

Effects of Training Intensity on Maximum Oxygen Uptake

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ABSTRACT. We examined the effects of training intensity on $\dot{V}O_2\text{max}$ and the time course of changes with training and detraining. After 4 months rest, 10 Thoroughbred horses were trained on a treadmill inclined at 6° slope, at speeds equivalent to 40% (slow) and 80% (fast) of $\dot{V}O_2\text{max}$ over a daily distance of 3000 m, 6 days a week for 6 weeks. Both groups increased their $\dot{V}O_2\text{max}$ by about 10% during the first 2 weeks of training, from mean pre-training values (\pm SEM) of $149 \pm 4.1 \text{ ml kg}^{-1} \text{ min}^{-1}$ and $142 \pm 5.6 \text{ ml kg}^{-1} \text{ min}^{-1}$, respectively, in the slow and fast groups. There was no difference in $\dot{V}O_2\text{max}$ between the two groups and no further increase in $\dot{V}O_2\text{max}$ occurred during the subsequent 4 weeks of training. After 2 weeks of detraining, $\dot{V}O_2\text{max}$ decreased towards pre-training levels. We concluded that at a total daily exercise distance of 3000 m, the rate of change of $\dot{V}O_2\text{max}$ was not markedly affected by training intensity, when the intensity was submaximal and the percentage of $\dot{V}O_2\text{max}$ at which the horses are trained remains constant. However, short term training can result in rapid adaptations of aerobic capacity.

Key words. Horses; oxygen uptake; training; plasma volume; haemoglobin

INTRODUCTION

While there is only one report of training at submaximal intensities producing an increase in maximum oxygen uptake ($\dot{V}O_2\text{max}$) in horses,⁴ in a number of investigations there is circumstantial evidence suggesting that $\dot{V}O_2\text{max}$ increases in response to training.^{1,12–14} It remains to be determined how the training-induced increases in the functional capacity of various steps in the oxygen transport chain are related to increases in $\dot{V}O_2\text{max}$. Training may result in a coordinated enhancement of the activity of each step in the oxygen transport chain. Alternatively, training may increase the capacity of a single limiting step closer to the functional capacity of other steps in the chain. In a previous study, the increase in aerobic capacity was associated with an increase in maximum stroke volume,⁴ however, there is no information about the time course of changes in $\dot{V}O_2\text{max}$ in the horse or the influence of training intensity.

The aim of this study was to investigate

the hypothesis that training horses at an intensity approaching $\dot{V}O_2\text{max}$ would result in a greater and more rapid increase in $\dot{V}O_2\text{max}$ than that in horses undergoing lower intensity training. Furthermore, as most training investigations in the horse have evaluated effects of progressive increases in exercise intensity, we questioned whether submaximal training at the same percentage of $\dot{V}O_2\text{max}$ would result in increases in measurements of fitness as training progressed.

MATERIALS AND METHODS

Ten Thoroughbred horses, 9 geldings and 1 filly, were used. Their ages ranged from 2 to 9 years (4.8 ± 0.7 years, mean \pm SEM). Because of problems unrelated to the training programme, only 8 horses completed the study. Prior to training, all horses had been resting for 4 months and were confined to yards for 3 months before the study. Before the period of rest, all horses had received

regular treadmill exercise once or twice per week. All horses previously had one of their carotid arteries surgically relocated to a subcutaneous position.

The experiment began with a 1 week acclimatisation period of exercise on a treadmill (Beltalong, Euroa, Australia) inclined at 10% slope. The horses were given short (<2 min) daily sessions of walking and slow trotting, while wearing a respiratory gas collection mask. The gas collection apparatus used was a flow through system utilising a mask without valves,⁸ for measuring oxygen uptake ($\dot{V}O_2$). The coefficient of variation for $\dot{V}O_2$ measurement using this system was about 5%.

