Effects of training on maximum oxygen consumption of ponies

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Objectives—To establish maximum oxygen consumption ($\dot{V}O_2max$) in ponies of different body weights, characterize the effects of training of short duration on $\dot{V}O_2max$, and compare these effects to those of similarly trained Thoroughbreds.

Animals—5 small ponies, 4 mid-sized ponies, and 6 Thoroughbreds.

Procedure—All horses were trained for 4 weeks. Horses were trained every other day for 10 minutes on a 10% incline at a combination of speeds equated with 40, 60, 80, and 100% of $\dot{V}O_2max$. At the beginning and end of the training program, each horse performed a standard incremental exercise test in which $\dot{V}O_2max$ was determined. Cardiac output ($\dot{Q}$), stroke volume (SV), and arteriovenous oxygen content difference (C [a-v] O_2) were measured in the 2 groups of ponies but not in the Thoroughbreds.

Results—Prior to training, mean $\dot{V}O_2max$ for each group was 82.6 ± 2.9, 97.4 ± 13.2, and 130.6 ± 10.4 ml/kg/min, respectively. Following training, mean $\dot{V}O_2max$ increased to 92.3 ± 6.0, 107.8 ± 12.8, and 142.9 ± 10.7 ml/kg/min. Improvement in $\dot{V}O_2max$ was significant in all 3 groups. For the 2 groups of ponies, this improvement was mediated by an increase in $\dot{Q}$; this variable was not measured in the Thoroughbreds. Body weight decreased significantly in the Thoroughbreds but not in the ponies.

Conclusions and Clinical Relevance—Ponies have a lower $\dot{V}O_2max$ than Thoroughbreds, and larger ponies have a greater $\dot{V}O_2max$ than smaller ponies. Although mass-specific $\dot{V}O_2max$ changed similarly in all groups, response to training may have differed between Thoroughbreds and ponies, because there were different effects on body weight. (*Am J Vet Res* 2000; 61:986–991)

Maximum oxygen consumption ($\dot{V}O_2max$) is usually regarded as the best predictor of cardiovascular fitness.[1,2] In comparison to other athletic animals, Thoroughbreds and Standardbreds have $\dot{V}O_2max$ values that are high (usually > 130 ml/kg • min^{-1}) and that increase significantly in response to relatively short periods of exercise training.[1,3-6] In Thoroughbreds, increases of 13 to 25% have been observed following 2 to 7 weeks of training.[1-5] It has been demonstrated that these increases are attributable to an increase in **cardiac output ($\dot{Q}$)** as a result of an increase in **stroke volume (SV)**[1-3,5] or to a widening of the **arteriovenous oxygen concentration difference (C[a-v]O_2)**.[4]

Ponies are sometimes used to study effects of strenuous exercise in horses. However, $\dot{V}O_2max$ values have not been widely reported for ponies, nor has the effect of training on $\dot{V}O_2max$ of ponies been investigated. Thus, the validity of applying findings from studies of ponies to larger, more athletic breeds of horses is not clear. To facilitate comparisons between the responses of ponies and athletic breeds of horses to exercise, the effect of 4 weeks of training on $\dot{V}O_2max$ in 2 groups of ponies was investigated and compared with the response observed in Thoroughbreds undergoing training at the same work intensities. The purposes of the study reported here were to establish $\dot{V}O_2max$ for ponies, to demonstrate how improvements in oxygen usage were mediated in 2 groups of ponies, and to compare the effect of training on $\dot{V}O_2max$ for 2 groups of ponies and a group of Thoroughbreds that were trained under similar conditions.

Materials and Methods

Horses—Two groups of ponies and 1 group of Thoroughbreds were used. All ponies were in good physical condition, whereas the Thoroughbreds had gained some weight while turned out at pasture for the previous 2 to 5 months following their last period of regular exercise. Group 1 consisted of 5 adult Shetland or Shetland-cross ponies with a mean (± SEM) pretraining body weight of 171.4 ± 6.5 kg and ranged in age from 3 to 7 years. Group 2 consisted of 4 larger mixed-breed adult ponies with a mean pretraining body weight of 318.8 ± 12.9 kg and ranged in age from 2 to 10 years. Group 3 consisted of 6 adult Thoroughbreds with a mean pretraining body weight of 487.3 ± 11.7 kg and ranged in age from 3 to 14 years. Thoroughbreds were involved in a concurrent study that precluded the insertion of catheters for obtaining blood samples. Thus, only changes in $\dot{V}O_2max$ could be assessed in them. All animals were judged to be clinically healthy by hemogram and physical examination findings, which included endoscopy of the upper portion of the respiratory tract. Each pony in group 1 had the left carotid artery translocated to a subcutaneous position in the neck at least 1 month prior to the start of the study. Ponies in group 2 were owned by another academic department, and relocation of segments of their carotid arteries was not possible. All horses were acclimated to exercise on a treadmill[a] while wearing a respiratory gas collection mask. None of the

