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ORIGINAL ARTICLE

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Effects of training in normoxia and normobaric hypoxia on time to exhaustion at the maximum rate of oxygen uptake

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Abstract The effects of endurance training in normoxia or in hypoxia on time to exhaustion (T_{lim}) at the work rate corresponding to peak oxygen uptake ($\dot{V}O_{2peak}$) were examined at sea level in 13 healthy subjects. Before and after training the subjects performed the following: (1) incremental exercises up to exhaustion to determine peak oxygen uptake in normoxia ($\dot{V}O_{2peak}N$), the percentage of this value at the 4 mmol l^{-1} blood lactate concentration ($\dot{V}O_2 4\%N$) and the work rate corresponding to $\dot{V}O_{2peak}N$ ($Pa_{peak}N$), (2) a 5-min 90% $Pa_{peak}N$ exercise followed by a 10-min passive recovery to determine the maximal blood lactate concentration (La_{max}) measured during the recovery, and (3) a T_{lim} at $Pa_{peak}N$. Training consisted of pedalling 2 h a day, 6 days a week, for 4 weeks. Five subjects trained in normobaric hypoxia [HT; partial pressure of inhaled oxygen (P_IO_2) 89 mmHg] and eight subjects trained at the same relative work rates in normoxia (NT; P_IO_2 141 mmHg). The training-induced improvement of all the measured parameters were closely matched between the HT and the NT ($P > 0.05$). Training increased T_{lim} by 59.7% [164(40) s]. The value of T_{lim} was related to $\dot{V}O_2 4\%N$ and to La_{max} before and after training. Also, the training-induced improvement of T_{lim} was related to the concomitant decrease in La_{max}. It is concluded that: (1) endurance training including continuous high-intensity exercises improves T_{lim} for exercises performed at the same relative (higher absolute) work rate after training, (2) intermittent hypoxic training has no potentiating effect on T_{lim} as compared with training in normoxia, and (3) the intra-individual training-induced improvement of T_{lim} was associated with metabolic alteration in relation to lactate accumulation.

Keywords Humans · Lactate · Performance · Longitudinal study

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Introduction

Oxidative capacity, myoglobin and haemoglobin concentrations, haematocrit and capillary supply have been shown to be higher in animals and human natives living at altitude (Valdivia 1958; Reynafarje et al. 1959; Reynafarje 1962; Faura et al. 1969). The higher capacity of the blood to carry oxygen improves aerobic capacity (Ekblom et al. 1972; Warren and Cureton 1989) and may have a direct effect on sea-level performance. Therefore, attempts have been made to use altitude as an extra stimulus for improving aerobic capacity. However, prolonged exposure to altitude may also have negative effects. Previous studies described a loss of muscle mass and a decrease in mitochondrial density and oxidative capacity after extreme exposure to altitude (Hoppeler et al. 1990; Howald et al. 1990). To avoid these negative effects, training schedules in hypoxia have been designed with the primary goal being to enhance the training stimulus (Levine 2002) while the athletes live in normoxia the remainder of the day (living low–training high). Unfortunately, any booster effect of living low–training high on sea level performance or maximum rate of oxygen uptake ($\dot{V}O_{2max}$) is largely unproven, especially when training is performed at the same relative work rate in normoxia and hypoxia, i.e. at a lower absolute work rate in hypoxia (Terrados et al. 1988; Desplanches et al. 1993; Engfred et al. 1994; Böning 1997; Emonson et al. 1997; Vogt et al. 2001; Truijens et al.

