Training in Hypoxia
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Editor’s note: The primary discussion topic of the 1992 FISA Coaches Conference was altitude training. Several international experts spoke at the conference as well as a number of coaches who have many years of experience in training rowers at altitude. We present here papers by two of the featured speakers. Dr. Cerretelli of the University of Geneva is well known in rowing circles for his early work in the exercise physiology of rowing. His paper will provide a theoretical background. Dr. Uli Hartmann of the German Sport University in Cologne is well known for his years of experience, along with Dr. Alois Mader, working with the German National Rowing Team in St. Moritz. His paper presents practical recommendations for altitude training.

As is well known, exercise training as well as exposure to chronic hypoxia induce changes both in muscle mass and function. Besides, both conditions represent a stress for the respiratory and for the cardiovascular system. This is the reason why, recently, hypoxic training has become popular. The aim of such procedure is the coupling of exercise to hypoxia on the assumption that the physiological changes elicited separately by any of these conditions might become synergic or even potentiate each other. Hypoxic training is carried out at high altitude, typically between 2000 and 2500 meters above sea level or, alternatively, in normobaric hypoxia in a low pressure chamber.

What follows is a short account of the results obtained so far from controlled experiments performed in the above conditions.

Altitude training

Altitude training has become rather popular because of two reasons:

1) The frequent occurrence of athletic competitions at altitudes between 1500 and 3700 m. Thus, acclimatization and training protocols must necessarily be adopted to prevent symptoms of maladaptation and to give athletes the opportunity to test and practice the more favourable pace imposed by the reduced environmental P02;

2) The common observation that East African highlanders, normally training at altitudes >2000 m perform particularly successfully in endurance competitions held at sea level. The possibility has been suggested that prolonged training at

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higher elevations, besides raising the blood hemoglobin concentration, may confer special benefits because of a hypothesized altitudetraining synergistic effect. The latter would materialize in an enhanced muscle respiratory capacity, the underlying mechanism being a promotion of the oxidative machinery by the hypoxic stimulus.

Whereas the usefulness of training in hypoxia in view of performances at altitude cannot be questioned, the possible benefits from hypoxic training for competing at sea level have been, and still are, matters for debate. Several studies aimed at determining the evolution of VO2 max upon exposure to moderate hypoxia (usually corresponding to ±2500 m altitude) and sudden return to sea level have been carried out over the last 25 years (for details, see Bouissou et al., 1987; Levine et al., 1992). Because of the lack of standardization of the experimental protocols, the results appear to be rather controversial, the average improvement being small (±5%) and not statistically significant. This appears to be the case particularly for those subjects whose absolute baseline VO2 max values were on the high end of the scale (Bouissou et al., 1987) even though there is evidence of an extreme variability of the data even among highly trained subjects (Rahikila and Rusko, 1982).

A reason for the decrease of VO2 max, after altitude exposure, as found by some authors, could be the result of an overall detraining effect, the cause of which is the reduction of the absolute workload at which most subjects can train. Indeed, above 1500 - 1800 m, maximal exercise could enhance the normal tendency for athletes toward a decrease of the arterial blood O2 saturation which would necessarily limit the maximal aerobic performance. Upon return to sea level the athlete would have lost his habitual normoxic pace and therefore have undergone a limitation of his performance.

Physically active, though non athletic subjects, when brought to higher altitudes (±5000 m), after the initial loss of VO2 max, tend to slightly improve their maximal aerobic power. However, upon return to sea level, VO2 max never reaches prealtitude levels (Cerretelli, 1976; Cymerman et al., 1989; Grassi et al., personal communication, 1992) in spite of the increased blood [Hb] concentration. This is due mainly to detraining, even though, at these altitudes, hypoxiainduced muscle deterioration might also be involved (Martinelli et al., 1990).

Recently, Terrados et al. (1988) have studied in competitive cyclists the effects of training at sea level and at simulated altitude (corresponding to ±2300 m) on VO2 max and maximal work capacity measured at sea level and at altitude. These authors concluded that work capacity at altitude was increased more by training at altitude (33%) than at sea level (14%) whereas work capacity at sea level was equally improved (33 and 22%, N.S.) by the two training conditions. A decrease of muscle phosphofructokinase activity was found after simulated altitude training.

