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ORIGINAL ARTICLE

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Linear decrease in $\dot{V}O_{2max}$ and performance with increasing altitude in endurance athletes

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Abstract It has been hypothesized that one reason for decreased $\dot{V}O_{2\,max}$ in hypoxia could be the lower maximal exercise intensity achieved in incremental, time or distance trial tests. We hypothesized that (1) $\dot{V}O_{2\,max}$ would be decreased at altitude even when exercising at the same absolute maximal exercise intensity as at sea level and; (2) the decline in $\dot{V}O_{2\,max}$ in endurance-trained athletes (ETA) would be linear across the range from sea level through moderate altitudes. Eight ETA performed combined $\dot{V}O_{2\,max}$ and performance tests running to exhaustion at the same speed in a randomized double blind fashion at simulated altitudes of 300, 800, 1,300, 1,800, 2,300 and 2,800 m above sea level using a hypobaric chamber. Douglas bag system was used for respiratory measurements and pulse oximetry was used to estimate arterial O_2 saturation. $\dot{V}O_{2\,max}$ declined linearly from 66 ± 1.6 ml kg^{-1} min^{-1} at 300 m to 55 ± 1.6 ml kg^{-1} min^{-1} at 2,800 m corresponding to a 6.3% decrease per 1,000 m increasing altitude (range 4.6–7.5%). Time to exhaustion (performance) at a constant velocity associated with 107% of sea level $\dot{V}O_{2\,max}$ decreased with 14.5% ($P < 0.001$) per 1,000 m altitude between 300 and 2,800 m. Both $\dot{V}O_{2\,max}$ and performance decreased from 300 to 800 m ($P < 0.01$; $P < 0.05$). Arterial haemoglobin oxygen saturation at test cessation (SpO_{2min}) declined from 89.0 ± 2.9% at 300 m to 76.5 ± 4.0% at 2,800 m ($P = 0.001$). This study report that in ETA during acute exposure to altitude both performance and $\dot{V}O_{2\,max}$ decline from 300 to 800 m above sea level and continued to decrease linearly to 2,800 m.

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Introduction

In endurance sport, most training and competition take place at altitudes below 3,000 m. As altitude increases, aerobic performance and maximal oxygen uptake ($\dot{V}O_{2\,max}$) are reduced due to the reduced partial pressure of O_2 in the inspired air (Fulco et al. 1998). However, the decrease in $\dot{V}O_{2\,max}$ with increasing altitude has been shown to vary between individuals (Koistinen et al. 1995; Lawler et al. 1988). Although the reasons for this individual response are not clear, fitness level seems to be of importance as endurance-trained athletes (ETA; $\dot{V}O_{2\,max} > 60$ ml kg^{-1} min^{-1}) have demonstrated a larger decline in $\dot{V}O_{2\,max}$ with increasing altitude compared with untrained individuals (Gavin et al. 1998; Koistinen et al. 1995; Lawler et al. 1988). It has been suggested that this is due to the fact that ETA have developed exercise-induced desaturation already at sea level (Chapman et al. 1999; Gore et al. 1996; Terrados et al. 1985) and work at the steeper part of the oxygen equilibrium curve (Ferretti et al. 1997) at low altitudes. Furthermore, pre-exposure to altitude influences the degree of decline of $\dot{V}O_{2\,max}$ (Fulco et al. 1998). Theoretically, the altitude-induced decrease in $\dot{V}O_{2\,max}$ should be largest in sea level resident, not altitude-acclimatized athletes with a high $\dot{V}O_{2\,max}$. Several studies have shown a reduction of $\dot{V}O_{2\,max}$ in ETA with moderate hypoxia under laboratory conditions (Friedmann et al. 2004; Chapman et al. 1999; Gore et al. 1997, 1996; Peltonen et al. 1995, 2001; Lawler et al. 1988; Paterson et al. 1987; Squires and Buskirk 1982; Terrados et al. 1985). Three studies showed that $\dot{V}O_{2\,max}$ declines even at low altitudes around 750–900 m (Gore et al. 1996, 1997; Terrados et al. 1985), indicating that the decrease is linear from sea level to 3,000 m. However, none of these studies have tested $\dot{V}O_{2\,max}$ from sea level (0–300 m) to very low (300–1,000 m), low (1,000–2,000 m) and