2003). However, recent experiments underlined that intermittent hypoxic training could improve performance and the aerobic system to a substantial extent (Meeuwsen et al. 2001; Hendriksen and Meeuwsen 2003). Recent papers have emphasized that the duration of exposure to altitude or hypoxia may be a critical factor for the responses to exercise and for muscle adaptations (Levine 2002; Truijens et al. 2003). On this matter, it is worth mentioning that in the studies of Engfred et al. (1994), Emonson et al. (1997), Vogt et al. (2001) and Truijens et al. (2003), the subjects trained for only 20–45 min per day at 65–70% $\dot{V}O_{2max}$ three to five times a week for 5–6 weeks, while those of Meeuwsen et al. (2001) and Hendriksen and Meeuwsen (2003) trained 2 h a day at 60–70% $\dot{V}O_{2max}$ over a 10-day period. Therefore, whether the differences between the studies is related all or partly to the longer exposure to hypoxia during a single training session needs to be investigated.

During the last decade, the time to exhaustion (T_{lim}) at a work rate or velocity eliciting $\dot{V}O_{2max}$ has been a subject of interest. The reason is linked to the fact that T_{lim} appeared attractive and successful for assessing the performance capacity of athletes and for conducting their training schedule (Billat et al. 1998; Smith et al. 1999; Demarle et al. 2001). However, if Demarle et al. (2001) showed that T_{lim} is improved by training when an event is performed at the same absolute work rate before and after training, the results obtained when T_{lim} is performed at the same relative work rate are contradictory. While Billat et al. (1999) and Heubert et al. (2003) did not observe any improvement in T_{lim} after training at a velocity eliciting $\dot{V}O_{2max}$, Smith et al. (1999) and Demarle et al. (2003) did observe an improvement. In addition to this discrepancy, the training schedules performed in these previous experiments (Billat et al. 1999; Smith et al. 1999; Demarle et al. 2001, 2003; Heubert et al. 2003) included interval training sessions. It is now well-documented that endurance training programmes including moderate- or high-intensity continuous exercises shift the blood lactate curve to the right during an incremental exercise at absolute as well as relative work rates (Hurley et al. 1984; MacRae et al. 1992; Bergman et al. 1999). Since T_{lim} is associated with this shift (Billat et al. 1994, Demarle et al. 2003), endurance training including continuous exercise performed at moderate or high intensity would also be expected to improve T_{lim} for exercises performed at the same relative work before and after training. The mechanisms that contribute to delay lactate accumulation during graded exercise (Oyono-Enguéllé et al. 1990; MacRae et al. 1992) also contribute to its lower accumulation after a single bout of exercise (Jacobs et al. 1983; Bergman et al. 1999). Therefore, reliable results ought to be obtained using lactate kinetics after an acute submaximal exercise to evaluate endurance fitness for a specific form of exercise (Jacobs 1986). In the present study, the maximal blood lactate concentration measured during the recovery following a single exercise bout will be considered.

This study was designed, first, to compare the effects of training in normobaric hypoxia on T_{lim} with those obtained in normoxia and, second, to assess the effects of endurance training per se (including continuous high-intensity exercise) on T_{lim} performed at the same relative work rate before and after training, i.e. at an anticipated higher absolute work rate after training. Parts of the results have been published elsewhere for other purposes (Messonnier et al. 2002).

Methods

Subjects

Thirteen healthy subjects (three females and ten males) participated in the study. Before giving their written consent, they were informed of the nature, the potential risks involved and the benefits of the study. The experiments received the assent of the Ethics Committee on Human Research of Saint-Etienne. The experiments comply with the current laws of France.

Experimental design

All exercise tests were performed in the upright position on a cycle ergometer (Monark 818E, Stockholm, Sweden). The power output and the pedalling frequency (set at 75 rpm) were displayed on-line by a custom-made computer device. The experiments and training sessions were conducted either in normoxia [partial pressure of inhaled oxygen (P_IO_2) 141 mmHg] or in normobaric hypoxia (P_IO_2 89 mmHg). Hypoxia was obtained by reduction to 13.2% of the inspired oxygen fraction. This corresponds to a P_IO_2 equivalent to that at 3,800 m altitude.

