Zusammenfassung Millet, 2010 (10-10)

The ability to repeat sprints is further enhanced by intensive training in hypoxia than in normoxia.

Introduction

Specific altitude training modalities for intermittent sports (e.g. team and racquet sports) are still unknown. Vogt et al. have shown specific muscular adaptations induced by high intensity hypoxic training (Vogt et al., 2001) . However, it remains unclear whether and how training in hypoxia would enhance intermittent glycolytic performance and repeated sprint ability. In addition, hypoxia is known to raise oxidative stress and cumulate with the effect of exercise (increased reactive oxygen species). Therefore, antioxidant supplementation could be advantageous for performance, although it might blunt ventilatory adaptations. Our study evaluates the effects of repeated sprints training in hypoxia or in normoxia on specific RSA performance.

We hypothesize that specific high intensity training in hypoxia is more beneficial than the same training in normoxia due to molecular adaptations at the muscular level induced during hypoxic training.

Methods

50 trained subjects (35 ± 7 years, 75 ± 9 kg, 179 ± 5 cm) participated to the study and were assigned to 3 different groups (Control (C), training in normoxia (N), training in hypoxia (H)). The specific training consisted in 8 cycling repeated sprints sessions (3 x 5 sprints per session) during 4 weeks with 2 sessions/week. All training sessions were performed in a normobaric hypoxic chamber in a single blind fashion (Hypoxic groups at an altitude of 3000 m; F_IO_2=14.7% and normobaric groups at an altitude of 485 m; F_IO_2=20.9%).

PRE and POST the training period, subjects underwent several tests including: blood samples and muscle biopsies, 10 s isolated sprint, repeated sprint ability (RSA) test with 10 s sprints (1:2 sprint to rest ratio) until exhaustionand, 30s Wingate test and 3min all-out cycling aerobic test

Data were analyzed with 2-way ANOVA for repeated measures (condition (H vs N vs C) x time (pre vs post).

Results

Specific training increased significantly ($p<0.01$) the number of repeated sprints in H (9.4 ± 4.8 vs. 13 ± 6.2 sprints) but not in N (9.3 ± 4.2 vs. 8.9 ± 3.5) or in C (11.0 ± 7.1 vs 10.3 ± 6.2). 10s sprint and Wingate performance improved ($p<0.01$) similarly in H and N. 3 min all-out performance and post-RSA lactate were similar PRE and POST in all groups (Table 1).

Table 1 Performance results PRE and POST training in the Hypoxic (H), Normoxic (N) and control (C) groups

	Sprint 10s (W)		[La] post-RSA (mmol/l)		3 min all-out (W)		Wingate (W)	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
H	870 ±132	925±120*	15.0±2.3	15.4±2.1	368±45	383±39	699±102	718±94*
N	879 ±131	940±131*	14.2±1.7	14.8±1.6	371±49	382±47	688 ±75	723±86*
C	890 ±151	877±163	14.8±2.0	13.8±1.5	385±48	378±48	670 ±87	689±105

From the muscle biopsies at rest, we found a significant upregulation of the mRNA gene expression of hypoxia inducible factor (HIF-1a) (+55%), myoglobin (Mb) (+16%) and citrate anhydrase (CA3) (+35%).

Discussion

Specific repeated sprints training in hypoxia allows further enhancement of repeated sprint performance than the same training in normoxia. Systemic aerobic (3min all-out), glycolytic (Wingate, [La]) and alactic (isolated sprint) performances being similar H and N, this improvement in RSA can only be due to peripheral molecular adaptations at the muscular level induced by high intensity hypoxic training. The upregulation of genes involved in oxygen signaling (HIF-1a), oxygen carrying (Mb) and pH regulation (CA3) suggest an enhanced oxygen transportation and buffer capacity at the muscular level. The improvement of RSA performance observed in the hypoxic training group could therefore be explained by improved oxygen uptake kinetics at the muscular level.