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Moderate exercise in hypoxia induces a greater arterial desaturation in trained than untrained men

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During moderate exercise breathing a low inspired O_2 fraction (F_IO_2), arterial O_2 desaturation may depend on the fitness level. Seven trained (TM) and seven untrained men (UTM) cycled in normoxia and in hypoxia (F_IO_2 = 0.187, 0.173, 0.154, 0.13 and 0.117). We compared TM and UTM at submaximal intensities below the ventilatory threshold. Ventilatory variables were monitored and arterial oxygen saturation was measured by pulse oximetry. O_2 saturation was not different between groups at sea level. In hypoxia, O_2 saturation was lower in TM than in UTM at F_IO_2 = 0.154 (87.3 ± 2.9% vs 90.4 ± 1.5% at 90 W) and below. Both the ventilatory-equivalent and the end-tidal O_2 pressure were lower in TM at sea level and at every F_IO_2, with the differences between TM and UTM becoming apparent at lower exercise intensity and increasing in magnitude as the severity of hypoxia increased. O_2 saturation was correlated with the ventilatory parameters at every F_IO_2 and the correlations were stronger in severe hypoxia. These results demonstrate that a moderate exercise carried out in hypoxia, contrary to normoxic conditions, can lead to a greater arterial desaturation in TM compared with UTM. This phenomenon could be partly attributed to a relative hypoventilation in trained subjects.

During maximal exercise in hypoxia, arterial desaturation is greater in trained compared with untrained subjects (Lawler et al., 1988; Powers et al., 1988; Martin & O'Kroy, 1993; Gore et al., 1996; Woorons et al., 2005), and only a few studies have focused on arterial desaturation at submaximal exercise (Dempsey & Wagner, 1999). At sea level, no difference was reported in arterial O_2 saturation (SaO_2) between trained subjects not showing severe hypoxemia and untrained ones (Harms et al., 1998). Similarly, no difference in SaO_2 was found between untrained and moderately or highly trained subjects during moderate exercise at sea level (Powers et al., 1988). The likelihood of developing greater arterial desaturation in trained men (TM) is weaker during a submaximal exercise. This is because diffusion limitation (Dempsey et al., 1984) and mechanical constraints of respiration (Johnson et al., 1992) occur at or near maximal exercise. As marked acidosis does not develop at moderate intensities, a decline in SaO_2 is induced by a low ventilatory response to exercise (Rice et al., 1999; Durand et al., 2000) and/or a worsened $\dot{V}A/\dot{Q}$ mismatch (Wagner et al., 1986). In hypoxia, more than in normoxia, these two factors may induce differences in SaO_2 between the two populations at moderate exercise. At sea level or at low altitudes, arterial oxygen pressure (PaO_2) generally remains above 60 mmHg. Owing to the flat shape of the oxyhemoglobin dissociation curve (ODC) at high PO_2 values, changes in PaO_2 above 60 mmHg may not have much effect on SaO_2. In this case, a greater arterial desaturation in trained compared with untrained subjects is unlikely to occur. On the other hand, a difference in SaO_2 between populations is more probable at altitudes inducing a PaO_2 lower than 60 mmHg. When located on the steep part of ODC, even a slight difference in PaO_2 caused by a relative hypoventilation and/or a $\dot{V}A/\dot{Q}$ mismatch could have significant consequences on SaO_2.

So far, no study has ever compared trained and untrained subjects at different degrees of hypoxia during submaximal exercise. Thus, the objective of this study was to determine whether, during a moderate exercise, a threshold altitude or exercise intensity induces a lower SaO_2 in TM compared with untrained men (UTM). As PaO_2 values below 60 mmHg are reached from altitudes of about 2500 m (Ferretti et al., 1997; Cardus et al., 1998; Anchisi et al., 2001) and considering the shape of the ODC, we postulate that this level could constitute the

threshold altitude from which trained subjects have a greater arterial desaturation than untrained ones.