At the completion of the acclimatisation period, the horses were given an incremental exercise test to determine their $\dot{V}O_{2\max}$.⁹ Arterial and mixed venous blood samples were collected from catheterised carotid and pulmonary arteries.⁴ Heart rate (HR) was determined using telemetry electrocardiography.³ The test consisted of 3 min at 4 m s⁻¹, 90 s at 6, 8 and 10 m s⁻¹, 60 s at 11 m s⁻¹ and 60 s at 12 m s⁻¹. Not all horses completed the test, which was terminated when the horse could no longer keep pace with the speed of the treadmill.

During the last 5–10 seconds at each of the exercise speeds, arterial and mixed venous samples were collected anaerobically into heparinised syringes, HR was recorded and expired gases were collected for measurement of $\dot{V}O_2$. Blood gas and acid base analyses and haemoglobin concentrations were performed on the arterial and mixed venous blood using a blood gas machine (ABL 3, Radiometer, Copenhagen). Using the machine's in-built computer, oxygen contents of arterial (CaO₂) and mixed venous blood (C $\bar{v}O_2$) were calculated. A thermistor, placed via the jugular vein to the level of the right atrium,⁸ allowed measurement of central venous blood temperature for correction of blood gas and pH values. At the completion of the incremental exercise test, a venous blood sample for haematocrit determination was collected into a tube containing dipotassium ethylenedia-

minetetraacetic acid. Evans blue dye was then injected through a jugular venous catheter for determination of blood volume by the dye dilution technique.⁶ Stroke volume (Vs) was calculated using the direct Fick method.

Following the incremental tests, a $\dot{V}O_2$ versus speed regression equation over the linear region of the data (4–10 m s⁻¹) was calculated for each horse. Using this equation, the treadmill speed equivalent to 40% and 80% $\dot{V}O_{2\max}$ was calculated for each horse. The horses were randomly allocated to two groups. Half were trained at 40% max (slow group), the other at 80% $\dot{V}O_{2\max}$ (fast group). Training speeds on the inclined treadmill ranged from 3 to 5 m s⁻¹ in the slow group and from 7 to 9 m s⁻¹ in the fast group. Each training session consisted of horses undergoing a 3 min warm up at a treadmill speed equivalent to 25% $\dot{V}O_{2\max}$, followed by acceleration to their predetermined training speed, at which they exercised for a distance of 1500 m. Training sessions were given twice daily, 6 days a week. At the beginning of the third and fifth weeks of training, the horses were given incremental tests to determine $\dot{V}O_{2\max}$, and $\dot{V}O_2$ vs speed regression equations. The recalculated $\dot{V}O_2$ vs speed regression equation was used to adjust training speed to ensure that horses continued to train at the same relative work intensity. Where HR was measured, HR vs speed regression equations were used to determine the speed at HR of 200 bpm (V_{200}).

At the end of the 6 week training period, the horses were catheterised as described for the pre-training test and given an incremental exercise test which included the range of measurements described for the pre-training test.

During the 6 week detraining period, the horses were confined in their yards. They were given incremental tests at the beginning of the third, fifth and seventh weeks of detraining. The tests at the third and fifth weeks examined changes in $\dot{V}O_{2\max}$, whereas the end of detraining test involved the

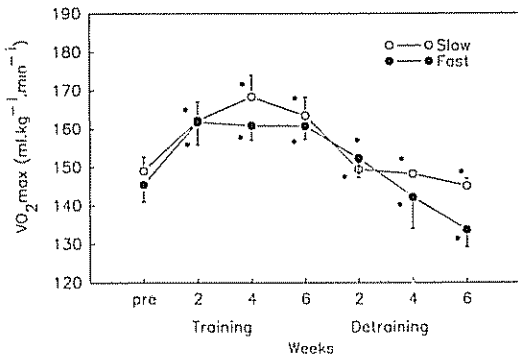


Fig 1 Maximum oxygen uptake ($\dot{V}O_{2\max}$) values for horses during training and detraining. Fast group was trained at 80% $\dot{V}O_{2\max}$, slow group at 40% $\dot{V}O_{2\max}$. Values presented as mean \pm SEM. * Indicates a significant difference from pre-training value ($p < 0.01$)

same measurements as those prior to, and at the end of training.

