

# Effects of a Cold Environment on Exercise Tolerance in the Horse

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## Summary

*This study examined the physiological response of horses to exercise at a subzero temperature. Five Standardbred horses were subjected to a standardized incremental exercise test on a treadmill housed in a climate chamber. After training on the treadmill in the chamber, the horses were tested first at an ambient temperature of 17°C and then again 5–6 days later at –25°C. Exercise in the cold reduced respiratory rate at rest during the early stages of exercise and during recovery and also resulted in a lower blood temperature following exercise. No changes were observed in heart rate, blood lactic acid, blood-gas tensions, gait or lung tissue morphology as a result of exercise at –25°C. The horses showed no signs of discomfort during exercise in the cold. The major effect of exercise in cold environment is a lower respiratory rate possibly associated with a reduced need for heat dissipation.*

*Index terms:* Cold stress; blood gas tensions; heart rate; lactic acid; gait; lung morphology; respiratory frequency.

## Introduction

The racing season in Sweden extends throughout the year which means that ambient temperatures can vary between 30°C and –25°C. There are no reports on effects of cold stress in performing horses. The purpose of this study was to examine the physiological response of horses to exercise in a cold-air environment and to evaluate possible injury to the respiratory tract.

## Materials and Methods

*Horses.* Five Standardbred horses, one mare and four geldings, from 7 to 12 (mean 9.8) years of age, weighing from 415–490 (mean 461) kg, with their right carotid artery relocated to a subcutaneous position, were studied. All horses had been retired from racing and had been used for exercise studies during the previous 4 years. Each horse was carefully examined before and after each work test, with special examination of the respiratory tract by endoscopy. During the test period the horses were housed in an ambient temperature of 18°C.

*Exercise testing.* The horses were exercised on a treadmill placed in a climate chamber. The first exercise test was conducted at an ambient temperature of 17°C, which represents an average summer temperature and the second five to six days later at -25°C, which represents an extreme winter temperature. A compressor and three fans located two meters in front of the treadmill were used to produce an airstream (6 m/sec) with a preselected temperature ( $\pm 0.6^\circ\text{C}$ ). After accommodating to the treadmill, each horse walked for 5 minutes and then trotted for 2 minutes at each treadmill velocity, 5, 6, 7, 8 and 9 m/sec at a treadmill slope of 6.25%. Heart rate (HR) was continuously monitored with a bipolar electrocardiogram lead (Siemens Elema Mingograph 804) and recorded during the last 15 seconds at each speed and 2 and 5 minutes after exercise. The mean stride frequency (SF) was determined by timing 20 steps of one forelimb at each speed and the mean stride length (SL) was calculated from the treadmill velocity and SF. Respiratory rate (RR) was recorded at the end of each 2-minute speed interval and at 2 and 5 minutes after exercise.

*Blood samples.* Arterial blood samples were collected from a cannula in the relocated carotid artery, immediately before exercise, during the last 30 seconds at each speed and 2 and 5 minutes after exercise. Arterial blood was collected anaerobically into 20 ml heparinized glass syringes which were capped and stored in ice until analyzed within 1 hour for blood gas tensions ( $\text{PaO}_2$ ,  $\text{PaCO}_2$ ; ABL 3, Radiometer, Copenhagen, Denmark).

Blood lactate was determined enzymatically (Boehringer Test Combination No 124842 Mannheim, West Germany) on 0.1 ml aliquots of arterial blood, collected before, during exercise and 2 and 5 minutes after exercise. The blood was deproteinated with 0°C perchloric acid (1 ml) and then stored at 4°C for later analysis, usually within a one-week period.

*Temperature.* Blood temperatures were recorded at rest, at each exercise speed and 2 and 5 minutes after exercise with a thermistor (Swan-Ganz Standard Thermo Dilution Catheter, Edwards Lab. Santa Ana, California, USA) and a digital thermometer (EDT D515, Dale Instruments Ltd., Cambridge). The thermodilution catheter was introduced into the horse's left jugular vein using an 8F percutaneous sheath introducer set (Arrow International Inc., Pennsylvania, USA) and by monitoring the pressure curve, the catheter tip was located in the pulmonary artery. Muscle temperature was recorded with a thermistor probe inserted 3–4 cm into the right medial gluteal muscle before and immediately after each exercise test. Rectal temperatures were recorded before and directly after each exercise test.