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ponies had been previously trained, whereas the Thoroughbreds had been trained on the treadmill. All animals had ad libitum access to alfalfa, grass hay, and water.

Training—All groups were trained for 4 weeks, using a protocol similar to that used for Thoroughbreds[1,3-5,b,c] (**Table 1**). Training speeds were based on each horse's $\dot{V}O_2max$, which was determined on an inclined (10%) treadmill at the beginning of each week, using an incremental step test similar to one commonly used for such measurements in horses.[7] All horses had a warm-up period of 2 minutes at 2 m/s (ponies) or 4 m/s (Thoroughbreds). For ponies, this was followed by a 1 m/s increase in treadmill velocity every 60 seconds until the pony could no longer maintain its speed on the treadmill, or until the fractional concentration of expired oxygen reached a plateau or decreased despite an increase in treadmill speed. Thoroughbreds followed a similar protocol, with the exception that treadmill speed was initially increased in 2 m/s increments, up to 8 m/s. Thereafter, velocity was increased in 1 m/s increments. Maximum oxygen consumption was defined as having been reached when there was a plateau or drop in the $\dot{V}O_2$ versus treadmill speed relationship.

After completing this incremental step test, a $\dot{V}O_2$ versus speed regression equation for the linear portion of the curve was determined for each horse. From this equation, speeds that induced 40, 60, 80, and 100% of $\dot{V}O_2max$ were calculated. Horses were trained every other day, 4 d/wk for 10 minutes on a 10% incline. Training speeds and overall training intensity were increased each week (Table 1).

Exercise testing—At the beginning and end of the training period, all horses underwent an incremental step test, similar to that used for determination of $\dot{V}O_2max$. Oxygen consumption and **carbon dioxide production ($\dot{V}CO_2$)** were measured in all groups. **Heart rate (HR)**, arterial and mixed-venous oxygen contents, and hemoglobin and arterial lactate concentrations were measured in groups 1 and 2 only. All tests were performed with the treadmill at a 10% incline. The exercise test began with a 2-minute warm-up period, at 2 m/s for the ponies or 4 m/s for the Thoroughbreds. For the ponies, the warm-up period was followed by a 0.5 to 1.0 m/s increase in treadmill velocity every 60 seconds until the pony could no longer stay at that speed or until $\dot{V}O_2max$ was achieved. After warming up, Thoroughbreds ran at 6 and then 8 m/s for 60 seconds, followed by subsequent increases of 1 m/s until $\dot{V}O_2max$ was achieved. All measurements and blood samples were obtained before the exercise period began and during the last 15 seconds of exercise at each intensity until $\dot{V}O_2max$ was achieved. A large fan was placed in front of the treadmill, with 2 smaller fans on either side of the horse, to assist thermoregulation. The treadmill was housed in a temperature and humidity controlled room (20 to 22 C; 25 to 30%, respectively).

Instrumentation—Catheterizations of the relocated carotid artery (18-gauge, 5.1-cm Teflon catheter,[d] group 1) or transverse facial artery (20-gauge, 3.2-cm catheter,[d] group 2) and pulmonary artery were performed to facilitate blood sampling during exercise. For the latter, a 110-cm Swan-Ganz catheter[e] was advanced to the pulmonary artery via the right jugular vein. Correct position of the catheter tip was verified by reading pressure waves displayed on an oscilloscope. A digital-display cardiotachometer[f] was placed around the girth of each pony for recording HR. Instrumentation was performed 1 to 2 hours prior to the exercise test.