Two weeks before the start of the experiments, all the subjects were submitted to an inclusion protocol. This consisted of a physical examination, anthropometric measurements (Table 1) and an incremental exercise up to exhaustion in hypoxia, which also allowed the subjects to become accustomed to the equipment and testing procedures. The experimental protocol consisted of pre-training, training and post-training periods.

Pre-training period

This involved four exercise. The first two sessions were incremental exercises up to exhaustion in normoxia (N)

Table 1 Mean values (SEM) of some anthropometric parameters. *NT* and *HT* Groups of subjects who trained in normoxia and normobaric hypoxia respectively, *WG* whole group of subjects

Subjects	Age (years)	Height (m)	Body mass (kg)
HT ($n=5$)	20.2 (0.4)	1.76 (0.06)	73.8 (11.2)
NT ($n=8$)	20.8 (0.7)	1.75 (0.04)	67.9 (4.0)
WG ($n=13$)	20.5 (0.5)	1.75 (0.03)	70.2 (4.8)

and hypoxia (H). Except for the work rate at the first step and the breathing gas composition, the subjects performed the same protocol. The test began with a 2-min rest period on the ergometer, followed by 2 min of warm-up cycling at 0 W. The exercise started at 60 W (N) or 40 W (H) for the males and 40 W (N) or 20 W (H) for the females. After 2 min of cycling at one of these loads, the work rate was increased every 2 min by 30 W for the males and 20 W for the females. This procedure was followed until exhaustion, which was defined as the point at which a subject could no longer maintain the requested pedalling frequency. Expired gas was analyzed continuously for oxygen uptake ($\dot{V}O_2$). Capillary blood samples were collected from the fingertip prior to the exercise and during the last 20 s of each exercise step for determination of the arterialized blood lactate concentration. These sessions were carried out to estimate the peak oxygen uptake ($\dot{V}O_{2peak}$) in normoxia and hypoxia ($\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$, respectively), the corresponding work rates ($Pa_{peak}N$ and $Pa_{peak}H$), the work rates corresponding to 60, 70, 80 and 90% of $Pa_{peak}N$ and $Pa_{peak}H$, the $\dot{V}O_2$ corresponding to the 4 mmol l^{-1} blood lactate concentration expressed as a percentage of $\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$ ($\dot{V}O_24\%N$ and $\dot{V}O_24\%H$) and the blood lactate concentration at $\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$ ($La_{peak}N$ and $La_{peak}H$).

The third session consisted of a constant-load exercise in normoxia. The subjects were tested in the morning about 0900 hours at ambient room temperature (20–23°C) after a light standard breakfast taken 1.5 h prior to the experiments. A hyperaemic ointment (Capsic) was applied to the earlobe 10 min before the start of the exercise to facilitate local blood flow and to improve arterialization of the blood. After a 3-min warm-up exercise (30–40% $Pa_{peak}N$), the subjects performed a 5-min 90% $Pa_{peak}N$ exercise. Blood samples were taken at the end of the exercise and during the recovery at the following times after the end of exercise: 0.5, 1, 1.5, 2, 2.5, 3, 3.5, 4, 4.5, 5, 6, 8, 10, 12, 15 and 20 min to determine the maximal blood lactate concentration (La_{max}).

The last session was a time to exhaustion event in normoxia at $Pa_{peak}N$. This session began by a warm-up (30–40% $Pa_{peak}N$ exercise for 3 min), which was immediately followed by a constant-load exercise at $Pa_{peak}N$ until T_{lim}. A blood lactate measurement (La_{Tlim}) was performed 3 min after the event's completion.

Training period

Training consisted of pedalling on the ergometer 2 h a day, 6 days a week, for 4 weeks. All the subjects trained at the same relative work rate but in two different oxygen partial pressures, this resulting in different absolute work rates. The subjects were randomly assigned to one of the two following groups. One group (HT) of five subjects (one female and four males) trained in normobaric hypoxia, while the other group (NT) of eight subjects (two females and six males) trained in normoxia. Initially, it was intended to compare seven subjects involved in HT with eight in NT but two of the HT subjects could not complete the study. The relative work rates were set successively at 60–70%, 70–80% and 80% of the pre-training $Pa_{peak}N$ and $Pa_{peak}H$ for the first, second and last 2 weeks, respectively (Table 2).