*Lung biopsy technique.* Biopsies were obtained one week before the exercise test and within one hour after the exercise test in the cold environment. Lung biopsies were sampled using the technique described by Raphael and Gunson (1981) but with the following modification: a device was designed in which a specially resharpened 14G cutting biopsy needle (Tru Cut, Travenol Laboratories Inc. Deerfield, IL) could be mounted so that by pulling a trigger a compression coil instantaneously shot the outer cannula over the specimen rod. Thus a tissue specimen with a diameter of 1.4 mm and a mean length of 10 mm was obtained (Fig. 1). The biopsy site was in the 10th right intercostal space, approximately 15 cm above the ventral lung border. The biopsy needle mounted in the biopsy instrument was advanced approximately 3 cm into the lung parenchyma. The trigger was pulled and the biopsy set was withdrawn. The procedure was repeated

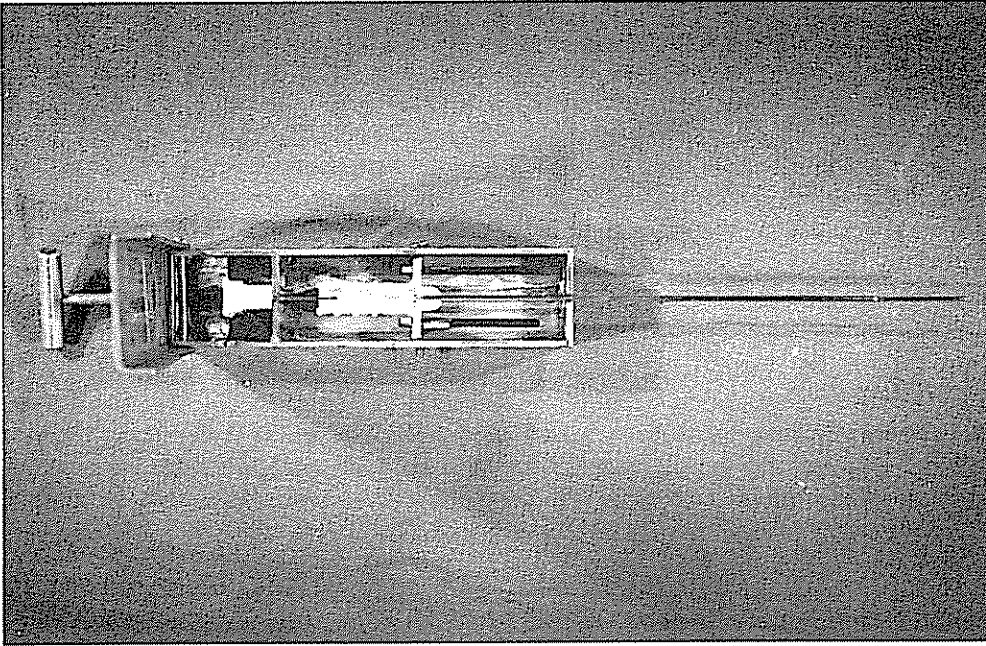


FIGURE 1. Biopsy needle mounted in instrument.

in order to obtain two biopsies from each horse. The biopsy specimens were fixed in 10% neutral buffered formalin and in Bouin's solution and then embedded in paraffin and stained with hematoxylin and eosin for microscopic examination.

*Data analysis.* The treadmill velocity which produced a HR of 200 bpm ( $V_{200}$ , m/sec), was interpolated from the linear regression of HR on treadmill velocity ( $V$ ), and the velocity producing a blood lactate concentration of  $4 \text{ mmol} \cdot \text{l}^{-1}$  ( $V_{LA4}$ , m/sec), was interpolated from the exponential regression of lactate on velocity (Persson, 1983).

*Statistical methods.* Data was analyzed with paired t-tests and by regression analysis (SAS Institute, 1985). The results are presented as means  $\pm$  standard deviations (SD).

### Results

The physiological responses to exercise in a cold air environment are shown in Table 1. No differences in the treadmill velocity at heart rate of 200 bpm ( $V_{200}$ , m/sec), the stride length at 9 m/sec (SL9) or the stride frequency at 9m/sec (SF9) were found.

The treadmill velocity associated with a lactate accumulation of  $4 \text{ mmol/l}$  ( $V_{LA4}$ , m/sec) was not altered. There were no significant differences in blood lactate accumulated at 9 m/sec (LA9) or immediately after exercise (LAO'). There was no difference in HR-response to exercise between exercise in room temperature and cold environment. The respiratory rates at rest and during the early stages of exercise and 2 and 5 minutes after exercise were significantly lower at  $-25^{\circ}\text{C}$  than at  $17^{\circ}\text{C}$  (Fig. 2). Blood temper-

atures in the cold tended to be lower at higher speeds and were significantly lower ( $P < 0.05$ ) 2 and 5 minutes after exercise (Fig. 3).