Collection and analysis of expired gas—A loosely fitting face mask was used for all groups of horses.[8,9] An open-circuit gas collection system was used, with air drawn through the mask and around the muzzle into a 20-m long, 15-cm (inside diameter) flexible hose that led to a motor-driven blower. Flow rate through the system for group 1 was approximately 1,500 L/min for pre-exercise measurements and approximately 3,000 L/min for exercise measurements. Flow rate prior to exercise for groups 2 and 3 was approximately 3,000 L/min, whereas during exercise it was approximately 6,000 L/min. Flow rates for the ponies had been previously determined by comparing arterial blood gases sampled during rest and exercise periods, during which the ponies did not wear a mask, with those measured at various open-circuit flow rates while the ponies wore the mask. Flow rate was measured with a hot film anemometer[g] that was calibrated weekly by use of the nitrogen dilution technique[10] and yearly in a wind tunnel (traceable to the National Institute for Standards and Technology). The O_2 and CO_2 analyzers were calibrated daily with certified gas mixtures that contained 18.5% O_2, and 0.1 and 3.0% CO_2. At rest and during the last 15 seconds of work at each intensity, samples of expired gas were obtained simultaneously with blood samples. Oxygen consumption and $\dot{V}CO_2$ were calculated and converted to standard temperature pressure dry units. The **respiratory exchange ratio (R)** was then determined.

Blood sampling and analysis—Arterial and mixed venous blood samples were collected anaerobically into 3-ml heparinized syringes. Samples were capped and stored in an ice bath and analyzed within 1 hour of collection. Arterial blood samples for determination of plasma lactate concentration were collected into tubes containing sodium fluoride, stored in an ice bath, and analyzed in duplicate

Table 1—Four-week training schedule and training velocities (mean ± SEM) for 2 groups of ponies and 1 group of Thoroughbreds to assess maximum oxygen consumption ($\dot{V}O_2$ max)

Week	Duration (min)	Intensity (% $\dot{V}O_2$max)	Group*	Mean velocity (m/s)
1	4	40	1	1.7 ± 0.0
			2	1.8 ± 0.1
			3	3.2 ± 0.4
	6	60	1	2.8 ± 0.2
			2	3.1 ± 0.4
			3	5.4 ± 0.4
2	4	40	1	1.8 ± 0.1
			2	1.7 ± 0.0
			3	4.0 ± 0.3
	5	60	1	3.1 ± 0.3
			2	3.0 ± 0.4
			3	5.9 ± 0.1
	1	80	1	4.8 ± 0.4
			2	5.3 ± 0.5
			3	7.9 ± 0.2
3	4	40	1	1.9 ± 0.1
			2	2.0 ± 0.2
			3	3.8 ± 0.4
	4	60	1	3.3 ± 0.3
			2	3.6 ± 0.6
			3	6.0 ± 0.2
	2	80	1	4.9 ± 0.4
			2	5.9 ± 0.5
			3	8.1 ± 0.3
4	4	40	1	1.7 ± 0.0
			2	1.8 ± 0.1
			3	4.1 ± 0.4
	4	60	1	3.2 ± 0.3
			2	3.0 ± 0.3
			3	6.2 ± 0.3
	1	80	1	5.0 ± 0.5
			2	5.9 ± 0.3
			3	8.2 ± 0.4
	1	100	1	7.0 ± 0.6
			2	6.8 ± 0.3
			3	10.1 ± 0.6

Group 1 = 5 small ponies. Group 2 = 4 mid-sized ponies. Group 3 = 6 Thoroughbreds.

Table 2—Mean (± SEM) metabolic and cardiovascular data for 2 groups of ponies and 1 group of Thoroughbreds during exercise at $\dot{V}O_2$max before and after 4 weeks of training. ($\dot{V}O_2$max = Maximum oxygen consumption; $\dot{V}CO_2$ = Carbon dioxide production)

