High altitude acclimatization and athletic performance in horses

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Abstract

High altitude acclimatization produces a suite of physiological changes that might support an improved athletic performance at low altitude and thus lead to the strategy of athletic training at high altitude. Although there is substantial literature on high altitude physiology in humans, there are few studies on horses. Our interest in the physiological responses to high altitude in equids has been driven by the concerns of how athletic performance is altered at altitude and how conditioning at altitude may improve performance subsequently at low altitude. This review serves to illustrate what is currently known about the physiological changes of horses to altitude, at rest and during exercise, and to highlight how performance is impacted both at high altitude and subsequent return to low altitude.

Keywords: hypoxaemia; 2,3-diphosphoglycerate; pulmonary artery pressures; hypoxia; equine

Hypobaric hypoxia can produce a suite of physiological changes that would seem to support an improved athletic performance if these changes were to occur at low altitude, including increases in red cell number\(^1\), blood volume\(^2\), muscle capillarity\(^3\) and the metabolic capacity of muscles\(^4,5\). Consequently, the strategy of training at high altitude to enhance subsequent athletic performance at low-altitude competitions has long been popular. Despite the mass of literature on how humans adjust to high altitude, there is a paucity of data on how horses adapt. Our laboratory has asked the questions, ‘At altitude, what acclimatization occurs in horses and how rapidly do these changes occur?’

In humans, one of the most significant responses to hypobaric hypoxia is an increase in red cell number\(^6-8\). In earlier studies on horses, conclusions were contradictory. In one study, red cell number increased\(^9\) and another study found no changes\(^10\). The limitation in both studies was failure to control for splenic contraction (both were done in resting horses). In resting, non-stressed/non-exercised horses, the spleen may sequester as much as half the red blood cell population\(^11\). In response to sympathetic stimulus, the spleen contracts, dumping red cells into the general circulating vascular pool\(^11,12\).

In one study on five horses and one pony\(^13\), splenic contraction was induced by maximal exercise on a treadmill before and after nine days of acclimatization at 3800 m. Haematocrit increased after acclimatization. More importantly, total blood volume, a better measure of performance\(^11\), increased by 19%. Erythropoietin increased during the first day of exposure at 3800 m, but rapidly returned to low-altitude values and was not elevated even by exercise at high altitude\(^14\).

In humans, acclimatization to high altitude produces increases in 2,3-diphosphoglycerate (2,3-DPG)\(^15\), which has been argued to be an adaptive response to altitude\(^15,16\). 2,3-DPG is increased in horses with exposure to altitude\(^13\). An increase in 2,3-DPG shifts the oxygen dissociation curve to the right (decreases affinity), increasing the release of oxygen at any given oxygen partial pressure (PO\(_2\)) - a process that would facilitate oxygen unloading at the tissues. Even after return to low altitude, 2,3-DPG will remain elevated at least for a limited period of time\(^1\). In horses, 2,3-DPG was still increased 48 h after return to near sea level\(^13\). Sampling was terminated after 48 h, so we are unsure for how long this increase may be observed.

High-altitude acclimatization also produces metabolic changes in muscle. In horses, lactate dehydrogenase activity decreased 7% after nine days at 3800 m\(^17\) - a
finding consistent with that observed in humans. The enzymes whose activity is correlated with aerobic capacity, citrate synthase and β-hydroxyacyetyl-CoA dehydrogenase were unchanged. The decrease in an enzyme whose activity is correlated with anaerobic capacity, i.e. lactate dehydrogenase, in an environment that is oxygen-limited appears to be paradoxical. However, the decrease in anaerobic enzyme activity has been attributed to a tighter coupling between oxidative phosphorylation and glycolytic flux. The advantage would be less perturbation of adenylate ratios that could contribute to homeostasis of metabolites during exercise, quicker recovery and prevention of over-activation of glycolytic pathways. The down-regulation of lactate production has been described in high-altitude natives and in lowlanders after 6 weeks of acclimatization at high altitude (5400 m). Capillarity of muscle may also be increased with acclimatization, but has not been measured in horses.

How rapidly does this acclimatization occur? In an earlier study on horses (2200 m), the authors concluded that, based on red cell number, acclimatization was not complete until 35 days. However, this conclusion was based on changes in the packed cell volume in non-stressed horses. Unless the animal is appropriately stressed, say by exercise, the volume of red cells sequestered in the spleen is unknown. In the five horses transported to 3800 m, the most profound alterations in cardiovascular, haematological, respiratory and metabolic function occur within the first 24 h. By 72 h most variables have returned to low-altitude values or have stabilized. Because blood volume and muscle enzymes were not measured until after 9 days, the exact time course of these changes is unknown.