Muscle and rectal temperatures did not significantly differ between exercise in the cold and warm environment (Table 1). No differences were observed in arterial oxygen tensions ( $\text{PaO}_2$ ) but the arterial carbon dioxide tensions ( $\text{PaCO}_2$ ) at 5 m/sec and 2 minutes after exercise in cold were elevated (Fig. 4). The horses showed no signs of discomfort during the exercise at  $-25^\circ\text{C}$ . Apart from a few streaks of blood and approximately 30 petechial hemorrhages in the lower part of the trachea in 2 horses at endoscopy, there were no clinical signs of disease. The petechiae disappeared within 2 days. No morphological changes were identified in lung tissue samples collected after exercise at  $-25^\circ\text{C}$ .

### Discussion

We found no indications of an increased energy metabolism, as indicated by the unchanged post-exercise muscle temperatures and the unchanged heart rates and blood lactate responses to exercise during the cold exposure. As the experiments were con-

TABLE 1 Treadmill velocity at the heart rate of 200 bpm ( $V_{200}$ ); stride length at 9 m/sec (SL9); stride frequency at 9 m/sec (SF9); velocity at a blood-lactate level of 4 mmol/l ( $V_{LA4}$ ); lactate accumulation at 9 m/sec (LA9); lactate accumulation immediately after exercise (LAO'); heart rate at 9 m/sec (HR9); heart rate 2 and 5 min after exercise (HR2', HR5'); respiratory rate immediately after, 2 min and 5 min after exercise (RRO', RR2', RR5'); muscle and rectal temperatures immediately after exercise (MTO', RTO') at  $17^\circ\text{C}$  and  $-25^\circ\text{C}$  environmental temperatures.

	17°C Mean $\pm$ SD	Significant difference paired t-test	-25° Mean $\pm$ SD
$V_{200}$	7.98 $\pm$ 0.35	n s	7.52 $\pm$ 0.91
SL9	4.75 $\pm$ 0.20	n s	4.69 $\pm$ 0.15
SF9	114.5 $\pm$ 4.7	n s	116.2 $\pm$ 3.9
$V_{LA4}$	8.24 $\pm$ 1.02	n s	7.43 $\pm$ 0.88
LA9	5.98 $\pm$ 2.61	n s	9.43 $\pm$ 5.38
LAO'	7.11 $\pm$ 3.51	n s	7.84 $\pm$ 4.24
HR9	214.0 $\pm$ 7.9	n s	211.0 $\pm$ 7.5
HR2'	105.0 $\pm$ 5.5	n s	105.2 $\pm$ 6.9
HR5'	94.0 $\pm$ 8.3	n s	95.2 $\pm$ 10.5
RRO'	95.2 $\pm$ 10.3	n s	93.6 $\pm$ 16.4
RR2'	121.6 $\pm$ 3.6	$P < 0.001$	77.6 $\pm$ 11.6
RR5'	132.8 $\pm$ 17.1	$P < 0.02$	58.4 $\pm$ 26.2
MTO'	41.3 $\pm$ 1.2	n s	40.1 $\pm$ 0.9
RTO'	40.0 $\pm$ 0.5	n s	39.2 $\pm$ 0.5