Methods

Subjects

Fourteen men, sea-level natives and residents, volunteered for this study. They were all non-smokers and had no history of cardiovascular or respiratory disease. The subjects were divided into two groups: TM ($n = 7$) and UTM ($n = 7$). TM were triathletes who trained seven times a week (10 h) on average. UTM were sedentary or active in recreational sports, but who had never engaged in systematic endurance training. Written informed consent was obtained from each subject; all procedures were approved by the ethical committee of Necker Hospital, Paris, France. Subjects were asked to avoid strenuous physical activity 48 h before the tests. The physical characteristics of the subjects are shown in Table 1.

Experimental protocol

Each subject performed six maximal and incremental exercise tests (single blind) on an electrically braked cycle ergometer (Jaeger ER 900; Jaeger, Wuerzburg, Germany) in three different sessions. Each session, composed by two tests, was separated by 7 days. The first test was always carried out in normoxia [inspired oxygen fraction (F_IO_2) = 0.209, P_IO_2 = 150 mmHg]. In the other tests, the subjects breathed gas mixtures with F_IO_2 of 0.187, 0.173, 0.154, 0.13 and 0.117 (P_IO_2 of 131.8, 123.9, 108.1, 94.3 and 81.5 mmHg, respectively) for simulated altitudes of 1000, 1500, 2500, 3500 and 4500 m, respectively, according to the International Civil Aviation Organization (1964). The F_IO_2 of each test in hypoxia was randomly assigned.

Table 1. Physical characteristics of the subjects

	TM ($n = 7$)	UTM ($n = 7$)
Age (years)	26.6 ± 6.8	28.3 ± 3.5
Height (cm)	177.0 ± 8.4	178.1 ± 6.6
Weight (kg)	72.2 ± 7.5	79.0 ± 9.3
$\dot{V}O_{2max}$ (mL/kg/min)	66.1 ± 2.7*	43.0 ± 5.4
SpO_2 at $\dot{V}O_{2max}$ (%)	93.6 ± 1.3*	95.1 ± 1.2

Values are mean ± SD.

*Significantly different from UTM ($P < 0.05$).

TM, trained men; UTM, untrained men; $\dot{V}O_{2max}$, maximal oxygen consumption; SpO_2; arterial oxygen saturation.

Before each test, the subjects breathed the desired gas mixture for 5 min under resting conditions, to allow physiological variables to stabilize before starting exercise. The exercise test began with a 3 min warmup at a power output (PO) of 60 W, followed by increments of 30 W every 2 min, until the subjects could no longer maintain a pedaling frequency of 70 rev/min. The subjects were verbally encouraged to continue exercise as long as possible.

The maximal and incremental exercise tests allowed us to identify the ventilatory threshold (VT) for each altitude *a posteriori*. This was done using the criteria of Wasserman et al. (1973): (1) non-linear increase in VE, (2) non-linear increase in VCO_2 and (3) an increase in end-tidal O_2 pressure ($PetO_2$) without a corresponding decrease in end-tidal carbon dioxide pressure ($PetCO_2$). We then compared both groups at exercise intensities below VT corresponding to workloads of 60, 90, 120 and 150 W at F_IO_2 = 0.187, 0.173, 0.154 and up to 120 W at F_IO_2 = 0.13 and 0.117. The values of PO and O_2 consumption ($\dot{V}O_2$) corresponding to VT as well as the maximal power output in each group and for each F_IO_2 are presented in Table 2.

Altitude was simulated using the AltiTrainer®200 (S.M. TEC, Geneva, Switzerland), which produces a normobaric hypoxic mixture (reduced oxygen fraction) by addition of nitrogen in ambient air with a short response time (between 15 and 50 s). The gas mixture passes through a 30 L buffer tank before being inhaled by the subjects. An O_2 probe (electrochemical O_2 probe MOX3, City Technology, Portsmouth, UK) continuously controls the O_2 partial pressure of the inhaled gas mixture. According to the manufacturer, the maximal difference between the PO_2 measured by the AltiTrainer®200 O_2 probe and the PO_2 calculated from the O_2 fraction measured by an external probe (Servomex 720A, Geneva, Switzerland) is less than 1 mmHg over the whole range of PO_2 (150–69 mmHg). This device is reliable for altitudes below 5500 m and ventilation less than 200 L/min.

