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Equine Exercise Physiology 3: 188–195, 1991

Rates of Blood Lactate Disappearance Following Exercise of Different Intensities

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ABSTRACT. Lactate was analysed in whole blood and plasma following 2 min treadmill exercise on separate occasions at 6, 7, 8, 9, 10, 11 or 12 m s^{-1} on a 5° incline. Peak concentrations of lactate in plasma were found to occur immediately post-exercise (0 min recovery) when the peak concentration was less than 20 mmol l^{-1} and at 5 min recovery between 20–46 mmol l^{-1}. In whole blood, peak lactate concentrations occurred immediately post-exercise for concentrations less than 10 mmol l^{-1}. Between 15 and 30 mmol l^{-1}, peaks occurred at both 5 and 10 min recovery. Mean blood or plasma lactate disappearance rates (mmol l^{-1} h^{-1}) did not change significantly with increasing speed. Rates of disappearance ranged from 25.1–63.0 mmol l^{-1} h^{-1} for plasma and from 16.8–33.4 mmol l^{-1} h^{-1} for whole blood.

Key words Horses; exercise; blood; lactate kinetics.

INTRODUCTION

The kinetics of blood lactate appearance and disappearance following maximal field and treadmill exercise in the Thoroughbred horse have been described previously.[10,14] Following field gallops of either 800 or 2 000 m or treadmill gallops of 1 400 m at 12 m s^{-1} (5° incline), peak post-exercise blood lactate contents were found to occur between 5 and 10 min into recovery. Blood lactate appearance and disappearance could be described by a bi-exponential equation previously used to describe postexercise lactate kinetics in man.[7,8]

In both of the previous studies, lactate kinetics following exercise were examined over a relatively narrow range of initial lactate concentrations. The present investigation was, therefore, undertaken to investigate lactate kinetics over a much wider range of peak concentrations.

MATERIALS AND METHODS

Animals Six trained Thoroughbred horses (FR—5 yo; HR—6 yo; JW—13 yo; MR—4 yo; TB—5 yo; LB—4 yo) were used in this study.

Exercise protocol Each horse performed 7 experimental treadmill sessions, each separated by at least 3 days. Each experimental session consisted of a warm-up period during which the horses were walked for 15 min at 1.6 m s^{-1}, followed by a canter for 2 min at 6 m s^{-1} and a further 5 min walk at 1.6 m s^{-1}. After the warm-up period the horses performed a 2 min test-exercise at a speed of 6, 7, 8, 9, 10, 11 or 12 m s^{-1} (see Fig. 1). Only one speed was used per experimental session and the order of the sessions was randomised. The treadmill was set at 5° at all times during the warm up and exercise periods and at 0° during the recovery period. Environmental conditions were maintained between 18–20°C and 60–70% relative humidity.

Sampling Blood samples were collected from an indwelling catheter (Becton–Dickinson, 14 g with 17 g×20 cm inner needle) located in the left or right jugular vein and attached to a 100 cm extension line (Lectrofelx, PVC, Vygon, UK, 2 ml capacity). Pa-

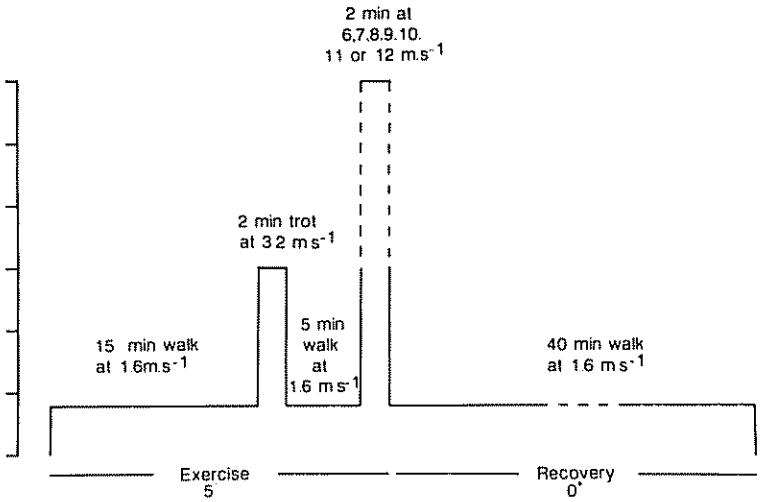

Fig 1 Exercise protocol.

tency was maintained by filling and flushing with heparinized saline.

