in horses at rest, at the end of 2 min exercise and at 10, 30 and 70 min recovery with (S) and (T).

Recovery: time (min)	(S)	(T)
Pre-exercise		7.417 ± 0.021
0		7.004 ± 0.092
10	7.071* ± 0.076	7.139 ± 0.087
30	7.219** ± 0.082	7.415 ± 0.039
70	7.396** ± 0.016	7.455 ± 0.013

(S) significantly different from (T).

\* $p < 0.05$

\*\* $p < 0.01$

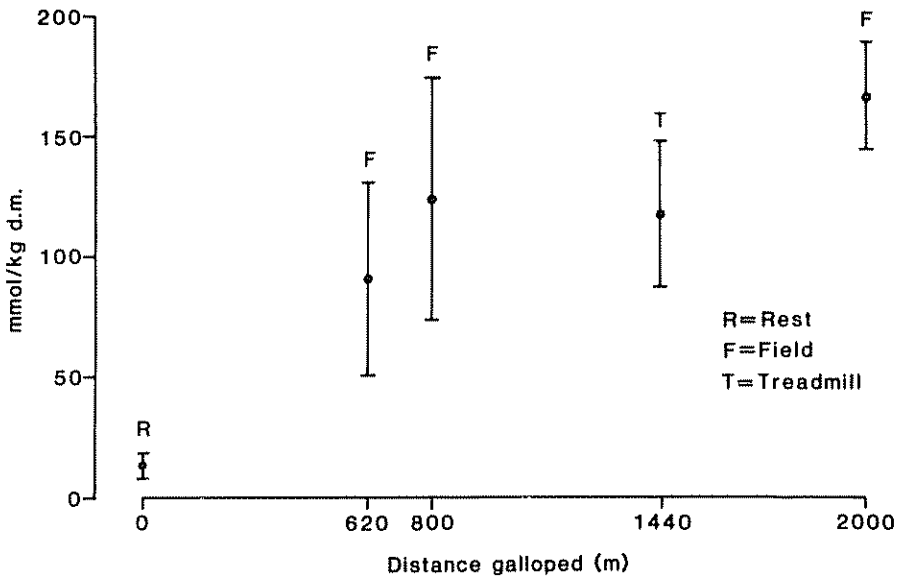


FIGURE 5 Muscle lactate contents of horses galloped maximally over 620, 800 and 2000 m in the field and over 1440 m at 12 m/sec on the treadmill (620 m—Snow *et al.*, 1985; 800 and 2000 m—Harris *et al.*, 1987; 1440 m—present study).

Quite possibly recovery in gradients is linked to recovery in muscle pH. It has been shown that in frog sartorius muscle (Seo, 1984) permeability of the muscle membranes to lactate is decreased with lowered extracellular pH. In trotting recovery (T) blood pH increased much more rapidly compared to (S) which on this basis would favor an increase in muscle lactate efflux. This may in part explain the shorter half-times for muscle lactate disappearance with (T).

The ability to rapidly remove metabolites during exercise from both muscle and blood could be considered a distinct physiological advantage to an athlete. As may be seen in Fig. 3, rate constants for blood lactate disappearance varied between the five horses as did also their response to increased activity. Throughout, SL showed the highest rate constant at each recovery mode. Horse SL was also the most capable of the five horses and was the least fatigued by the exercise. This is consistent with studies in man showing that blood lactate disappearance is increased with fitness. Paradoxically, however, SL showed the lowest rate of lactate disappearance from muscle with (S) recovery and was still one of the lowest when trotting (Fig. 6). In general, muscle disappearance rates with (S) recovery were inversely related to blood disappearance rates, as were also the proportionate changes in either with increased activity. Put simply, where the capacity of muscle to release lactate was high then the fall in concentration in blood was slower. This is exemplified by JW. In the case of SL a slower rate of lactate release from muscle with both (S) and (T) recoveries would seem to be the explanation for the faster rate of disappearance from blood. Factors which could account for the variation between individuals in muscle lactate disappearance rates are muscle perfusion (Tesch and Wright, 1983), fiber composition (Bonen *et al.*, 1979) and level of fitness (Evans and Cureton 1983). The question remains as to whether the differential response of