Post-training period

During the post-training period, the subjects were submitted to the same experimental sessions as during the pre-training protocol.

Measurements

Ventilation and $\dot{V}O_2$ were measured using a Med-Graphics CPX/D metabolic system (St. Paul, Minn.). An EKG (Siemens Mingograph 32) and arterial oximetry (Datex Satelite pulse oximeter) were used during the graded exercise tests for measurements of heart rate and arterial oxygen saturation (S_aO_2), respectively. Exercise

Table 2 Heart and work rates during 5-min 90% maximum work rate (*5-min 90% Pa_{peak}*) exercises and during the 4-week training period for the HT ($n=5$) and NT ($n=8$) groups. Values are means (SEM). *ND* Not done, *NS* not significant between HT and NT (Mann–Whitney *U* test)

	5-min 90% Pa_{peak} before training	Week 1	Week 2	Week 3	Week 4	5-min 90% Pa_{peak} after training
Heart rate (beats min^{-1})						
HT	ND	160 (5)	162 (5)	167 (4)	168 (4)	ND
NT	ND	165 (5)	166 (4)	167 (4)	168 (4)	ND
Significance	–	NS	NS	NS	NS	–
Work rate (W kg^{-1})						
HT	2.73 (0.23)	1.60 (0.13)	2.05 (0.14)	2.21 (0.12)	2.30 (0.15)	3.15 (0.27)***
NT	2.87 (0.11)	2.07 (0.07)	2.35 (0.08)	2.54 (0.09)	2.71 (0.12)	3.31 (0.14)***
Significance	NS	**	**	**	*	NS

*$P<0.06$ between HT and NT (Mann–Whitney *U* test)
**$P<0.05$ between before and after training for HT and NT (Wilcoxon signed-rank test)
***$P<0.05$ between before and after training for HT and NT (Wilcoxon signed-rank test)

power outputs and heart rates were determined by linear interpolation from the corresponding work rate and heart rate versus $\dot{V}O_2$ curves, respectively. Micropunctures for 20 µl of blood were taken. Lactate concentrations were determined enzymatically in haemolyzed blood with an LA 640 Kontron lactate analyzer (Roche Bio-electronics, Hoffman La Roche, Basel, Switzerland).

Statistical analysis

Descriptive statistics are expressed as means (SEM). Once the equality of the variances tested, the influence of the training mode (intergroup difference between the HT and the NT subjects) and the training-induced changes (before vs after training) were assessed by ANOVA (factorial analysis). Differences in pre- and post-training parameters and between the HT and NT were sought for by means of the Wilcoxon signed rank test and the Mann–Whitney U test, respectively. Differences were considered to be significant for $P \leq 0.05$ and to constitute a tendency when $0.05 < P \leq 0.10$.

Results

Pre-training

Before training, there were no significant differences between HT and NT for $\dot{V}O_{2peak}N$, $\dot{V}O_{2peak}H$, $Pa_{peak}N$, $Pa_{peak}H$, $\dot{V}O_24\%N$ and $\dot{V}O_24\%H$ (Table 3).

On average (pooled subjects), $\dot{V}O_{2peak}N$ was significantly higher than $\dot{V}O_{2peak}H$ [41.9 (1.4) vs 31.0 (1.9) ml $kg^{-1}min^{-1}$, respectively; $P<0.01$]. $La_{peak}N$ and $La_{peak}H$ were not different [11.4 (0.7) vs 11.6 (0.7) mmol l^{-1}, respectively]. In contrast, heart rates and S_aO_2 at $\dot{V}O_{2peak}N$ [194 (3) beats min^{-1} and 95.7 (0.5)%, respectively] were significantly higher than measured at $\dot{V}O_{2peak}H$ [190 (3) beats min^{-1}, $P<0.05$ and 80.3 (0.9)%, $P<0.01$, respectively]. $\dot{V}O_24\%N$ [66 (1)%] and $\dot{V}O_24\%H$ [70 (3)%] were not significantly different from each other.