moderate altitudes (2,000–3,000 m) in the same athletes. The $\dot{V}O_{2max}$ test used in prior studies was either an incremental protocol to exhaustion (Chapman et al. 1999; Gore et al. 1996, 1997; Lawler et al. 1988; Paterson et al. 1987; Peltonen et al. 1995, 2001; Squires and Buskirk 1982; Terrados et al. 1985) or an all out test for a given distance (Peltonen et al. 1995) or time (Gore et al. 1997). Under hypoxia, these protocols result in reduced absolute exercise intensity. It has therefore been hypothesized that one reason for the decreased $\dot{V}O_{2max}$ in hypoxia is the result of reduced maximal exercise intensity (Noakes et al. 2001; Peltonen et al. 2001). The aim of the present study, therefore, was to evaluate $\dot{V}O_{2max}$ and performance with a combined $\dot{V}O_{2max}$ and performance test, using the same absolute exercise intensity, from sea level to low and moderate altitudes in unacclimatized ETA. We hypothesized that both $\dot{V}O_{2max}$ and exercise performance would decrease at altitudes $<1{,}000$ m and continue to decrease linearly to low and moderate altitudes in ETA.

Methods

Subjects

Eight healthy endurance-trained sea level resident athletes (age 24.4 ± 3.5 years) participated in the study (Table 1). The study was approved by a Regional Norwegian Ethics Committee and has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. Subjects gave their written informed consent before entering the study.

Study design

An outline of the study design is shown in Fig. 1. Subjects performed nine tests (T1–T9) on a motor-driven treadmill in a hypobaric chamber over a period of 4 weeks with a minimum of 48 h between two tests. The first two exercise tests (T1 and T2) were pre-experimental tests and followed the test protocol A. These tests were used to ensure that the subjects met the inclusion criteria ($\dot{V}O_{2max} > 60$ ml kg^{-1} min^{-1}), to calculate the running speeds for the test protocol B and to familiarize themselves with the test environment and equipment. All the other exercise tests at the different altitudes (T3–T9) followed the test protocol B. In these tests, the subjects ran with the same absolute constant submaximal and the same absolute "maximal" running speed at the different altitudes.

The tests (T3–T9) at the different altitudes were performed in randomized order except that the first (T3) and the last test (T9) were at "sea level" (300 m, 977 hPa). The simulated altitudes were 800 m (921 hPa), 1,300 m (868 hPa), 1,800 m (818 hPa), 2,300 m (772 hPa) and 2,800 m (727 hPa) above sea level (T4–T8). Subjects and experimenters were not told the simulated altitudes and times of ascent were standardized to reduce their possibility to guess the altitude. Subjects were asked to avoid strenuous physical activity for at least 24 h and to refrain from eating 3 h prior to testing. All tests for each subject were performed at the same time of the day (± 1 h).

Table 1 Characteristics of the eight subjects

	Height (cm)	Weight (kg)	$\dot{V}O_{2max}$ at sea level (l/min)	(ml kg^{-1} min^{-1})
Mean	181	72	4.8	66
SD	3.5	4.2	0.5	4.3
Range	177–187	68–79	4.3–5.7	61–74

Test protocol A (T1–T2)

Figure 2 shows an outline of the test protocol A. After a 10 min warm up, subjects ran at four different submaximal velocities for 5 min each ①. On every stage, the oxygen uptake was measured during the last minute to determine the individual velocity/ $\dot{V}O_2$ relationship. After a 3 min break, subjects performed an incremental/ $\dot{V}O_{2max}$-test ②. Subjects started with a running intensity around their estimated individual anaerobic threshold (14.3 ± 1.5 km/h). The running speed was then increased every minute with 1 km/h until exhaustion. For the estimated last minute of the test, the subjects could choose to increase the running intensity with 0.0 or 0.5 km/h instead of 1 km/h. The mean maximal running speed was 17.3 ± 1.2 km/h. The total test time until exhaustion was 308 ± 49 s. The inclination was set at 5.3% throughout the whole test protocol A.

From these data, the running speeds to reach 55, 60, 95 and 107% of $\dot{V}O_{2max}$ (velocity associated with the 100% of $\dot{V}O_{2max} = v\dot{V}O_{2max}$) were inter- and extrapolated ③. These running speeds were used in the test protocol B at the different simulated altitudes (T3–T9). The individual calculated running velocities at different percentage of $v\dot{V}O_{2max}$ are presented in Table 2.

Test protocol B (T3–T9)

This test protocol B consisted of a submaximal and a maximal exercise test.