Measurements

Gas exchange was recorded breath by breath at rest and during exercise by using an integrated computer system. We used a rigid mouthpiece connected to a "Y" system fixation with a two-way valve, which ensured anti-return (Jaeger, Germany). An inspiratory valve, connected to the AltiTrainer®200, allowed the subject to inhale the hypoxic mixture; expired gases were collected into a metabograph (Oxycon, Jeager, Germany) to measure expired minute volume of gas at body temperature and pressure saturated (VE_{BTPS}), $\dot{V}O_2$, $PetO_2$ and $PetCO_2$. An electrocardiogram was recorded continuously.

Transcutaneous arterial O_2 saturation (Sp^1O_2) was measured by an ear pulse oximeter (Ohmeda Biox 3740, Louisville,

Table 2. $\dot{V}O_2$ and PO corresponding to VT and PO_{max} values

	F_IO_2	0.209	0.187	0.173	0.154	0.13	0.117
$\dot{V}O_2$ at VT	TM	3.98 ± 0.83*	3.83 ± 0.85*	3.76 ± 0.66*	3.50 ± 0.49*	3.24 ± 0.49*	2.77 ± 0.17*
	UTM	2.71 ± 0.53	2.49 ± 0.50	2.56 ± 0.43	2.36 ± 0.41	2.12 ± 0.30	1.98 ± 0.31
PO at VT	TM	274.3 ± 65.8*	265.7 ± 53.2*	261.4 ± 54.0*	244.3 ± 43.9*	214.3 ± 43.9*	192.9 ± 38.2*
	UTM	180.0 ± 39.3	171.4 ± 34.8	168.4 ± 34.8	162.9 ± 27.1	141.4 ± 26.4	137.1 ± 21.9
PO_{max}	TM	338.6 ± 51.9*	330.0 ± 51.5*	319.3 ± 49.1*	304.3 ± 43.0*	267.9 ± 27.1*	240.7 ± 31.1*
	UTM	247.5 ± 25.6	237.5 ± 23.6	235.0 ± 25.5	225.0 ± 26.0	213.3 ± 21.9	195.8 ± 14.6

Values are mean ± SD.

*Significantly different from UTM ($P < 0.05$).

$\dot{V}O_2$, oxygen consumption; VT, ventilatory threshold; PO, power output; PO_{max}, maximal power output; TM, trained men; UTM, untrained men.

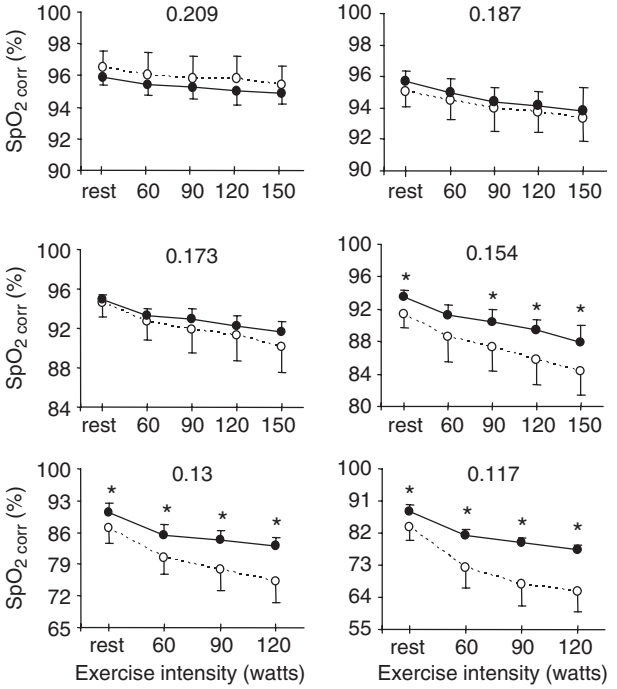

Fig. 1. Arterial O_2 saturation corrected (SpO_{2corr}) at rest and at submaximal exercise. ●, untrained men; ○, trained men; *significant difference between groups; bars indicate SD.