Since $Pa_{peak}N$ of NT and HT were not different, absolute work rates for the 5 min 90% $Pa_{peak}N$ exercise and for the time to exhaustion event were similar in the two groups before training (Table 2). La_{max} values measured during the recovery following the 5 min 90% $Pa_{peak}N$ exercise were also similar for the NT and HT before training (Table 3).

The T_{lim} and La_{Tlim} values were not different between the groups before training (Table 3).

Training conditions

HT and NT performed the same training schedule in terms of relative work rate but, in terms of absolute value, HT trained at work rates lower by 15–29% than NT (Table 2). HT and NT displayed similar heart rates during the training sessions (Table 2).

Table 3 Data obtained before and after 4 weeks of endurance training for HT ($n=5$) and NT ($n=8$) groups and for WG ($n=13$). Values are means (SEM). $\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$ (ml kg^{-1} min^{-1}) Peak oxygen uptake rates in normoxia and hypoxia, respectively. $Pa_{peak}N$ and $Pa_{peak}H$ (W kg^{-1}) Work rates corresponding to $\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$, respectively. $\dot{V}O_24\%N$ and $\dot{V}O_24\%H$ (%) Oxygen uptake rates corresponding to the 4 mmol l^{-1} blood lactate concentration expressed as a percentage of $\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$. S_aO_2H and S_aO_2N Arterial oxygen saturations measured in hypoxia and normoxia, respectively. $La_{peak}N$ and $La_{peak}H$ (mmol l^{-1}) Blood lactate concentrations at $\dot{V}O_{2peak}N$ and $\dot{V}O_{2peak}H$. La_{max} (mmol l^{-1}) Maximal blood lactate concentration measured during the recovery following a 5-min 90% $Pa_{peak}N$ exercise. T_{lim} (s) Time to exhaustion at $Pa_{peak}N$. La_{Tlim} Blood lactate concentration measured 3 min after T_{lim}. *Asterisks* Statistical significance between the corresponding values obtained before and after 4 weeks of endurance training (Wilcoxon signed ranked test)

	Pre-training			Post-training		
	HT	NT	WG	HT	NT	WG
Incremental exercise in normoxia						
$\dot{V}O_{2peak}N$	39.6 (2.6)	43.3 (1.5)	41.9 (1.4)	44.5 (3.0)	46.9 (1.9)	45.9 (1.6)**
$Pa_{peak}N$	3.03 (0.26)	3.19 (0.13)	3.13 (0.12)	3.55 (0.30)	3.73 (0.16)	3.66 (0.14)**
S_aO_2N	95.2 (0.9)	96.0 (0.5)	95.7 (0.5)	96.0 (0.6)	95.5 (0.7)	95.4 (0.5)
$\dot{V}O_24\%N$	68.6 (2.5)	64.4 (1.7)	66.0 (1.5)	75.1 (2.4)	75.9 (2.8)	75.6 (1.9)**
$La_{peak}N$	10.7 (1.2)	11.8 (0.7)	11.4 (0.6)	10.9 (0.7)	11.9 (0.5)	11.5 (0.4)
Incremental exercise in hypoxia						
$\dot{V}O_{2peak}H$	28.4 (3.7)	32.7 (1.9)	31.0 (1.9)	35.5 (2.4)	37.9 (1.6)	37.0 (1.3)**
$Pa_{peak}H$	2.45 (0.27)	2.71 (0.12)	2.62 (0.12)	2.95 (0.29)	3.01 (0.11)	2.99 (0.12)**
S_aO_2H	81.2 (1.5)	79.8 (1.2)	80.3 (0.9)	78.6 (2.9)	78.5 (1.5)	78.5 (1.4)
$\dot{V}O_24\%H$	70.8 (6.9)	69.2 (3.2)	69.8 (3.2)	78.8 (1.7)	78.1 (2.1)	78.3 (1.4)*
$La_{peak}H$	11.4 (1.6)	11.7 (0.6)	11.6 (0.7)	10.6 (0.7)	10.6 (0.5)	10.6 (0.4)*
Constant load exercise (5-min 90% $Pa_{peak}N$)						
La_{max}	12.4 (1.6)	12.5 (0.8)	12.4 (0.7)	9.5 (1.1)	9.5 (0.8)	9.5 (0.7)**
Time-to-exhaustion						
T_{lim}	291 (21)	299 (23)	296 (16)	416 (26)	486 (62)	460 (39)**
La_{Tlim}	14.3 (1.2)	15.4 (0.6)	15.0 (0.6)	10.8 (1.9)	13.2 (1.0)	12.3 (1.0)**