Sample treatment and analysis. Venous blood samples (approximately 15 ml) were collected at rest (Pre), at the end of the warm up (St), immediately at the end of the test-exercise and at 5 min intervals to 40 min recovery. Approximately 2–3 ml of blood was immediately deproteinized with 5 ml of 1 mol l^{-1} perchloric acid in pre-weighed tubes. A further 10 ml was dispensed into a tube containing lithium heparin and placed on ice.

Lactate was analysed enzymically in perchloric acid extracts of whole blood or plasma by the method of Hohorst.[13]

Rates of blood lactate disappearance were calculated on the linear portion of the recovery curve by linear regression of concentration against time. Rates are expressed in mmol l^{-1} h^{-1}.

RESULTS

Changes in whole blood and plasma lactate with exercise and recovery are shown in

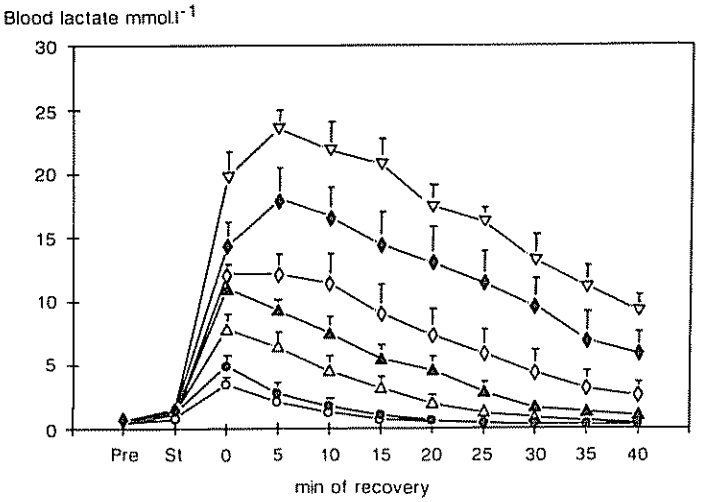

Fig 2 Time courses for whole blood lactate (mmol l^{-1}, mean ± SE, $n=6$ except 12 m s^{-1}, $n=5$) at rest (Pre), immediately before (St) 2 min treadmill exercise at 6, 7, 8, 9, 10, 11 or 12 m s^{-1} on a 5° incline, and during 40 min walking recovery (1.6 m s^{-1}, 0° incline). ○, 6 m s^{-1}; ●, 7 m s^{-1}; △, 8 m s^{-1}; ▲, 9 m s^{-1}; ◇, 10 m s^{-1}; ◆, 11 m s^{-1}; ▽, 12 m s^{-1}.