CO, USA). This device is not affected by subject motion (Barker, 2002) and its accuracy has been demonstrated (Trivedi et al., 1997). Pulse oximetry has been used in several previous experiments at low F_IO_2 levels (Martin & O'Kroy, 1993; Gavin et al., 1998; Peltonen et al., 1999). Nevertheless, at SaO_2 values below 75%, as it may be the case at $F_IO_2 = 0.13$ and 0.117 in the present study, the oximeter values may be less accurate (Trivedi et al., 1997). To obtain more reliable values of arterial O_2 saturation, Sp^1O_2 values at every F_IO_2 were corrected using a linear regression established from 142 simultaneous measurements of SaO_2 determined from capillary blood sampling. The accuracy of arterialized capillary blood sampling compared with arterial sampling is very much operator dependent. A technician is especially trained in our laboratory to perform the arterialized measurements that have already been used and compared with arterial values under pathological conditions (Lamberto et al., 2004).

The regression data used in the present experiments have already been used in a previous study carried out at altitudes up to 4500 m (Woorons et al., 2005) ($SpO_{2corr} = 0.86 \times Sp^1O_2 + 12.99$, $r = 0.98$, $P < 0.01$). Before attaching the ear clip, the earlobe was massaged vigorously and pre-warmed with a vasodilating capsaicin cream to increase perfusion. The breath-by-breath measurements and SpO_{2corr} were averaged over 15 s intervals. For calculation of $\dot{V}O_{2max}$, data were averaged over the two highest consecutive 30 s periods in which at least two of the three criteria were met: (1) a heart rate in excess of 90% of age-predicted maximum (220-age), (2) a respiratory exchange ratio of ≥ 1.10 and (3) a plateau in $\dot{V}O_2$ (≤ 150 mL increase over 2 min) with an increase in workload.

Statistical analysis

To determine the training effect (TM vs UTM) at each level of F_IO_2 and for each workload, we performed a two-way analysis of variance (ANOVA). When a significant main effect was found, a Tukey's *post hoc* test comparing the groups at rest and at each exercise intensity was carried out. Pearson's product–moment correlations with pooled population data allowed us to determine the relationships between SpO_{2corr} and ventilatory parameters at an exercise intensity of 120 W. This workload was used because it was the highest exercise intensity that was observed in all F_IO_2. Data are expressed as mean ± SD. A value of $P < 0.05$ was considered to indicate a statistically significant difference.

Results

SpO_{2corr}

At rest, SpO_{2corr} was lower in TM compared with UTM at $F_IO_2 = 0.154$, 0.13 and 0.117. During submaximal exercise, a significant difference between groups was observed at $F_IO_2 = 0.154$ at an intensity of 90 W. At $F_IO_2 = 0.13$ and 0.117, the differences were evident at the lowest exercise intensity (60 W) (Fig. 1).

At maximal exercise at sea level, SpO_{2corr} was significantly lower in TM compared with UTM (Table 1). None of the subjects had an SpO_{2corr} below 92% (range [92–95%] and [94–96%] in TM and UTM, respectively).

Ventilatory variables

At rest, $PetO_2$ was lower in TM compared with UTM at $F_IO_2 = 0.154$, 0.13 and 0.117. On the other hand, we did not find any difference between groups in $PetCO_2$ and $\dot{V}E/\dot{V}O_2$ (Table 3, Fig. 2).

During exercise, $PetO_2$ was lower in TM in normoxia (150 W) and at $F_IO_2 = 0.187$ and 0.173 (120 and 150 W) (Table 3). At lower F_IO_2, there was a difference in $PetO_2$ between groups from the lowest intensity (60 W). $\dot{V}E/\dot{V}O_2$ was also lower in TM than in UTM at 120 and 150 W at $F_IO_2 = 0.209$, 0.187 and 0.173, and at 60 W and higher at every other level of hypoxia (Fig. 2). Finally, we found a significant main group effect in $PetCO_2$ at $F_IO_2 = 0.13$ and 0.117, but the post-test failed to find any difference at any given workload (Table 3).

Oxygen consumption

There was no significant difference in $\dot{V}O_2$ between groups at every F_IO_2 and at each workload. For each group and at each workload, we did not find any difference in $\dot{V}O_2$ between altitudes. The mean $\dot{V}O_2$ values corresponding to workloads of 60, 90, 120 and 150 W were 1.35 ± 0.09, 1.66 ± 0.09, 1.99 ± 0.12 and 2.34 ± 0.10 L/min, respectively.