The data were analysed by a repeated measures analysis of variance to determine whether there were significant effects of training state or training intensity on the values measured. The results are presented as mean \pm SEM.

RESULTS

Training resulted in an increase in $\dot{V}O_{2\max}$ for all horses. The results of the changes in $\dot{V}O_{2\max}$ are presented in Fig. 1. The mean pre-training $\dot{V}O_{2\max}$ (all $\dot{V}O_2$ given in STPD) was 149.1 ± 4.1 ml kg⁻¹ min⁻¹ for

horses allocated to the slow training group, and 142.4 ± 5.6 ml kg⁻¹ min⁻¹ for horses allocated to the fast group. The two values were not statistically different. After 2 weeks of training, mean $\dot{V}O_{2\max}$ had increased ($p < 0.01$) to 162.1 ± 5.6 ml kg⁻¹ min⁻¹ for the slow group, and to 158.2 ± 7.0 ml kg⁻¹ min⁻¹ for the fast group. There was no further change in mean $\dot{V}O_{2\max}$ over the course of the training programme. At the end of training, mean $\dot{V}O_{2\max}$ in the slow group was 163.6 ± 5.4 ml kg⁻¹ min⁻¹, and 157.2 ± 5.3 ml kg⁻¹ min⁻¹ in the fast group.

After 2 weeks of detraining, mean $\dot{V}O_{2\max}$ had fallen ($p < 0.01$) to 149.5 ± 2.0 ml kg⁻¹ min⁻¹ for horses in the slow group and 147.9 ± 6.9 ml kg⁻¹ min⁻¹ for horses in the fast group, neither of which were different from the pre-training value. Mean $\dot{V}O_{2\max}$ continued to fall throughout the detraining period. At the end of the detraining period, mean $\dot{V}O_{2\max}$ for both groups was 138.4 ± 4.8 ml kg⁻¹ min⁻¹ which was lower than the mean pre-training value ($p < 0.05$).

There was no difference in the mean $\dot{V}O_{2\max}$ values between the training groups at any stage of training or detraining. The slopes of the $\dot{V}O_2$ vs speed regression were unchanged throughout training or detraining.

The haematocrit (PCV) and haemoglobin concentration increased during training and detraining (Table 1) while plasma volume increased during training in both groups ($p < 0.005$), from a mean of 22.9 ± 0.8 l be-

Table 1. Effect of training state on haematological and cardiovascular parameters at $\dot{V}O_{2\max}$ (mean \pm SEM)

Parameter	Pre-training	End training	End detraining
PCV (l l ⁻¹)	0.60 \pm 0.01	0.60 \pm 0.01	0.62 \pm 0.01*
C(a - \bar{v})O ₂ (ml dl ⁻¹)	23.9 \pm 0.5	25.2 \pm 0.5*	25.6 \pm 0.6*
Hb (g l ⁻¹)	255.5 \pm 8.3	244.5 \pm 3.6*	246.5 \pm 3.5*
TBV (l)	53.3 \pm 0.8	63.9 \pm 1.8**	72.6 \pm 4.6**
Vs (ml)	1 391 \pm 54	1 426 \pm 50	1 271 \pm 68*

Significant differences from pre-training values: * $p < 0.05$, ** $p < 0.01$.

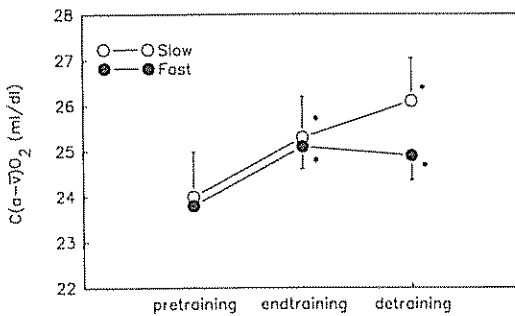


Fig. 2 Arteriovenous oxygen content differences ($C(a-\bar{v})O_2$) in horses before and after training and after detraining. Fast group was trained at 80% $\dot{V}O_{2\max}$, slow group at 40% $\dot{V}O_{2\max}$. Values presented as mean \pm SEM. * Indicates a significant difference from pre-training values ($p < 0.05$).

fore training to 27.7 ± 1.8 l at the end of training. There was no change in plasma volume during the period of detraining (Fig. 2).