Variable	Group*	Before training	After training†
$\dot{V}O_2$ max (ml/kg/min)	1	82.6 ± 2.9	92.3 ± 6.0‡
	2	97.4 ± 13.2	107.8 ± 12.8‡
	3	130.6 ± 10.4	142.9 ± 10.7‡
$\dot{V}CO_2$ (ml/kg/min)	1	94.7 ± 2.4	103.6 ± 7.1
	2	111.9 ± 12.2	135.7 ± 21.7
	3	147.3 ± 9.3	163.1 ± 14.2
Weight (kg)§	1	171.4 ± 6.5	172.2 ± 4.8
	2	318.8 ± 12.9	320.5 ± 14.3
	3	487.3 ± 11.7	465.0 ± 11.2‡
Treadmill speed (m/s)	1	6.9 ± 0.6	7.3 ± 0.4
	2	7.4 ± 0.8	7.7 ± 0.5
	3	10.0 ± 0.3	10.8 ± 0.6
Work rate	1	1,172.2 ± 137.1	1,226.3 ± 59.3
	2	2,287.9 ± 282.6	2,427.0 ± 190.0
	3	4,758.9 ± 183.3	4,912.2 ± 205.4
Respiratory exchange	1	1.15 ± 0.03	1.12 ± 0.02
ratio	2	1.16 ± 0.05	1.25 ± 0.12
	3	1.14 ± 0.06	1.14 ± 0.03
Heart rate (beats/min)	1	222.2 ± 3.3	223 ± 3.3
	2	211.3 ± 5.1	215.8 ± 8.1
	3	Not collected	Not collected
Cardiac output	1	457.1 ± 47.7	489.1 ± 42.3‡
(ml/kg/min)	2	481.9 ± 58.5	591.8 ± 46.6‡
	3	Not collected	Not collected
Stroke volume	1	2.1 ± 0.23	2.2 ± 0.20
(ml/kg)	2	2.4 ± 0.14	2.8 ± 0.09
	3	Not collected	Not collected

*Group 1 = 5 small ponies. Group 2 = 4 mid-sized ponies. Group 3 = 6 Thoroughbreds. †Four weeks of training for groups 1 and 2; 3 weeks of training for group 3. ‡Significantly ($P < 0.05$) different from value before training. §Significantly ($P < 0.05$) different response to training among groups.

with an autoanalyzer[h] within 2 hours of collection. **Arterial and mixed-venous oxygen (P_aO_2 and P_vO_2)** and **carbon dioxide tension (P_aCO_2 and P_vCO_2)**, pH, and **hemoglobin oxygen saturation (S_aO_2 and S_vO_2)** were determined by use of a portable blood gas analyzer[i] and corrected for body temperature. Temperature in the pulmonary arterial blood was recorded at the same time blood was collected. **Hemoglobin concentration ([Hb])** was determined with a co-oximeter.[j]

Determination of other variables—Arterial and mixed-venous oxygen content (C_aO_2 and C_vO_2) were calculated as:

$$C_aO_2 \text{ (or } C_vO_2) = (1.39 \times [Hb] \times S_aO_2 \text{ [or } S_vO_2]) + (0.003 \times P_aO_2 \text{ [or } P_vO_2])$$

where [Hb] = g/100 ml and S_aO_2 (or S_vO_2) = % Hb saturation of arterial or venous blood. Cardiac output and SV were calculated from the Fick equation.

Power output in watts was calculated as:

$$\text{Body weight (kg)} \times \text{treadmill speed (m/min)} \times 6.12^{-1} \times \sin \text{(of treadmill angle)}$$

The speed at which $\dot{V}O_2$max was reached was determined from the regression equation for $\dot{V}O_2$ and treadmill speed.

Statistical analyses—All measurements are expressed as mean ± SEM. Linear regression was used to evaluate the relationships between HR, SV, $\dot{Q}$, or C(a-v)O_2 and speed or $\dot{V}O_2$ during exercise (data was used from all stages of the exercise test up to and including those at $\dot{V}O_2$max). A paired *t*-test was used to determine whether there were significant changes in the slopes of these equations with training. A commercially available statistical analysis computer software program[k] was used to perform a 2-way repeated measures ANOVA, followed by the Bonferroni *t*-test to compare the response of mass-specific and absolute

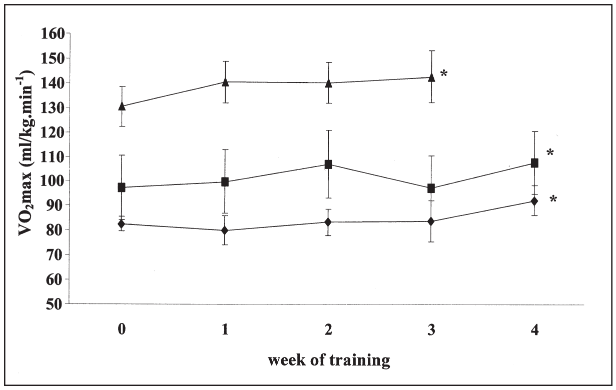

Figure 1—Mean (± SEM) weekly responses of maximal oxygen consumption ($\dot{V}O_2$) during 4 weeks of training in 2 groups of ponies and 3 weeks of training in a group of Thoroughbreds. ◆ = Group 1 (n = 5 ponies). ■ = Group 2 (n = 4 ponies). ▲ = Group 3 (n = 6 Thoroughbreds). * = Significantly ($P \leq 0.05$) different from value before training.