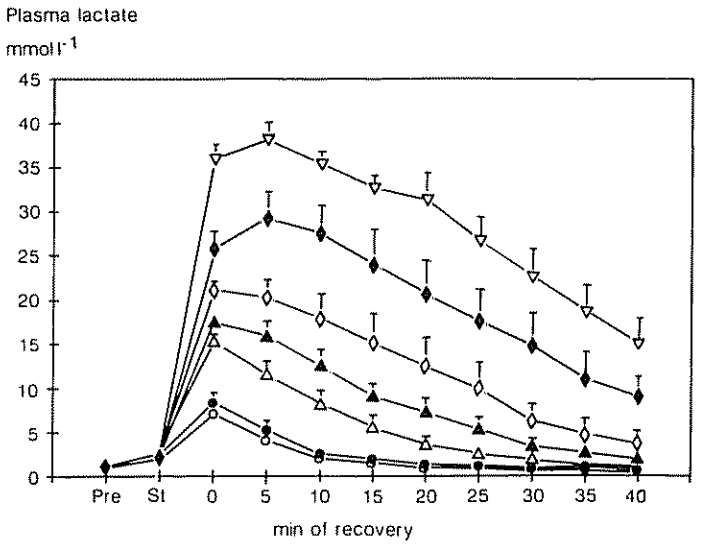

Fig. 3. Time courses for plasma lactate (mmol l^{-1}, mean ± SE, $n=6$ except 12 m s^{-1}, $n=5$) at rest (Pre), immediately before (St) 2 min treadmill exercise at 6, 7, 8, 9, 10, 11 or 12 m s^{-1} on a 5° incline, and during 40 min walking recovery (1.6 m s^{-1}, 0° incline). ○, 6 m s^{-1}; ●, 7 m s^{-1}; △, 8 m s^{-1}; ▲, 9 m s^{-1}; ◇, 10 m s^{-1}; ◆, 11 m s^{-1}; ▽, 12 m s^{-1}.

Figs. 2 and 3, respectively, and showed similar kinetics although the plasma concentrations were always higher. Peak whole blood and plasma lactate were significantly correlated ($p<0.001$, $r=0.971$, $y=0.661x-1.299$).

The time at which peak concentrations occurred in both whole blood and plasma was dependent upon the lactate concentration (Fig. 4). In plasma, where the peak concentration was below approximately 20 mmol l^{-1}, the peak occurred at 0 min recovery (immediate post-exercise). With peak concentrations between 20 and 47 mmol l^{-1}, the peak occurred after 5 min recovery. In whole blood, peaks occurred in both the 0 and 5 min samples when concentrations were approximately 10–15 mmol l^{-1} and in both the 5 and 10 min recovery samples when peak concentrations were in the range of 15–30 mmol l^{-1}.

Based on either the peak or 5 min recovery sample, mean plasma and whole blood lactate both showed an approximately exponential increase with increasing speed (Fig. 5).

Lactate disappearance following exercise (mmol l^{-1} h^{-1}) in either whole blood or plasma was virtually independent of exercise intensity (equivalent to speed) or concentra-

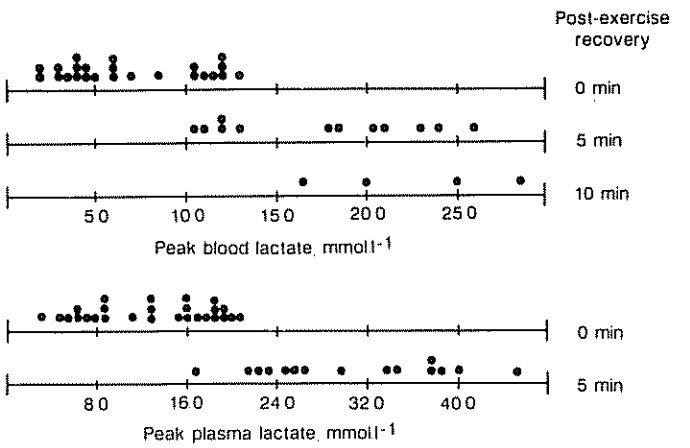

Fig. 4. Time of occurrence of peak lactate contents (mmol l^{-1}) in whole blood and plasma.