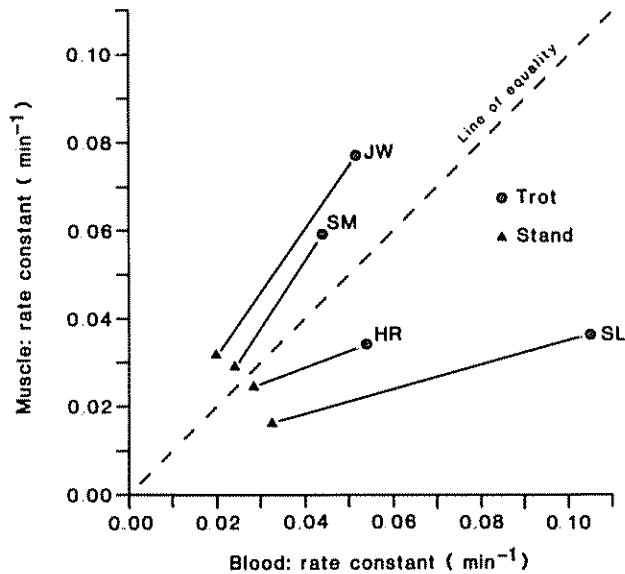


FIGURE 6 Rate constants for muscle lactate disappearance in relation to rate constants for blood lactate disappearance in 4 horses for (S) and (T) recoveries.

individuals to post-exercise activity on blood and muscle lactate disappearance rates is in any way related to their abilities as stayers or sprinters in the field.

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# Changes in Free and Bound Carnitine in Muscle with Maximal Sprint Exercise in the Thoroughbred Horse

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## Summary

*To study the changes in carnitine in muscle and blood with sprint exercise, Thoroughbreds performed a 2-min treadmill test at 12 m/sec, and field gallops of 800 m and 2000 m. Biopsies of the middle gluteal and plasma samples taken before and after exercise and during recovery were analyzed for total free and acetylcarnitine, and other metabolites. Mean muscle total carnitine content at rest was  $31.0 \pm 2.0$  mmol/kg d.m. Approximately 80% was free carnitine and 15% acetylcarnitine. Acylcarnitine was estimated at 5–10%.*

*Both the field and treadmill exercise resulted in the marked accumulation in muscle of lactate (100 mmol/kg d.m. or more) and glycerol-3-P. Accumulation of glycerol was much less, but as in other studies continued to rise during recovery. Sprint exercise (treadmill) did not affect total carnitine, but free carnitine fell from  $24.3 \pm 4.1$  to  $9.6 \pm 3.2$  mmol/kg d.m. (60% decrease,  $P < 0.01$ ) with an almost equivalent rise in acetylcarnitine. Recovery of free and acetylcarnitine took approximately 30 min. After 70 min recovery muscle total carnitine was significantly higher than at rest indicating uptake from plasma. Similar findings were obtained in the field studies.*

*It is concluded that in sprint exercise above the lactate threshold in the Thoroughbred horse, carnitine assists in the regulation of the acetylCoA/CoA ratio by buffering excess acetylCoA production.*

*Index terms: CoA; acetylCoA; aerobic metabolism.*

## Introduction

Carnitine ( $\gamma$ -trimethylamino- $\beta$ -hydroxybutyrate) is a low molecular weight, highly soluble compound with a structure similar to the amino acids. The occurrence of carnitine seems to be nearly ubiquitous in animals, many microorganisms and plants, although its concentration varies widely between species and tissues. The highest levels in mammals are found in heart and skeletal muscle tissue (Bremer 1983).

The primary role of carnitine is as a cofactor to the transport of fatty acids (FA), and in particular long chain FA, across the inner mitochondrial membrane (Fritz 1955). The