$\dot{V}O_2$max between and within the 3 groups before and after training, and to compare $\dot{Q}$, SV, HR, and C(a-v)O_2 at $\dot{V}O_2$max before and after training between and within the 2 groups of ponies. Values of $P \leq 0.05$ were considered significant.

Results

Training was not associated with any change in body weight in groups 1 or 2 (**Table 2**). However, body weight was significantly lower after training in group 3. Additionally, there was no effect of training on power output or on treadmill speed at $\dot{V}O_2$max for any group. Prior to training, mean mass-specific $\dot{V}O_2$max for groups 1, 2, and 3 was 82.6 ± 2.9, 97.4 ± 13.2, and 130.6 ± 10.4 ml/kg/min, respectively. After 4 weeks of training, mass-specific $\dot{V}O_2$max for groups 1 and 2 had increased by 11.7 ± 5.0% to 92.3 ± 6.0 ml/kg/min, and by 10.7 ± 6.8% to 107.8 ± 12.8 ml/kg/min, respectively. Because of a malfunction with the flow of the open-circuit expired gas collection system, accurate $\dot{V}O_2$max data was only obtained for 4 of 6 Thoroughbreds (group 3) at the end of the fourth week of training. Mean mass-specific $\dot{V}O_2$max for group 3 at that time was 131.5 ± 10.1 ml/kg/min. At the end of 3 weeks of training, mass-specific $\dot{V}O_2$max for all 6 Thoroughbreds was 142.9 ± 10.7 ml/kg/min. This represented an increase of 9.4 ± 5.8% above pretraining values. Thoroughbreds for which valid $\dot{V}O_2$max measurements were not obtained in the fourth week each had mass-specific values > 160 ml/kg/min after 3 weeks of training. Increases in mass-specific $\dot{V}O_2$max for each group were of similar magnitude and were significant ($P = 0.04$) in all 3 groups (**Fig 1**). However, changes in absolute $\dot{V}O_2$max were only significant in groups 1 and 2.

At $\dot{V}O_2$max, a significant ($P = 0.03$) training-related change in $\dot{Q}$ for the 2 groups of ponies was detected, with no difference between groups with respect to response to training. Significant changes in SV or C(a-v)O_2 in response to training for both groups of ponies was not detected; however, each group had similar responses to training.

Four weeks of training in groups 1 and 2 did not result in differences in rates of change (ie, slope) of

Table 3—Mean (± SEM) blood gas and acid-base concentrations for 2 groups of ponies during exercise at $\dot{V}O_2max$ before and after 4 weeks of training