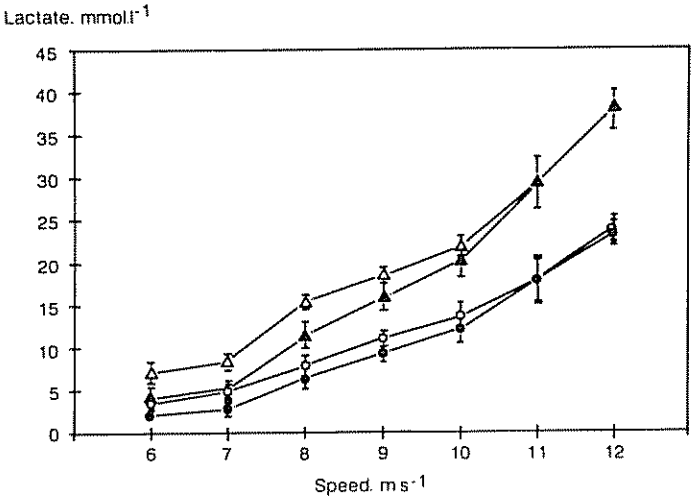

Fig. 5 Peak and 5 min recovery plasma and whole blood lactate concentrations in relation to treadmill exercise test speed (mean ± SE, $n=6$ except 12 m s^{-1}, $n=5$). △, peak plasma; ▲, 5 min recovery plasma; ○, peak whole blood; ●, 5 min recovery whole blood.

tion (Figs. 6, 7 and 8) and for the majority of time was linear (following any further increase immediately post-exercise). Rates of lactate disappearance calculated by regression over the linear portion of the recovery curves varied from 25.1–63.0 mmol l^{-1} h^{-1} for plasma and from 16.8–33.4 mmol l^{-1} h^{-1} for whole blood. There was a trend in both plasma and whole blood for the lowest disappearance rates to be at the lower lactate concentrations.

DISCUSSION

In our analysis of the changes in lactate concentrations in plasma and whole blood, we conclude that lactate disappearance during recovery is best described as a linear process. This is in contrast to inferences made in a previous publication from this laboratory.[10] We now consider it inappropriate mathematically to attempt to force-fit exponential functions to these processes (apart from in a purely descriptive sense) and to then draw

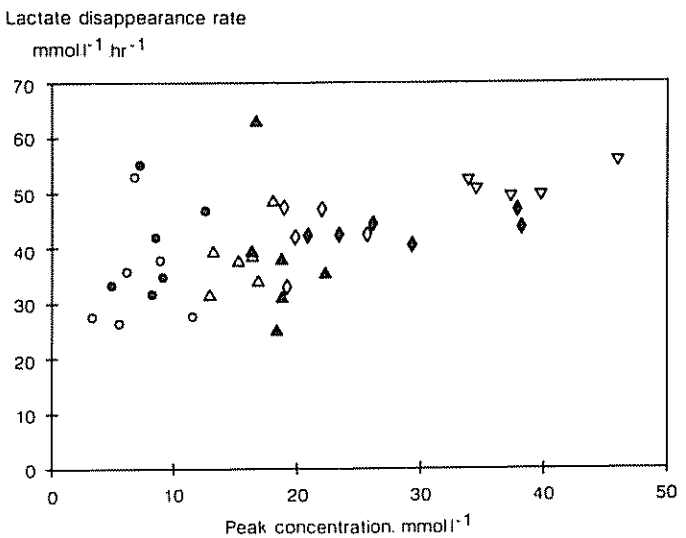

Fig. 6 Rate of blood lactate disappearance (mmol l^{-1} h^{-1}) in relation to peak concentration. ○, 6 m s^{-1}; ●, 7 m s^{-1}; △, 8 m s^{-1}; ▲, 9 m s^{-1}; ◇, 10 m s^{-1}; ◆, 11 m s^{-1}; ▽, 12 m s^{-1}.