Submaximal exercise test

Every test (T3–T9) started with 15 min with submaximal running in normobaric conditions at 60% of $v\dot{V}O_{2max}$. During this sea level warm-up, oxygen uptake was measured to biologically control the reproducibility of the measurements. Thereafter, subjects continued to run at 55% of $v\dot{V}O_{2max}$ while the air

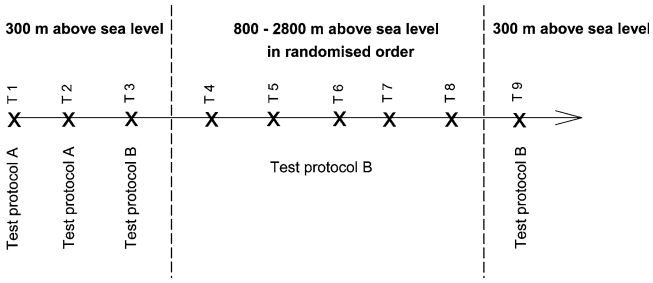

Fig. 1 Study design

pressure was gradually reduced over a period of about 2–3 min to the predetermined pressure. After the test altitude was reached, subjects continued to run for 5 min at this constant velocity. During the last 2 min of the exercise, a Douglas bag was sampled for analysis of oxygen uptake ($\dot{V}O_2$), ventilation ($\dot{V}E$), and respiratory exchange ratio (R). In the same period, arterial haemoglobin oxygen saturation (SpO_2, estimated with pulse oximetry) and heart rate (HR) were measured. After a 1 min rest, a blood sample was drawn for blood lactate ($[La^-]_b$) measurement.

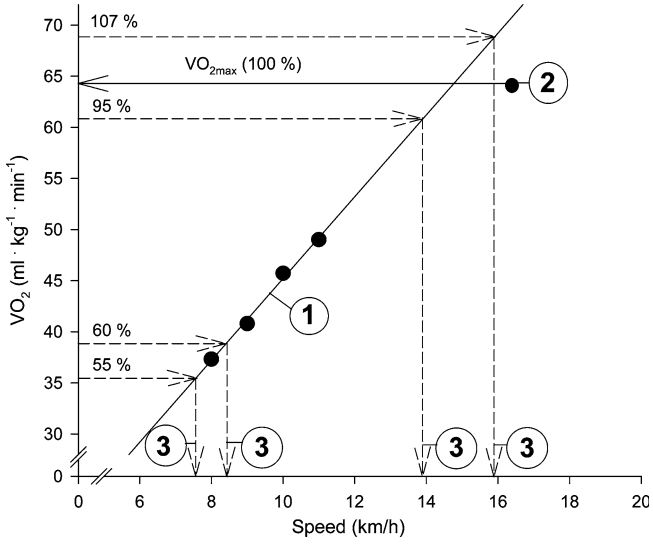

Fig. 2 Outline of test protocol A which was used to calculate the running velocities at 55, 60, 95 and 107% of $\dot{V}O_{2\,max}$. ①, ② and ③ refer to the text (for details, see text)

Maximal exercise test

Two minutes after cessation of the submaximal exercise test, the subjects began a combined $\dot{V}O_{2\,max}$ and performance test. They started at 95% of sea level $v\dot{V}O_{2\,max}$ for 1 min and continued at 107% until exhaustion. The subjects therefore ran at the same absolute velocity at all altitudes and performance was defined as time to exhaustion (TTE). Douglas bags were continuously sampled (30–40 s per bag) from approximately 90 s after the start of the test until exhaustion. SpO_2 and HR were registered every minute and at exhaustion. Blood lactate samples were taken at 1, 3 and 5 min after cessation. The highest $\dot{V}O_2$ value of one bag (collecting time 37 ± 7 s) was defined as $\dot{V}O_{2\,max}$. The highest $\dot{V}E$ and R values (always values of the last Douglas bag) were defined as $\dot{V}E_{max}$ and R_{max}. The lowest SpO_2 value and the highest HR value at test cessation was defined and SpO_{2min} and HR_{max}. The highest $[La^-]_b$ value was defined as $[La^-]_{b-max}$. TTE was defined as the total running time during the maximal test (including the 1 min at 95% of $v\dot{V}O_{2\,max}$ at the beginning of the test). The inclination was set at 5.3% throughout the whole test protocol B.

Instruments and analytical procedures

All tests were conducted in a hypobaric chamber (Norwegian Universal Technology AS, Haugesund, Norway). The tests were performed at 17 ± 0.2°C with 2.0 ± 0.2 m/s headwind and a relative humidity between 50 and 80%. Records of chamber pressure during each test showed a variation of < 1 hPa (SD). Subjects ran

Table 2 Submaximal and maximal oxygen uptake and the calculated corresponding running intensities to at 55, 60, 95 and 107% of $\dot{V}O_2$ in the eight male, sea level resident ETA (subjects A–H)