Correlations

At an exercise intensity of 120 W, SpO_{2corr} was correlated to $\dot{V}E/\dot{V}O_2$ and $PetO_2$ at $F_IO_2 = 0.187$,

Table 3. $PetO_2$ and $PetCO_2$ at a given workload and at every F_IO_2

	Rest		60 W		90 W		120 W		150 W		TR
	UTM	TM	UTM	TM	UTM	TM	UTM	TM	UTM	TM	
0.209											
$PetO_2$	107.2 ± 3.9	103.6 ± 5.9	100.3 ± 5.0	97.7 ± 6.1	99.2 ± 4.1	96.4 ± 5.5	101.7 ± 1.5	96.6 ± 6.1	103.2 ± 1.8	97.1 ± 5.0*	†
$PetCO_2$	38.3 ± 2.1	39.5 ± 4.4	43.8 ± 3.0	44.5 ± 3.7	46.5 ± 2.6	46.8 ± 3.3	47.2 ± 3.8	48.2 ± 4.2	47.2 ± 3.2	49.4 ± 4.4	NS
0.187											
$PetO_2$	94.5 ± 6.8	91.6 ± 6.3	84.3 ± 5.6	81.7 ± 3.9	84.0 ± 4.1	81.3 ± 4.7	86.0 ± 2.9	82.0 ± 3.2*	87.3 ± 3.4	82.6 ± 3.1*	†
$PetCO_2$	36.0 ± 6.4	37.7 ± 6.0	43.2 ± 6.0	44.3 ± 4.0	45.1 ± 5.6	45.8 ± 4.5	46.1 ± 4.8	46.7 ± 4.0	45.7 ± 4.9	47.7 ± 3.8	NS
0.173											
$PetO_2$	89.5 ± 5.6	84.0 ± 5.9	78.0 ± 2.3	74.0 ± 6.2	78.3 ± 2.9	73.6 ± 5.7	79.8 ± 2.9	74.6 ± 4.9*	81.7 ± 3.3	74.7 ± 4.6*	†
$PetCO_2$	35.1 ± 3.7	38.9 ± 4.9	43.1 ± 2.8	44.2 ± 3.7	44.7 ± 2.2	46.0 ± 3.9	45.4 ± 2.2	46.9 ± 4.0	45.4 ± 1.0	47.9 ± 4.1	NS
0.154											
$PetO_2$	74.7 ± 4.5	66.7 ± 4.8*	67.5 ± 2.7	60.7 ± 2.9*	67.3 ± 2.4	60.9 ± 2.6*	68.7 ± 2.6	61.4 ± 1.9*	70.2 ± 2.5	62.7 ± 2.2*	†
$PetCO_2$	35.1 ± 3.5	37.3 ± 4.9	39.9 ± 2.4	41.1 ± 4.1	41.2 ± 2.4	42.1 ± 3.5	41.6 ± 1.9	43.0 ± 3.6	41.6 ± 1.6	43.4 ± 3.2	NS
0.13											
$PetO_2$	64.2 ± 4.7	56.7 ± 4.9*	56.5 ± 3.6	50.5 ± 2.5*	57.4 ± 2.7	50.7 ± 2.4*	58.8 ± 1.9	51.5 ± 3.3*	–	–	†
$PetCO_2$	34.4 ± 2.8	39.2 ± 6.2	39.1 ± 2.6	41.7 ± 4.4	39.6 ± 2.2	42.7 ± 4.1	40.2 ± 1.5	42.9 ± 4.1	–	–	†
0.117											
$PetO_2$	55.8 ± 4.7	48.8 ± 6.6*	49.8 ± 3.4	44.0 ± 5.0*	50.2 ± 3.8	44.3 ± 3.2*	52.2 ± 2.9	45.5 ± 3.6*	–	–	†
$PetCO_2$	32.5 ± 3.1	36.6 ± 4.2	35.6 ± 3.2	38.1 ± 2.9	35.7 ± 3.6	38.4 ± 2.5	35.5 ± 3.1	37.8 ± 2.4	–	–	†

Values are mean ± SD.

*Significant difference from UTM ($P<0.05$).

†$P<0.05$.