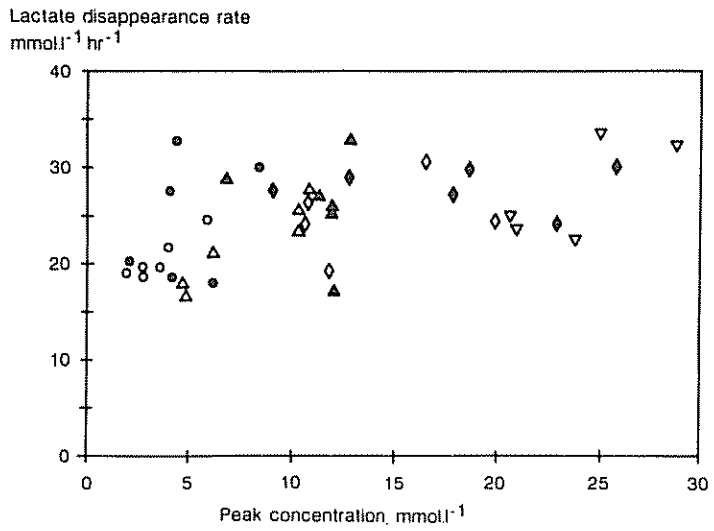

Fig 7 Rate of plasma lactate disappearance (mmol l^{-1} h^{-1}) in relation to peak concentration. ○, 6 m s^{-1}; ●, 7 m s^{-1}; △, 8 m s^{-1}; ▲, 9 m s^{-1}; ◇, 10 m s^{-1}; ◆, 11 m s^{-1}; ▽, 12 m s^{-1}

conclusions concerning half-times of disappearance. The calculation of half-times for processes which are not truly exponential can be misleading. The representation of lactate disappearance as primarily a linear process is more appropriate, easier to relate to and this concept has been applied previously to blood lactate disappearance following exercise in man.[3] These authors observed rates of lactate disappearance ranging from 16.1 to 46.4 mmol l^{-1} h^{-1} (mean rate 31.8±2.9 mmol l^{-1} h^{-1}) at a recovery exercise intensity corresponding to 40% VO_2max. This compares to a mean rate of disappearance in the present study of 25.5±5.6 mmol l^{-1} h^{-1}. The higher rates observed in man[3] may have been due to a higher level of activity during recovery compared with the activity undertaken by the horses in the present study (walking at 1.6 m s^{-1}, 0° incline—estimated to correspond to 10–15% VO_2max[18] and L. S. Anderson, personal communication). An increase in the post-exercise lactate disappearance rate with low intensity post-

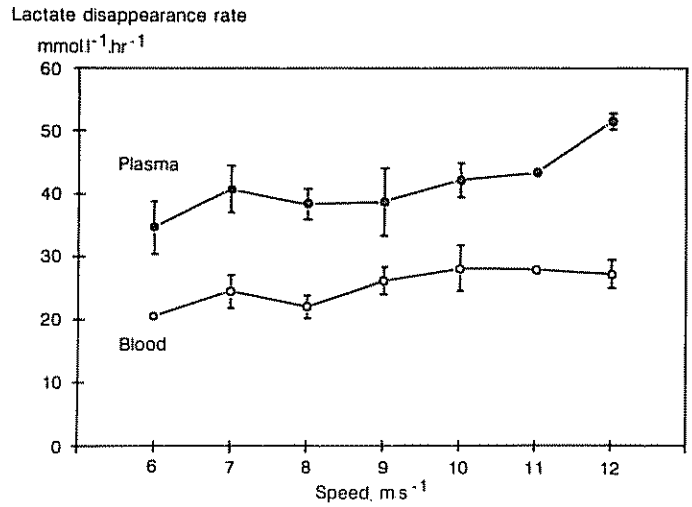

Fig 8 Disappearance rates for blood and plasma lactate (mmol l^{-1} h^{-1}, mean ± SE, $n=6$ except 12 m s^{-1}, $n=5$) during 40 min walking recovery (1.6 m s^{-1}, 0° incline) following 2 min treadmill exercise at 6, 7, 8, 9, 10, 11 or 12 m s^{-1} on a 5° incline. ●, plasma; ○, blood.

exercise activity has been demonstrated both in man[6,9] and in the horse.[14]