*$P<0.05$
**$P<0.01$

Influence of the oxygen partial pressure during training on the measured parameters

Post-training $\dot{V}O_{2peak}N$, $\dot{V}O_{2peak}H$, $Pa_{peak}N$, $Pa_{peak}H$, $\dot{V}O_24\%N$ and $\dot{V}O_24\%H$ were not different between NT and HT (Table 3). Thus, HT and NT were matched as regards to the absolute work rates developed after training during the 5-min 90% $Pa_{peak}N$ and T_{lim} exercises (Tables 2 and 3). La_{max} following the 5-min 90% $Pa_{peak}N$ exercise were similar in HT and NT after training (Table 3). NT and HT also displayed similar T_{lim} and La_{Tlim} after training (Table 3).

Specific effects of endurance training

The specific effect of 4 weeks of endurance training was determined by comparison of the pooled pre- and post-training data. Body mass tended to be lower ($P<0.10$) after than before the training regimen [69.2 (4.7) vs 70.2 (4.8) kg, respectively]. $\dot{V}O_{2peak}N$, $\dot{V}O_{2peak}H$, $Pa_{peak}N$, $Pa_{peak}H$, $\dot{V}O_24\%N$ and $\dot{V}O_24\%H$ were improved by training while $La_{peak}N$ and $La_{peak}H$ remained unchanged (Table 3). La_{max} was significantly lower after training than before (Table 3). The T_{lim} value increased in response to training. This improvement in T_{lim} was accompanied by a significant decrease in La_{Tlim} (Table 3).

Relationships among variables

The T_{lim} values were related to the La_{max} values before (Fig. 1A) and after (Fig. 1B) training. The training-induced changes in T_{lim} were also related to those in La_{max} (Fig. 2). T_{lim} was related to $\dot{V}O_24\%N$ before ($r=0.62$, $P=0.0229$) and after ($r=0.57$, $P=0.0433$) training. However, there was no correlation between the training-induced changes in T_{lim} and those in $\dot{V}O_24\%N$ ($r=0.36$, $P=0.233$).