UTM, untrained men; TM, trained men; $PetO_2$, end-tidal O_2 pressure (mmHg); $PetCO_2$, end-tidal carbon dioxide pressure (mmHg); TR, training effect; NS, not significant.

0.173, 0.154, 0.13 and 0.117. There was also a significant relationship between SpO_{2corr} and $PetCO_2$ at $F_IO_2 = 0.173$, 0.154, 0.13 and 0.117 (Table 4).

Discussion

The main finding of this study is that, during exercise below ventilatory threshold, TM show a greater arterial desaturation than UTM in hypoxia but not in normoxia. The differences were found from an F_IO_2 of 0.154, which corresponds to an altitude of 2500 m. The differences between TM and UTM increased in magnitude, and became evident at lower intensities as the severity of hypoxia increased.

While previous studies showed a greater arterial desaturation in trained subjects at maximal exercise, both at sea level and hypoxia (Lawler et al., 1988; Powers et al., 1988; Martin & O'Kroy, 1993; Gore et al., 1996; Woorons et al., 2005), this is the first study reporting such a phenomenon at submaximal workloads. Harms et al. (1998) found a lower SaO_2 at sea level in trained compared with untrained women at equal absolute intensities of exercise. However, all subjects showed severe hypoxemia at maximal exercise and were therefore not representative of a normal athlete population. On the other hand, the same authors found no difference in SaO_2 between trained women with mild exercise-induced hypoxemia and less fit women. Powers et al. (1988) also reported no difference in arterial O_2 saturation between trained and untrained male subjects at sea level.

The altitude at which we observed a difference in SaO_2 between groups could have possibly been lower. One explanation is that most of the subjects had only mild hypoxemia during maximal exercise in normoxia (SaO_2 of 93–95% according to Dempsey & Wagner (1999)), and athletes who develop marked hypoxemia during exercise at sea level show a greater arterial desaturation at altitude than athletes who do not (Chapman et al., 1999). The presence of severe hypoxemic athletes would have therefore probably accentuated the difference in SaO_2 between TM and UTM. Second, as we compared groups at a same absolute workload, the corresponding relative exercise intensity was therefore lower in trained than in untrained ones as the former achieved a higher maximal power output. Wagner et al. (1986) reported a worsened $\dot{V}A/\dot{Q}$ mismatch and therefore a wider alveolar-to-arterial O_2 pressure difference with exercise intensity. Thus, if we had compared TM and UTM at a same relative intensity, the difference in SaO_2 would have certainly been greater between populations at a given altitude. The threshold altitude and intensity from which we observed this higher desaturation in TM must be confirmed by further experiments.

The lower ventilation in TM than in UTM, both at sea level and in hypoxia, was probably due to reduced chemoresponsiveness. A weaker hypercapnic ventilatory responsiveness may reduce ventilation

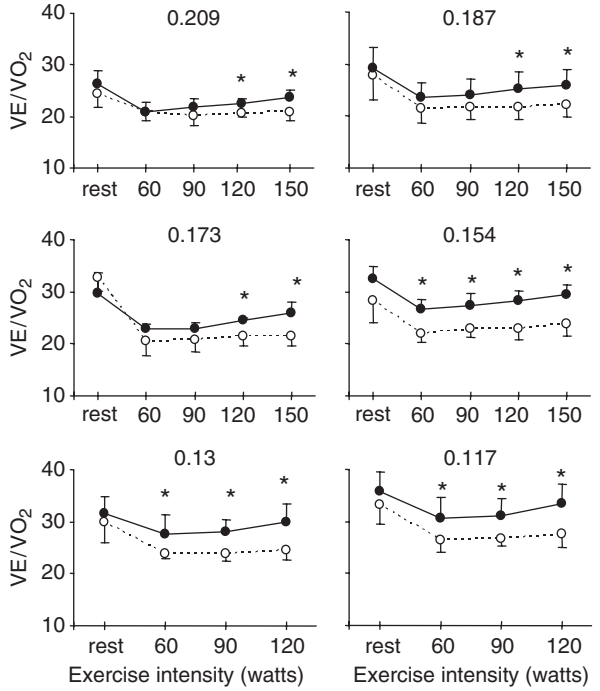

Fig. 2. $\dot{V}E/\dot{V}O_2$ at rest and at submaximal exercise. ●, untrained men; ○, trained men; *significant difference between groups; bars indicate SD.