Another factor which may have resulted in lower rates of lactate disappearance in the horse compared with man is the generally lower concentration of type I fibres in the major locomotory muscles of the horse. It has been demonstrated that in man the rate of lactate removal is correlated to the percentage of type I fibres in the vastus lateralis.[2,3]

With many exercise studies, a fixed sample time protocol is used, especially in field investigations. In the present study, adopting a fixed sample time of 5 min post-exercise could have resulted in an underestimation of the true peak blood lactate concentration by as much as 3–4 mmol l^{-1}. This effect would have been greatest at the lower concentrations, resulting in an underestimation of up to 40%. As all peaks occurred in plasma at 5 min recovery, when the peak concentration was in excess of 20 mmol l^{-1}, no error would have been incurred in this case. However, when the peak concentration was less than 20 mmol l^{-1}, an underestimation of up to 7 mmol l^{-1} (40–50%) may occur.

As indicated in Figs. 6, 7 and 8, the removal of lactate from plasma and blood appears to be a saturable process and saturation occurs at relatively low lactate concentrations (around 10 mmol l^{-1} for whole blood and 15 mmol l^{-1} for plasma). The fall in lactate concentration in the blood and plasma is a function of several concurrent processes. Physical redistribution of lactate throughout body tissues will have an immediate effect upon the apparent concentrations in both plasma and blood. Sustained lactate disappearance, however, is essentially the result of lactate metabolism through oxidation or intramuscular gluconeogenesis. In man, these processes have been estimated to account for around 40 and 43% of the lactate disappearance, respectively.[1] In the horse, intramuscular gluconeogenesis does not appear to account for significant amounts of lactate disappearance, interpreted from the much lower rates of muscle glycogen resynthesis.[10,14] Utilization of lactate by the liver in man has been estimated to be largely inhibited by the low plasma pH and to account for only around 10% of lactate disappearance.[1]

In the absence of strong evidence for significant gluconeogenesis from lactate following exercise, the principal fate of lactate appears to be oxidation. This is in agreement with studies by Brookes and Gaesser.[4] In addition, while gluconeogenesis does not appear to account for a significant amount of lactate disappearance, the effect of increased recovery activity, and therefore oxidation, has been demonstrated.[14] Thus, similarity in rates of lactate oxidation may account for the similarity in lactate disappearance rates, independent of concentration.

A route for removal of lactate from the circulation not previously considered is the lactate lost when red blood cells are resequestered by the spleen. However, this can be estimated to account for only 5% of the blood lactate disappearance. This estimate is based on a spleen blood content of 10 l,[17] a splenic PCV of around 0.71 l^{-1}[15] and a peak blood lactate content of 25 mmol l^{-1}. Values are available for the calculation of the distribution of lactate between red blood cell and plasma compartments.[11,15] For the purposes of this estimate, an average red blood cell lactate concentration during a one hour recovery period of 10.5 mmol l^{-1} red blood cells has been assumed. Thus, the uptake of 7 l of red blood cells by the spleen during this period represents the removal of 75 mmol of lactate from the circulation. For a peak whole blood concentration of 25 mmol l^{-1} and a blood volume of around 60 l,[5,17] total lactate in the circulation would be in the order of 1 500 mmol. Thus, the removal of 75 mmol from the circulation in red blood cells resequestered by the spleen could only account for around 5% of blood lactate disappearance.

At the higher concentrations, peak plasma lactate always occurred at 5 min into recovery, whilst for blood, lactate peaks occurred both at 5 and 10 min. It can be calculated

that in a hypothetical situation following exercise where there is no redistribution of lactate between the blood and the tissues (i.e. no loss of lactate from the circulation) then the increase in water space per litre of blood due to the re-uptake of red blood cells into the spleen will in itself result in an apparent increase in the whole blood lactate concentration. This is despite the removal of some of the lactate to the spleen within the red blood cells. Based on values previously presented[11] this apparent rise may be as high as 25% for a change in PCV from 0.7 to 0.51 l^{-1} during recovery.[15] In reality, however, such an increase will never be realized as redistribution will be taking place during this period. Even so, the influence of red blood cell removal probably accounts for the longer times to peak (5–10 min) seen in some whole blood values. Although this probably only represents a small error, it may be an argument for measuring plasma lactate, especially when examining kinetics. In the majority of situations, either whole blood or plasma lactate measurements are probably adequate as long as the same is used throughout. It would obviously be inappropriate to compare plasma and whole blood values.