Variable	Group*	Before training	After training
P_aO_2 (mm Hg)†	1	104.1 ± 1.2	92.4 ± 2.2‡
	2	89.2 ± 5.5	89.5 ± 4.8
P_vO_2 (mm Hg)	1	24.2 ± 3.3	22.8 ± 1.5
	2	24.3 ± 1.0	22.8 ± 5.2
P_aCO_2 (mm Hg)	1	30.2 ± 1.6	32.3 ± 1.6
	2	40.3 ± 1.1	39.8 ± 2.2
P_vCO_2 (mm Hg)	1	70.6 ± 2.7	79.7 ± 2.5
	2	93.8 ± 6.1	87.5 ± 14.6
$Hemoglobin_a$ (g/100 ml)	1	18.4 ± 0.6	17.7 ± 0.5
	2	18.2 ± 0.5	18.1 ± 0.6
$Hemoglobin_v$ (g/100 ml)	1	18.3 ± 0.4	17.5 ± 0.3
	2	19.0 ± 0.7	18.3 ± 2.1
Arterial oxyhemoglobin saturation (%)	1	96.8 ± 0.3	93.1 ± 1.2
	2	91.4 ± 1.3	91.7 ± 2.7
Mixed venous oxyhemoglobin saturation (%)	1	24.3 ± 5.1	16.5 ± 1.5
	2	18.6 ± 3.2	21.5 ± 4.4
Arterial oxygen content (ml/100 ml)†	1	25.1 ± 0.9	23.2 ± 0.8‡
	2	23.4 ± 0.6	23.3 ± 0.3
Mixed venous oxygen content (ml/100 ml)	1	6.34 ± 1.3	4.07 ± 0.4
	2	4.92 ± 0.8	5.21 ± 0.9
Arteriovenous oxygen difference (ml/100 ml)	1	18.8 ± 2.0	19.1 ± 1.0
	2	18.9 ± 1.0	18.1 ± 0.6
pH_a	1	7.39 ± 0.03	7.29 ± 0.05
	2	7.21 ± 0.03	7.31 ± 0.06
$[HCO_3^-]_a$ (mmol/L)	1	18.0 ± 2.1	15.5 ± 2.2
	2	19.5 ± 3.9	19.5 ± 2.6
$[HCO_3^-]_v$ (mmol/L)	1	24.2 ± 3.2	24.7 ± 2.5
	2	25.5 ± 2.7	25.9 ± 1.2
Lactate (mmol/L)	1	16.5 ± 2.5	20.3 ± 3.1
	2	18.3 ± 3.4	17.6 ± 5.2

*Group 1 = 5 small ponies. Group 2 = 4 mid-sized ponies. †Significantly ($P < 0.05$) different response to training between groups. ‡Significantly ($P < 0.05$) different from values before training. P_aO_2 = Arterial oxygen tension. P_vO_2 = Mixed venous oxygen tension. P_aCO_2 = Arterial carbon dioxide tension. P_vCO_2 = Mixed venous carbon dioxide tension. $[HCO_3^-]_a$ = Arterial bicarbonate concentration. $[HCO_3^-]_v$ = Mixed venous bicarbonate concentration.

HR, $\dot{Q}$, SV, or $C(a-v)O_2$ in relationship to treadmill speed, nor with respect to the slopes of the HR-, $\dot{Q}$-, SV-, or $C(a-v)O_2$ - $\dot{V}O_2$ curves. Training did not effect changes in $C(a-v)O_2$, S_aO_2, or arterial [Hb] at $\dot{V}O_2max$ in either group of ponies (**Table 3**). Although ponies in group 2 remained normoxemic at all exercise intensities, their P_aO_2 values were lower at $\dot{V}O_2max$ than ponies in group 1; ponies in group 2 also were isocapnic rather than hypocapnic at $\dot{V}O_2max$.

A significant effect of training on arterial blood pH, bicarbonate, P_vCO_2, or P_aCO_2 at $\dot{V}O_2max$ in either of the 2 groups of ponies was not detected (Table 2). A significant effect of training on HR or postexercise lactate concentrations for either of these 2 groups was not detected. There was also no effect of training on $\dot{V}CO_2$ or R during exercise at $\dot{V}O_2max$.

Discussion

To our knowledge, this is the first report documenting $\dot{V}O_2max$ in ponies of different sizes and evaluating the effect of training on $\dot{V}O_2max$. Recorded values for $\dot{V}O_2max$ were considerably below those reported for athletic breeds of horses.[6-8] This finding is compatible with the widespread impression that ponies are not as athletic in a comparative sense as athletic breeds of horses.

Reports of training responses in horses have revealed that $\dot{V}O_2max$ increases significantly in response to short periods of training.[3-5,c] It has been reported in several studies that horses increase $\dot{V}O_2max$ by > 20% after only 4 to 8 weeks of training,[1,3,5,b] whereas other studies have revealed that training for only 2 weeks increases $\dot{V}O_2max$ by 10 to 12%.[4,c] In our study, mean mass-specific $\dot{V}O_2max$ of the Thoroughbreds increased significantly (by 9%) in a short period, with the mean value after 3 weeks of training similar to that reported for other Thoroughbreds tested in our laboratory and elsewhere.[4,6-8] Similarly, mean mass-specific $\dot{V}O_2max$ in group 1 and group 2 ponies in our study also increased significantly by 12 and 13%, respectively.

One difference between the ponies and the Thoroughbreds was the significant reduction in body weight that was observed in the Thoroughbreds. The latter had been at pasture for at least 2 months before the start of the study and had gained weight during this time. In contrast, all ponies in each group had been purchased recently and were in good, but lean, condition. Consequently, it was not surprising the body weights of the ponies did not change.