There was no significant effect of training on V_{200} in either of the groups. V_{200} values ($m s^{-1}$) during training were as follows: 7.59 ± 0.41 (pre-training), 8.53 ± 0.29 (end of training) and 8.58 ± 0.31 (end of detraining).

The end of detraining value for V_s was lower than the pre-training and end of training values (Table 1). There was no effect of training regimen on the changes in V_s in either group. There was a progressive increase in V_s with increasing exercise intensity from 1060 ± 57 ml beat⁻¹ at 4 m s⁻¹ to 1330 ± 57 ml beat⁻¹ at 10 m s⁻¹ ($p < 0.01$).

Arteriovenous oxygen content difference ($C(a-\bar{v})O_2$) increased with increasing exercise intensity. Maximum $C(a-\bar{v})O_2$ was higher at the end of training (25.2 ± 0.5 ml dl⁻¹) and at the end of detraining (25.6 ± 0.6 ml dl⁻¹) than the pre-training value (23.9 ± 0.5 ml dl⁻¹) ($p < 0.05$) (Fig. 3).

DISCUSSION

Training at low exercise intensities may enhance aerobic capacity as, in this study, increases in $\dot{V}O_{2\max}$ were found after only 2 weeks training at a relatively low exercise

intensity. The exercise load chosen was that which we thought the horses could sustain in the untrained state. It is reported that $\dot{V}O_{2\max}$ increased by 23% during training, associated with an increase in V_s .⁴ In contrast to those findings, the increase in $\dot{V}O_{2\max}$ observed in this study was associated with an increase in the $C(a-\bar{v})O_2$.

CaO_2 was higher at the end of training compared with the pre-training values, while $C\bar{v}O_2$ tended to be lower, although this change was not significantly different. There are a number of possible reasons for the lack of change in V_s during training in the current study. The relatively small increase in $\dot{V}O_{2\max}$ and the indirect method for determining V_s may have meant that small changes in V_s were not detectable. Additionally, the low intensity and duration of training in the current study, compared to an earlier investigation,⁴ may have been insufficient to produce an increase in the maximum V_s measured. The failure to detect a change in V_{200} contrasts with other training studies, where decreases in HR at submaximal intensities were found following training,^{1,13} which could suggest that little cardiovascular adaptation had taken place. An increase in maximum V_s , without a decrease in submaximum HR during training has been found,⁴

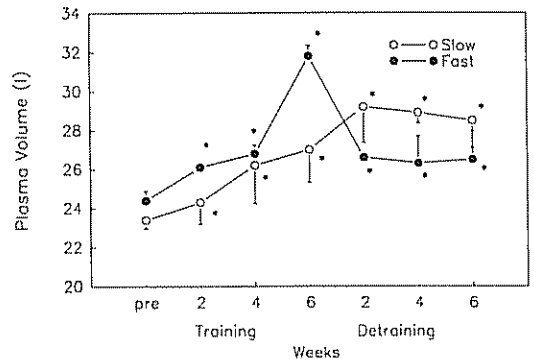


Fig. 3 Plasma volumes of horses during training and detraining. Fast group was trained at 80% $\dot{V}O_{2\max}$, slow group at 40% $\dot{V}O_{2\max}$. Values presented as mean \pm SEM. * Indicates a significant difference from pre-training value ($p < 0.01$).

while no change in V_s together with a training induced exercise bradycardia has also been reported.¹ Further work is needed to resolve the intensity and duration of training necessary to produce a change in V_s at $\dot{V}O_2\text{max}$.