Intermittent exercise models appear to alter lactate removal kinetics,[10,16] but this may be due to an apparent prolongation of the time to peak due to continued efflux from muscle. As reported by Harris et al.,[12] the mean rate of blood lactate disappearance following 4 gallops of 700 m of 22.2±6.8 mmol l^{-1} h^{-1} is very close to that seen in the present study.

In conclusion, the majority of lactate disappearance from blood appears to occur at a linear rate as opposed to at a true exponential rate. The plateauing of values in both blood and plasma may be a biological artefact caused by continued efflux from muscle, redistribution in tissues and changes in water content of the blood. The processes for lactate removal also appear to be saturable, saturation occurring in blood and plasma at approximately 10 and 15 mmol l^{-1}, respectively.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge support given by the Horserace Betting Levy Board, London.

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Reproducibility and Validity of VLA4 in Standardbred Pacer Horses on Track

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ABSTRACT. The reproducibility of the speed producing a blood lactate concentration of 4 mmol l^{-1} (VLA4) was evaluated in 7 Standardbred pacer horses on track, using a 3 min stepwise increasing speed protocol. The validity of VLA4 was assessed using a 5 min exercise step protocol. The lactate concentration of arterial blood (AB) was used. The reproducibility of VLA4 based on a test/retest experiment was doubtful (r=0.38, p-value = 0.62). There was no significant difference between mean values of VLA4. No horse reached a blood lactate steady state while running at VLA4. Fatigue time ranged from 10 to 40 min. It is concluded that the reproducibility of VLA4 on track is not acceptable, whereas mean values of VLA4 are very similar. Furthermore, the validity of VLA4 could not be confirmed with this exercise test protocol.

Key words: Horses; exercise test; blood lactate.

INTRODUCTION

It has been suggested that the intensity of continuous running in exercising horses can be best established using cardiorespiratory[3,5] and metabolic parameters.[11] However, the maximum intensity that can be used safely by a competent trainer, in terms of fatigue-related injuries, is referred to as the intensity just before the redline area[5] or that producing maximum blood lactate steady state (MLSS). As in the human,[4] it has been suggested that running a horse at a speed corresponding to a blood lactate concentration of 4 mmol l^{-1} (VLA4), determined using a stepwise increasing speed protocol, would produce MLSS.[11]

The validity of VLA4 has been the object of only one study.[11] They found VLA4 to provide a reasonable estimate of steady state conditions in the horse, although some questions regarding their materials and methods remain. The reproducibility of VLA4 has been reported to be less than for oxygen uptake variables and pulse/work relationships,[8] although the type of blood collected and other details of the exercise test protocol used, i.e. exercise step duration, pause period, running surface and exercise step progression, were not mentioned. How long a horse can exercise at VLA4 has not been studied.[10]

Even if some believe that horses running at VLA4 are at a blood lactate (La) steady state, we are not convinced, still less than they are at MLSS. Thus, the purpose of this study was to assess the validity of the concept of VLA4, i.e. running a horse at a speed corresponding to a La concentration of 4 mmol l^{-1}, determined using a stepwise increasing speed protocol, would produce MLSS. The study also assessed the reproducibility of VLA4 under the constraint of measuring it on track.

There is no standardized exercise test protocol established to determine VLA4. In this study, we arbitrarily selected a 3 min stepwise increasing speed protocol to assess the reproducibility of VLA4 in Standardbred pacer horses on track. The idea being that, at VLA4 we could predict MLSS during prolonged exercise. The validity of VLA4 was