As with athletic horses, $\dot{V}O_2max$ of human athletes can also increase significantly in response to short periods of training.[11-16] In humans, intensity and duration of training need to be considered to observe significant improvements in $\dot{V}O_2max$.[11,13-15,17,18] It has been observed that as long as the intensity of training is held constant at a minimum of 45% $\dot{V}O_2max$, $\dot{V}O_2max$ of previously sedentary people will improve in a short period.[14,15] However, $\dot{V}O_2max$ improves if the training intensities are greater than these submaximum work loads.[13,14,17,18] In several studies, it has been reported that cycle ergometer $\dot{V}O_2max$ measurements in humans increase linearly when training intensity is increased, and the relative magnitude of the adaptive response of $\dot{V}O_2max$ remains constant.[15,16] In contrast, several studies of the effect of training on Standardbreds and Thoroughbreds have revealed that horses have the ability to improve $\dot{V}O_2max$ substantially in a short period, but that increasing the intensity of exercise during training may not effect an improvement of $\dot{V}O_2max$, as long as exercise intensity is held constant at or above 40% $\dot{V}O_2max$.[1,3,4,19,c] In our study, the training intensities to which the ponies were subjected increased weekly, and the bouts were of short and constant duration (30 min/wk). Subsequent studies, therefore, are needed to determine whether the $\dot{V}O_2max$ of ponies would be different if training intensity or duration were altered.

None of the ponies had been trained or used as athletes prior to the start of the training program for this study. Comparatively, the Thoroughbreds used in our study and other horses used in the aforementioned studies had been trained and used on a regular basis for athletic events, and then detrained (ie, removed from training for an average of 4 months) prior to being examined.[3,4,b,c] Although it is possible that prior training may have affected the rate at which $\dot{V}O_2max$ changed with training, this was not substantiated in our study. The increase in mean mass-specific $\dot{V}O_2max$ in each group of ponies was slightly greater than the Thoroughbreds. When comparing the time course of adaptive response to training of sedentary and well-conditioned humans, 1 group of researchers found that there was no effect of prior training on rate or linearity of improvement of $\dot{V}O_2max$.[15] In contrast, another group found that increases in $\dot{V}O_2max$ after training were not as great in conditioned humans when compared with humans who were not conditioned.[20] Because of conflicting results found in the human literature, it is difficult to conclude that ponies (and possibly Thoroughbreds) are similar to humans with respect to prior training on $\dot{V}O_2max$.

The slightly lower increment in $\dot{V}O_2max$ observed in the Thoroughbreds could have been a reflection of the technical difficulties that led to the entire group only being evaluated over 3 weeks, rather than 4 weeks. Or, it may have been related to the previous training experience of the Thoroughbreds. All Thoroughbreds had been at pasture for 2 months, and some as long as 5 months, prior to the start of this study. Nevertheless, their initial fitness level may have been superior to that of the ponies, with the result that their $\dot{V}O_2max$ did not increase quite as much before it began to plateau.

Change in $\dot{V}O_2max$ for the ponies was caused primarily by an increase in $\dot{Q}$, with no change detected in $C(a\text{-}v)O_2$. This finding supports the contention that central factors (ie, increased SV) are primarily responsible for changes in $\dot{V}O_2max$ after training. This is consistent with findings of other studies of cardiovascular responses of mammals to training.[1,3,16,21,b] A 23% increase of $\dot{V}O_2max$ in a group of Thoroughbreds after 7 weeks of training has been reported; improvement in $\dot{V}O_2max$ was attributed to a 50% increase in SV.[3] Similarly, a 19% increase in $\dot{V}O_2max$ following 16 weeks of training, which was also attributed to a substantial increase in SV,[b] has been reported. Increased SV can result from a combination of factors, such as increased heart size, increased myocardial contractility, and increased preload caused by increased total blood volume, which augments ventricular filling.[16,22,23]