Mizuno et al. (1990) studied the effects of 2 weeks altitude (2100 - 2700 m) training in a group of well trained crosscountry skiers. No significant changes of VO2 max were found after hypoxic training, but short term running time was improved by 17%, probably as a result of increased glycolytic capacity. The gastrocnemius muscle maintained a prealtitude capillary supply with a 10% decreased mitochondrial enzyme activity, likely a consequence of detraining. The triceps brachii, by contrast, showed increased capillarization and unchanged mitochondrial enzyme activities as a result of a greater activation. Thus the hypothesis that altitude training could be beneficial from the standpoint of muscle respiration does not seem to be supported by the above experimental results.

The average very high performance capacity of East African endurance athletes is likely to be ascribed to higher specific VO2 max.

No published data are available, to the authors' knowledge, on the characteristics of muscle strength training in chronic hypoxia. This problem is particularly interesting in view of the possible implications from previous findings showing impaired muscle metabolism in chronic hypoxia. Preliminary results from a study in progress (Narici et al. personal communication, 1992) have indicated that, at 5050 m, a 30day strength training program of the biceps brachii muscle resulted, in a + 13.7% increase of the biceps cross sectional area, significantly less (p < 0.006) than at sea level (+ 19.8%). Thus muscle hypertrophy seems to be jeopardized by chronic hypoxia, possibly by a direct effect on protein synthesis.

In conclusion, endurance training at altitude appears to be beneficial, if not necessary, for performances to be carried out at altitude but does not seem to provide additional benefits compared to equivalent training at sea level for sea level competitions.

**Training in Normobaric Hypoxia**

Recent evidence from studies in which hypoxia was imposed only over fractions of the relatively short actual training time (in contrast to continuous hypoxia such as at high altitude), indicate that transient local lack of oxygen might in fact be a potent stimulus for the formation of muscle respiratory enzymes.
(Kayser et al., 1990; Terrados et al., 1990). In one study (Kayser et al., 1990) eight subjects performed one-legged bicycle exercise. Both legs were trained to the same workload but one leg was made partially ischemic during exercise by increasing the pressure around it to 50 mmHg. The leg trained in ischemic hypoxia improved its performance more than the leg trained under normal perfusion conditions. It was noted that there was a larger increase of citrate synthase activity in muscle biopsies obtained from the leg trained in ischemia than in the control limb.

Similar results are reported by Terrados et al. (1990). These authors had ten subjects carry out one legged bicycle exercise training in a large size pressure chamber. Again both legs were trained at the same workload, however one leg was trained under normobaric, the other leg under hypobaric conditions (2300 m). Time to fatigue increased significantly for both legs but more for the leg trained under hypobaric conditions. Concomitantly, there was a significantly larger increase in citrate synthase activity in the hypobarically trained leg. Moreover, myoglobin concentration decreased in the normobarically trained and increased in the hypobarically trained leg. It was concluded that “substrate flux, presumed to be similar in both conditions, is a less likely candidate as a stimulus for the enzyme synthesis. Instead, the stimulus seems to be related to the lowered blood oxygen content or tension” having a greater impact on the muscle metabolic machinery.

Preliminary results (Desplanches et al., 1992) confirm the finding of a greater increase in oxidative enzyme activities with training regimes using hypoxia as an additional stimulus during training. Five subjects were trained on a bicycle ergometer for three weeks (2h/day, 6 days/week) at a load corresponding to 80% of their peak heart rate measured in hypoxia breathing 10% O2 in N2, equivalent to an elevation of 5700 m altitude. After a 14 months detraining period, the subjects were retrained at the same absolute workload, but in normoxia. Training in normoxia had no effect on any of the investigated variables. Training in hypoxia increased hypoxic (but not normoxic) VO2 max, increased muscle cross-sectional area, interfibrillar mitochondrial volume density and the capillary fiber ratio by 10, 42 and 13%, respectively.

Studies in which intermittent hypoxia is induced during otherwise normoxic training sessions, open exciting new venues for gaining insight into the nature of the training stimulus. Apart from the fact that the energy balance of contracting muscles must be more profoundly altered with hypoxia as an added stimulus, other mechanisms governing muscle tissue malleability must also be considered including a number of “signal” peptides, oxygen regulated proteins and hypoxia associated proteins.

In conclusion, there are hints that exercise and hypoxia may represent additive stimuli to muscle adaptive changes during training. Their functional implications for the athlete are still matters for investigation.

References