One concern of training at high altitude is that of oedema. Pulmonary artery pressures are elevated at high altitude and can precipitate high-altitude pulmonary oedema in humans. The vasoconstriction and associated fluid shifts may not be confined to the pulmonary circuit, as elevations in systemic filtration pressures can produce cerebral oedema and, in cattle, a peripheral oedema. In horses, pulmonary artery pressures in the non-exercised animal are elevated by 60% upon acute exposure (3800 m). By day 2, pressures had decreased but were still 34% higher, even on day seven. In ponies, pulmonary artery pressures are elevated and continue to rise during 6 weeks at altitude. Mean pulmonary artery pressures in the one pony used by Greene et al. were similar to those in ponies used in Bisgard et al.'s study. The increases in pulmonary pressures could produce pathological conditions such as seen in humans, but mean pulmonary pressures of normal horses at sea level can reach impressively high values during exercise (mean of 75 mmHg). Even when horses are exercised at high altitude, no overt clinical signs were evident. There is one report of ruptured pulmonary arteries in horses at 3700 m.

Another important limitation to training at high altitude is that while acclimatization may induce changes that support exercise, the decreased oxygen reduces not only the maximal oxygen consumption but also the absolute workload, so that training intensity invariably decreases. This is also observed in horses. When equids performed a standardized exercise test (SET) at 3800 m, their speeds (set by heart rate rather than workload) decreased by 15% in horses and 30% in the pony, compared with the SET with the same heart rates at 225 m. Interestingly, over the course of the 9-day acclimatization period, speeds continued to decrease with each subsequent SET. In a subsequent study where workload (rather than heart rate) was standardized during a trotting exercise test, heart rates in horses at 3800 m increased by 16% compared with those at 225 m (Wickler S, unpublished results, California State Polytechnic University, Pomona). These observations certainly support the notion that time at altitude can have deleterious effects on performance, but it also may involve the magnitude of the altitude exposure. In the aforementioned study, horses were at 3800 m - high by most equine event standards.

The recognition that training at high altitude by humans may, in fact, limit subsequent performance at low altitude has resulted in a shift in the training paradigm to a concept of ‘sleeping high, training low’. Individuals are exposed to a hypoxic environment for limited intervals of non-exercise periods, while training occurs in normoxia. The popularity of this approach has found its way into the equine world, not surprisingly, with commercial systems that are available for use in the horse (Biomedtech, Melbourne, Australia; Equine O₂, Victoria, Australia). A mask system delivers a low PO₂ stimulus (mimicking altitudes up to 6700 m) for limited periods with the intent of inducing physiological adjustments that will enhance performance. The recommendations for intermittent hypoxic exposure in the human literature vary, although many utilize sleeping (i.e. several hours) in a hypoxic environment. Recommendations for horses are proprietary; we were unable to find studies in the literature on horses.

Does high altitude acclimatization confer enhanced athletic performance on subsequent exercise bouts at low altitude? There is one study on horses that has addressed this question. A standardized treadmill exercise test to maximum speed was performed on five horses at 225 m before and within 24 h of return from 9 days at 3800 m. Recovery times for heart rate and blood lactate were decreased in acclimatized horses, suggesting a positive effect of altitude acclimatization,
High altitude acclimatization and athletic performance in horses and may be a manifestation of increased 2,3-DPG concentrations, increased blood volume and changes in the metabolic profile of the muscle. That said, care needs to be taken in the interpretation of these data. Firstly, those horses were not elite athletes. The changes in packed cell volume and total blood volume were within the range of normal horses. Secondly, the equine lung limits performance and may be a manifestation of increased 2,3-DPG concentration and changes in lung structure during short-term acclimatization would need to arise. Given that studies involving training horses demonstrate that the ventilatory capacity has only a limited ability to adapt, it is unlikely that changes in lung function would change with acclimatization. The limitations to diffusion can be offset, somewhat, by increases in haematocrit, but horses naturally possess the ability to increase haematocrit with exercise. There is no evidence that haematocrit can be increased ‘further’ with high altitude. So, in conclusion, while high-altitude exposure may improve subsequent performance at low altitude, its role in conferring an ‘additional’ advantage is still in question.

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References