In contrast, a significant increase of $C(a\text{-}v)O_2$, with little to no change in $\dot{Q}$ in a group of Thoroughbreds after 2 weeks of training, has been reported.[4] A similar peripheral response was not evident in our study. At the end of the 4-week training period, there was no change in $C(a\text{-}v)O_2$ in the 2 groups of ponies, which indicated that increased extraction of oxygen by skeletal muscle did not make a substantial contribution to the improvement in $\dot{V}O_2max$. In humans, more efficient use of oxygen, as demonstrated by a decrease in C_vO_2 with no change in C_aO_2, may account for at least half of the improvement of $\dot{V}O_2max$ that was observed in young men as a result of 2 months of training.[16] The authors of that report concluded that the response in $\dot{V}O_2max$ was facilitated by increased capillary density in skeletal muscles, presumably resulting in decreased oxygen diffusion distances.[16] In contrast, horses that were trained for 7 weeks had no significant increase in capillary density in skeletal muscle.[24,25] However, Standardbreds that were trained for 6 months did develop increased skeletal muscle capillary density.[26] Therefore, it is possible that the duration of our training program was insufficient to allow for an adequate increase in capillary density in skeletal muscle; consequently, $C(a\text{-}v)O_2$ and C_vO_2 did not change. Our findings and those of others[1,3,24,25] suggest that central cardiovascular responses develop much more rapidly in horses in response to training, and that peripheral adaptations take longer than 4 to 7 weeks to develop.

All ponies remained normoxemic, compared with the arterial hypoxemia that was observed in Thoroughbreds exercising at comparable relative exercise intensities.[9,27-33] At maximal exercise intensities, ponies in group 2 remained isocapnic, whereas ponies in group 1 became hypocapnic. This difference may be associated with the higher $\dot{V}CO_2$ and P_vCO_2 that were observed in group 2, before and after training. This would be compatible with findings in exercising Thoroughbreds, which demonstrate that $\dot{V}CO_2$ and P_aCO_2 are closely related, and that the higher the mass-specific $\dot{V}CO_2$, the higher the P_aCO_2.[34] However, differences in gas exchange during exercise do not imply differences in the effect of training on $\dot{V}O_2max$. In fact, it has been reported that training has no short-term effect on gas exchange in Thoroughbreds.[5] Results of the study reported here indicated that ponies have the same response.

We had 3 objectives, and 2 of them were satisfactorily attained. We established that the mass-specific $\dot{V}O_2max$ of ponies is usually lower than that recorded in athletic breeds of horses. However, $\dot{V}O_2max$ can increase substantially in response to 4 weeks of intense training. These increases are mediated by central cardiovascular adjustments that are manifest by increases in $\dot{Q}$. The third objective was to compare the effect of training on $\dot{V}O_2max$ of 2 groups of ponies and a group of Thoroughbreds that were trained under similar conditions. When training-related changes in mass-specific $\dot{V}O_2max$ were compared, it appeared that the responses of ponies and Thoroughbreds to training were essentially the same; however, this may not actually be true. Body weights of the Thoroughbreds decreased significantly, whereas those of the ponies did not. Alternatively, absolute $\dot{V}O_2max$ of the ponies increased by a greater degree than that of the Thoroughbreds. The Thoroughbreds had been trained and the ponies had not. What influence prior training had on the results could not be assessed. To definitively address the third objective of this study, training responses of athletically naïve Thoroughbreds and ponies need to be compared, as do those of previously trained Thoroughbreds and ponies. Because little is known regarding responses of ponies to training, additional work needs to be performed with larger sample sizes to further investigate this, particularly with respect to the impact of alterations in intensity and duration of the conditioning program. This work needs to be done before the validity of using ponies as models for exercising horses can be established.

[a]Sato, Equispeed Technologies, Kansas City, Mo.
[b]Christley RM. Arterial hypoxemia during exercise in the horse. MS thesis, University of Sydney, Australia, 1995.
[c]Eaton MD. The energetics of equine performance. PhD thesis, University of Sydney, Australia, 1995.
[d]Novalon, Becton-Dickinson Co, Sandy, Utah.
[e]Model 93A1317F, American Edwards Laboratories, Santa Ana, Calif.
[f]Hippocard PEH200, Versailles, Ky.
[g]Series 2210, TSI Inc, St Paul, Minn.
[h]YSI 2300, Yellow Springs Instruments, Yellow Springs, Ohio.
[i]IRMA, Diametrics Medical Inc, St Paul, Minn.
[j]IL 282, Instrumentation Laboratory, Lexington, Mass.
[k]Number Cruncher Statistical Systems, Kaysville, Utah.

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