Mean  $\pm$  S D Non-significant differences are indicated by n s

## Respiratory rate

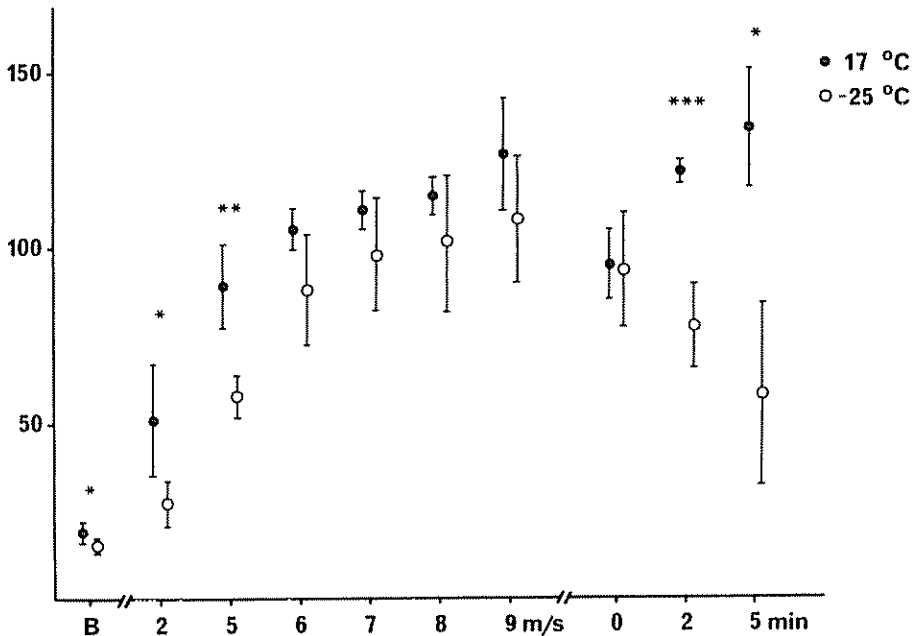


FIGURE 2. Respiratory rate (mean  $\pm$  SD) at rest before (B), during and after treadmill exercise in warm and cold environments. Treadmill speed and time after exercise ceased (min) are indicated on the x-axis. \* =  $P < 0.05$  \*\* =  $P < 0.01$  \*\*\* =  $P < 0.01$  (m/sec)

ducted during the later summer the animals were not acclimatized to the cold. All horses had short hair coats which might elevate the heat conductivity in the cold compared to a greater insulation of a winter hair coat. The large ventilation in the horse, minute volumes of 1200–1400 l/min at maximum exercise (Hörnigke *et al.*, 1983), implies that ventilation could be of importance for heat dissipation. The reduced respiratory rate and lower blood temperature in the cold seems to corroborate this assumption. The parameter  $V_{200}$  has been suggested to mirror the performance of the heart and the arterio-venous oxygen difference and to be closely associated with the oxygen uptake capacity (Persson, 1983). Thus, the unchanged value at exercise in the cold indicates that cold stress does not significantly influence the aerobic capacity in the horse. Likewise the fact that the  $V_{LA4}$ , which is considered to represent the anaerobic threshold in the horse (Persson, 1983), was unaffected by cold stress seems to substantiate this view.

The RR decreased immediately after exercise in both warm and cold environments. This indicates that the main determinant of RR during exercise is of central neurogenic or peripheral reflex origin initiated by muscle and joint receptors (Agostoni and D'Angelo, 1976; Iscoe and Polosa, 1976; Panda *et al.*, 1979). During recovery this locomotion induced stimulation of respiration appears to be replaced by a blood temperature dependent effect on respiration. Respiratory rate increased in the warm environment

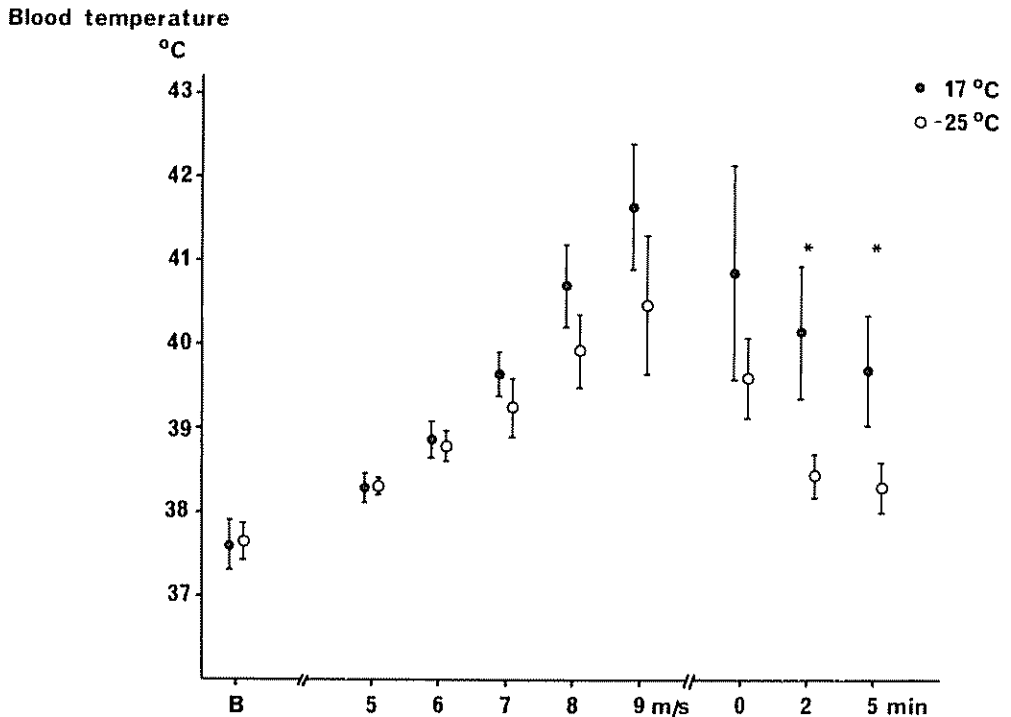


FIGURE 3. Blood temperatures (mean  $\pm$  SD) in the pulmonary artery at rest before (B), during and after treadmill exercise in warm and cold environments. Treadmill speed (m/sec) and time after exercise ceased are indicated on the x-axis.

whereas it decreased in the cold, concomitant with a similar recovery of HR and LA levels in both situations.