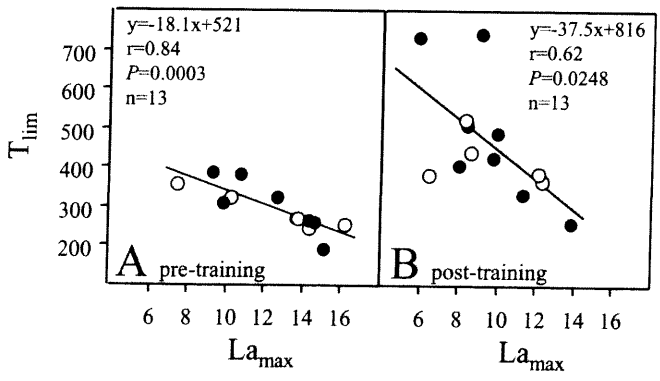

Fig. 1 Relationship between time to exhaustion and maximal blood lactate concentration before (**A**) and after (**B**) 4 weeks of endurance training. T_{lim} (s) Time-to-exhaustion at a work rate eliciting maximal oxygen uptake ($\dot{V}O_{2max}$). La_{max} (mmol l^{-1}) Maximal blood lactate concentration measured during the recovery following a 5-min exercise bout at a work rate eliciting 90% $\dot{V}O_{2max}$. Subjects trained in normoxia (●) and hypoxia (○)

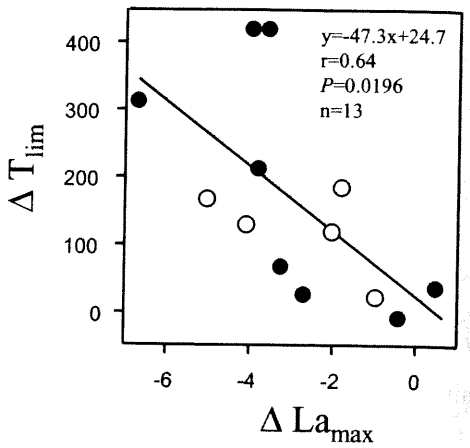

Fig. 2 Relationship between the training-induced changes in T_{lim} [ΔT_{lim} (s)] and those in La_{max} [ΔLa_{max} (mmol l^{-1})]. Subjects trained in normoxia (●) and hypoxia (○)

Discussion

Effect of oxygen partial pressure during training on sea-level T_{lim}

Most of the earlier studies provided no strong support for a potentiating effect of living low–training high for sea-level performance when training had been performed at the same relative work rate in normoxia and hypoxia (for details and references see Böning 1997; Levine 2002). In the present study, we hypothesized that this lack of benefit might be attributed to the short time of exposure to hypoxia during training. However, T_{lim} of NT and HT were matched before as well as after training. In other words, the two training regimens resulted in the same training-induced adaptations for T_{lim}. It should also be noted that none of the physiological parameters measured in the present study (Table 3) were improved to a greater extent after training in hypoxia. These results are similar to those of Emonson et al. (1997) and Truijens et al. (2003) who found that hypobaric exposure during training does not enhance $\dot{V}O_{2max}$, endurance time or performance more than training at sea level does. The lack of a potentiating effect of hypoxia is surprising since S_aO_2 measured during incremental exercise was significantly lower in hypoxia than in normoxia at all relative work rates, and also because evidence exists that local hypoxia is a strong candidate for regulating gene expression and stimulating muscle adaptation (Booth and Baldwin 1996; Hoppeler and Vogt 2001; Vogt et al. 2001). Vogt et al. (2001) showed clearly that intermittent hypoxic training results in an up-regulation of the regulatory subunit of hypoxia-inducible factor-1 involved in the expression of genes encoding among others for glycolytic enzymes and the vascular endothelial growth factor. Therefore, the similar training-induced adaptations of HT and NT might be related to the fact that both groups trained at the same relative work rates, i.e. at a

higher absolute work load for NT (Table 2). The higher mechanical stimulus and the higher rate of ATP turnover in NT may serve as an additional signal to up-regulate their muscle adaptations (Booth and Baldwin 1996). In accordance with Levine (2002), it is possible that the hypoxic up-regulation expected for the HT was similar to that linked to the higher absolute work load (metabolic flux) performed during training by NT. The similar effects in HT and NT might also be related to the fact that the stimulation of the β-adrenergic receptors involved in oxidative adaptations to endurance training (Booth and Baldwin 1996) was identical for HT and NT during training. A previous study (Kjaer et al. 1988) reported that epinephrine and norepinephrine responses to exercise were similar in untrained subjects whether the exercise was performed at the same relative work rate in hypoxia or in normoxia. Finally, if one considers heart rate as an indicator of the metabolic load for muscle tissues in exercise, it is interesting to note that HT and NT displayed the same heart rates during the training sessions (Table 2).