Table 4. Correlation coefficients matrix between ventilatory parameters and SpO_{2corr} at an exercise intensity of 120 W for each F_IO_2

	SpO_{2corr}					
F_IO_2	0.209	0.187	0.173	0.154	0.13	0.117
$\dot{V}E/\dot{V}O_2$	0.32	0.55*	0.73*	0.66*	0.73*	0.65*
$PetO_2$	0.46	0.72*	0.80*	0.61*	0.86*	0.89*
$PetCO_2$	−0.42	−0.31	−0.79*	−0.62*	−0.69*	−0.62*

$n = 14$.

*$P < 0.05$.

$PetO_2$, end-tidal O_2 pressure (mmHg); $PetCO_2$, end-tidal carbon dioxide pressure (mmHg).

in TM but the present data indicate a blunted chemoreceptor response to hypoxia. Therefore, the level of ventilation in TM is influenced by the degree of hypoxia. Ventilatory data were correlated to SaO_2 at simulated altitude but not during exercise at sea level. While several studies reported that, during submaximal exercise in trained subjects, hypoxemia is associated with a relative hypoventilation, (Dempsey et al., 1984; Harms et al., 1998; Rice et al., 1999; Durand et al., 2000) the present study suggests that a worsened $\dot{V}A/\dot{Q}$ in particular may influence SaO_2.

Another observation is that the difference in SpO_{2corr} between TM and UTM was significant only from an F_IO_2 of 0.154. The threshold for the hypoxic ventilatory response is approximately 60 mmHg (Loeschcke & Gertz, 1958), which is reached during exercise at an altitude of about 2500 m (Ferretti et al., 1997; Cardus et al., 1998; Anchisi et al., 2001) corresponding to the mentioned F_IO_2. Consequently, as shown by our results, the difference in ventilatory parameters between groups may be accentuated, leading to a greater decrease in PaO_2 and therefore a greater arterial desaturation in trained than in untrained subjects. Moreover, according to the shape of the ODC, the consequences of a decline of PaO_2 on SaO_2 are more important when the former falls below 60 mmHg. Thus, at and above 2500 m, even a slightly lower PaO_2 in TM may have aggravated the difference in SaO_2 between groups.

The results of this study could have an implication on the physiological responses of athletes compared with untrained subjects during a moderate exercise at high altitude. Indeed, at sea level, it has been suggested that at any given submaximal work rate, a lower SaO_2 could be compensated by a higher O_2 extraction (Harms et al., 1998). In that case, a reduced ventilation could be more economical and therefore advantageous. On the other hand, at high altitude, venous oxygen content may not decrease sufficiently to compensate the decline in SaO_2. Thus, a lower ventilation could therefore be a disadvantage as a lower SaO_2 would require a higher cardiac work to maintain a given $\dot{V}O_2$.

Limitations

Pulse oximetry was used to measure SaO_2, and to increase its accuracy, a highly significant linear regression was established from a high number of capillary blood samples, to adjust the values obtained by oximetry (Woorons et al., 2005). To further limit the effects of motion, exercise was carried out on a cycle ergometer instead of a treadmill, although SaO_2 values are similar using both exercise modes (Laursen et al., 2005). Finally, the ear was prewarmed to avoid poor perfusion during exercise.

In summary, this study showed that endurance-trained subjects had a greater arterial desaturation compared with untrained individuals during moderate exercise in hypoxia. The difference appeared at 2500 m and was partly attributed to a relative hypoventilation. Other mechanisms involved in the greater arterial desaturation observed in trained compared with sedentary subjects during submaximal exercise should be considered in future studies.

Perspectives

The present study suggests that a lower hypoxic ventilatory response (HVR) in trained subjects

may have influenced their ventilatory response. At maximal exercise, the role of HVR could be opposed by the presence of an expiratory flow limitation (Derchak et al., 2000). On the other hand, a relationship between ventilation and HVR is probably stronger during moderate exercise, especially in hypoxia. Further research is needed to determine the role of HVR under these exercise conditions.

Key words: hypoxemia, SaO_2, altitude, athletes, ventilation.

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