It is generally assumed that the rapid increase in  $\text{PaO}_2$  after exercise as well as the rapid decrease in  $\text{PaCO}_2$  is due to the high recovery RR induced by a post exercise metabolic acidosis (Thornton *et al.*, 1983). The fact that the post exercise  $\text{PaO}_2$  increased in both warm and cold environments suggests that hyperventilation prevailed relative to the oxygen consumption in both situations. The recovery  $\text{PaCO}_2$  being lower than the respective baseline values seems to corroborate this view. The difference in the hypocapnia at 2 and 5 minutes post exercise between warm and cold environments is probably a reflection of the difference in respiratory rates.

There was no indication that cold-induced bronchoconstriction occurred in these horses as there were no consistent differences either in  $\text{PaO}_2$  or in  $\text{PaCO}_2$  between cold and warm environments. Further no deleterious effect of the cold on measures of performance was detected. The petechiae found in the trachea of two of the horses after cold exposure, were similar to those observed in approximately 20% of Standardbred trotters after racing in different environmental temperatures (Forsberg and Dahl unpublished observations). Therefore it is quite possible that the presence of petechiae noted in this study is not causally related to the cold stress per se.

In conclusion, cold exposure ( $-25^\circ\text{C}$ ) did not seem to have any untoward effects on

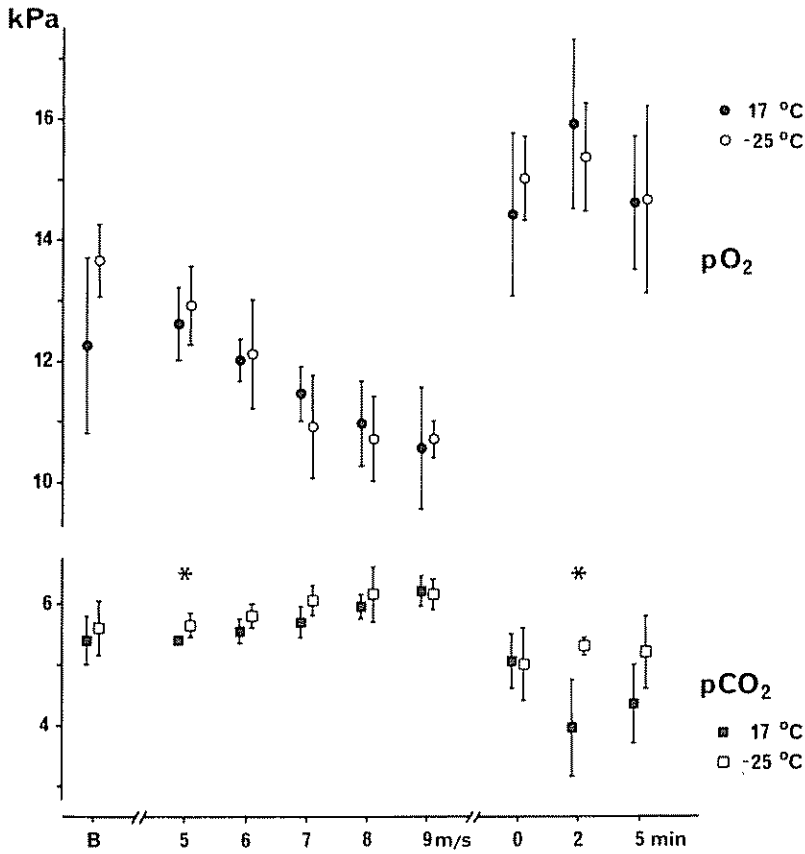


FIGURE 4 Mean ( $\pm$  SD) arterial oxygen tensions ( $\text{PaO}_2$ ) and arterial carbon dioxide ( $\text{PaCO}_2$ ) tensions at rest before (B), during and after treadmill exercise in warm and cold environments. Treadmill speed (m/sec) and time after exercise ceased are indicated on the x-axis. \* =  $P < 0.05$

near maximal exercise tolerance. On the contrary the cold environment seemed to be beneficial for heat dissipation during exercise. Further, no evidence of tissue damage in the respiratory tract was observed.

### Acknowledgments

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