Nonetheless, Meeuwsen et al. (2001) and Hendriksen and Meeuwsen (2003) found a substantial effect of intermittent training in hypobaric hypoxia on performance and aerobic capacity. Their findings differ from those obtained in the present study and raise the question of whether hypobaric hypoxia and normobaric hypoxia correspond to the same stimulus. One cannot exclude that the specificity of the experimental design used by Meeuwsen et al. (2001) and Hendriksen and Meeuwsen (2003) may have been more favourable for showing the positive effects of training in hypoxia on performance and parameters of aerobic capacity.

Effect of training per se on T_{lim}

The present results show an improvement of T_{lim} by 68% after 4 weeks of endurance training (Table 3). Previous studies provided inconsistent results (Billat et al. 1999; Smith et al. 1999; Demarle et al. 2003; Heubert et al. 2003) on the effects of interval training on T_{lim} at the same relative work rate before and after training (i.e. higher absolute work rate after training). The present results argue in favour of a training-induced improvement of T_{lim} even when T_{lim} is performed at the same relative work rate after training. One original finding of the present study lies in the fact that the training-induced improvement of T_{lim} appears also after a training regimen including continuous high-intensity exercises.

The positive relationships between T_{lim} and $\dot{V}O_2$4%N obtained here before and after training are in accordance with those obtained previously between T_{lim} and lactate threshold (Billat et al. 1994; Hill and Rowell 1996; Demarle et al. 2003). However, and as already reported in these previous experiments, no more than 38% of the variability of T_{lim} was explained by $\dot{V}O_2$4%N. Of note are the higher correlation coefficients obtained when considering La_{max} measured during recovery following a 5-min 90% $Pa_{peak}N$ exercise instead of $\dot{V}O_2$4%N. Figure 1A and B shows the relationships between La_{max} and T_{lim} before and after training, respectively. These relationships show clearly that the subjects who accumulated lactate at a slower rate during the 5-min high-intensity exercise were also those who were able to exercise for a longer time before fatigue (T_{lim}). Numerous experiments have underlined the damaging effect of lactate accumulation and the associated pH decrease on muscle fatigue and performance (Hermansen and Osnes 1972; Hogan and Welch 1984; Fitts 1994); both reduce tension development (Hogan et al. 1995; Andrews et al. 1996) and disrupt excitation–contraction coupling (Metzger and Fitts 1987; Favero et al. 1997). Additionally, elevated proton concentrations reduce Ca^{2+} sensitivity (Westerblad et al. 1991) and interfere with energy supply (Sahlin 1992). Therefore, the slower rate of lactate accumulation would result in a better maintenance of homeostasis, which in turn would allow the subjects to perform longer exercise before fatigue.

The lower post-training La_{max} indicates that the rate of lactate accumulation during the 5-min high-intensity exercise is decreased in response to the training regimen (Table 3). More interesting is the observation that the training-induced changes in T_{lim} were also related to those in La_{max} (Fig. 2). A larger training-induced decrease in lactate accumulation led to a greater improvement of T_{lim}. This latter result is in agreement with the recent study of Demarle et al. (2003) who report that any training-induced increase in velocity at the lactate threshold was associated with an improvement in T_{lim} for submaximal exercise.

Conclusions

The endurance training programme performed in normobaric hypoxia did not add improvement to T_{lim} over that observed in normoxia. On the other hand, endurance training per se increased T_{lim} significantly. The training-induced improvement of T_{lim} was associated with a reduction in the La_{max} measured after an acute bout of 5-min 90% $Pa_{peak}N$ exercise. All these results argue in favour of a better maintenance of cellular homeostasis after training in relation to a lower blood lactate accumulation. These factors may contribute to delay muscle fatigue and thus to improving T_{lim}.

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