The higher CaO_2 following training was due to an increase in $[Hb]$, while the saturation was unchanged. The increase in the oxygen carrying capacity of the arterial blood is in accordance with previous findings,⁷ reporting a correlation coefficient of 0.74 for total haemoglobin concentration with training state. Plasma volume at the end of training in the current study was 8% higher than the pre-training values. The unchanged PCV values throughout training indicate that an increase in red cell mass occurred, not simply an expansion of plasma volume due to water retention.⁵ Coinciding with the increase in plasma and red cell volume was a 9% increase in $\dot{V}O_2\text{max}$. However, $\dot{V}O_2\text{max}$ did not increase further during the training period, although plasma volume was higher at the end of training than after 2 weeks. During detraining, $\dot{V}O_2\text{max}$ decreased while plasma and red cell volumes were unchanged, suggesting that changes in $\dot{V}O_2\text{max}$ and plasma volume may not be related.

Mean $C\dot{v}O_2$ decreased by almost 33% at the end of training compared with the pre-training value, suggesting that training also enhances the capacity of the muscle to extract oxygen from the blood. Several factors could contribute to this effect. These include increased perfusion due to increased plasma volume, redistribution of blood flow to active tissues and enhanced muscle oxidative capacity.

The relatively high pre-training and detraining $\dot{V}O_2\text{max}$ values could reflect the high residual capacity of the cardiovascular system. In the horses used for the present study, muscle capillary density was higher than has been reported in previous studies.¹¹ The mean pre-training $\dot{V}O_2\text{max}$ of 146 ml $\text{kg}^{-1} \text{min}^{-1}$ compared with a mean value of 129 ml $\text{kg}^{-1} \text{min}^{-1}$ previously reported.⁴ However, mean $\dot{V}O_2\text{max}$ at the end of this

study and an earlier one⁴ was 160 ml $\text{kg}^{-1} \text{min}^{-1}$. A mean value of 154 ml $\text{kg}^{-1} \text{min}^{-1}$ has been reported in mature fit racing Thoroughbreds.¹⁰

It has been shown that the increases in $\dot{V}O_2\text{max}$ produced by training are not as large in previously trained individuals as in untrained subjects.¹¹ Additionally, a brief period of detraining produces a more rapid decline in $\dot{V}O_2\text{max}$ in human subjects with a high $\dot{V}O_2\text{max}$ or extensive training history.¹¹ In these subjects, changes in $\dot{V}O_2\text{max}$ may be more closely related to changes in the more labile elements of the oxygen transport chain, such as oxidative enzyme activity, plasma volume or vasomotor control. However, decreases in capillary density may also contribute. Cardiovascular adaptations may persist in detrained animals, with increases in $\dot{V}O_2\text{max}$ during training being more closely associated with increased ability to utilise the pre-existing cardiovascular capacity, and consequently following a more rapid time course than in previously untrained subjects.

From a practical training perspective, several important points emerge. The intensity of training, at the workloads used in the current study, does not appear to affect the rate of increase of $\dot{V}O_2\text{max}$. This could be because of an interaction between training intensity and duration, the horses in the slow group being exercised for about twice the duration of fast group. Further increases in aerobic capacity, if possible, may rely on increases in training distance, or longer training periods which elicit increases in one or more of the elements of the oxygen transport system. While $\dot{V}O_2\text{max}$ increased rapidly in response to training, it also decreased quickly with detraining, which may be important when considering the effects of interruptions to training schedules. It has been found that increases in the activities of oxidative enzymes associated with 5 weeks training in Standardbred horses persisted throughout 5 weeks of detraining.²

In conclusion, this study demonstrated that short term training at 2 submaximal exercise intensities performed with the rela-

tive workload unchanged throughout training resulted in rapid but small increases in $\dot{V}O_2\text{max}$. Within 2 weeks of the onset of detraining, the $\dot{V}O_2\text{max}$ values returned towards values prior to training. These findings suggest that short term interruptions to training schedules in horses may have a deleterious effect on aerobic capacity.

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