Submaximal and maximal oxygen uptake (test protocol A)						Calculated running intensities (speed in km/h used in test protocol B)		
Subject	8 km/h	9 km/h	10 km/h	11 km/h	$\dot{V}O_{2\,max}$	55% ($\dot{V}O_2$)	95% ($\dot{V}O_2$)	107% ($\dot{V}O_2$)
A	2.61	2.89	3.24	3.47	4.58	7.4	13.9	15.8
B	2.56	2.78	2.97	3.40	4.29	7.4	13.7	15.6
C	2.81[a]	2.98[a]	3.32[a]	3.60[a]	4.81	9.5	16.6	18.8
D	2.85	3.06	3.36	3.55	4.89	7.2	15.1	17.9
E	2.69	2.98	3.29	3.59	4.86	7.8	14.4	16.3
F	2.59	2.92	3.13	3.45	4.67	7.9	14.6	16.5
G	3.19	3.50	3.82	4.05	5.66	7.7	15.5	17.8
H	2.28	2.57	2.83	3.11	4.6	8.9	15.6	17.6
Mean	2.70	2.96	3.25	3.53	4.80	8.0	14.9	17.0
SD	0.26	0.27	0.30	0.26	0.40	0.8	1.0	1.2

[a] Subject C was a top elite runner and ran the submaximales stages at 10, 11, 12 and 13 km/h

on a treadmill (Bodyguard, Cardionics AB, Sweden) with a constant inclination of 5.3%. Room air was collected into Douglas bags before and after each test to measure inspired fraction of O_2 (FIO_2) and CO_2 ($FICO_2$). The mean values of the inspired air during the tests were 20.85 ± 0.03% oxygen and 0.15 ± 0.03% carbon dioxide. Expired air was collected via a mouthpiece, a two-way respiratory valve (Type 2700 series, Hans Rudolph inc, Kansas, USA), a 180 cm-long tube (diameter 44 mm) into 100 l Douglas bags (Hans Rudolph Inc.). Collecting time of expired air for each Douglas bag was automatically measured by a watch (Excelsior, Switzerland). The volume (Flow turbine, Type S-430, Kl Engineering, Northridge, USA) and gas concentrations (Oxygen analyzer type S-3A/1 and carbon dioxide analyzer type CD-3A, Amatek inc, Pittsburg, USA) were measured for each bag (including room air bags) in the laboratory at 200 m above sea level (300 m when corrected for the barometric pressure in this period). All Douglas bags where controlled for leakage before use and analysed within 20 min after the test. The $\dot{V}O_2$ and $\dot{V}CO_2$ uptake was calculated for STDP conditions. The $\dot{V}E$ values were calculated for BTPS conditions with the barometric pressure at the sampled altitude (300–2,800 m). The accuracy of the flow turbine was verified with a 3.00 l calibration syringe and was stable during the test period. Temperature and pressure sensors were controlled against a mercury temperature and pressure metre (Leybold, Hürth, Germany). Immediately prior to each test, the O_2 and CO_2 analyzer were calibrated with dry room air (20.93% O_2; 0.03% CO_2) and linearity was checked with a calibration gas with a known O_2 and CO_2 concentration (15.03% O_2; 5.02% CO_2). Fingertip $[La^-]_b$ was measured with an enzymatic lactate analyzer (Model 1500 Sport, Yellow Springs Instruments Co., Ohio, USA). SpO_2 was estimated with pulse oximetry (SC 6000, Siemens Inc., USA) using a fingertip probe. Heart rate was measured with a heart rate monitor (PE 3000, Polar Electro, Finland).

Statistics

Unless otherwise stated, the results are expressed as mean ± SE. The subjects served as their own control. Effect of simulated altitude on the different variables were analysed with one factor analysis of variance for repeated measures (SPSS). After a Mauchly test of sphericity, the P value in the F test for the intra-subject effect was calculated. When a significant main effect of the altitude was reached, Student t test for comparison of paired data was performed to test differences between 300 and 800 m as well as the rates of declines between 300–1,300 m and 1,800–2,800 m. Student t test was used to test, if there were differences between pre- and post-test. The relationship between the decrease in $\dot{V}O_{2\,max}$ and SpO_2 was compared by using linear regression and Pearson's coefficient. Differences were considered significant at $P < 0.05$; $P \leq 0.1$ was referred to as a tendency. To determine the reproducibility of measurements, the coefficients of variation (CV) were calculated from the data of the two tests at 300 m (T3 and T9).

Results

Validity of the results

There was no difference between $\dot{V}O_{2\,max}$ evaluated with the incremental test (protocol A) and the maximal exercise test (protocol B) (66.0 ± 3.8 vs. 66.1 ± 4.3 ml kg^{-1} min^{-1}, CV = 1.1%). There was no difference in $\dot{V}O_{2\,max}$ between pre- and post-test at 300 m (66.1 ± 4.3 vs. 66.6 ± 4.1 ml kg^{-1} min^{-1}). The test–retest CV determined from the two maximal exercise tests at 300 m were 1.4% ($\dot{V}O_{2\,max}$), 7.9% (TTE), 1.8% ($SpO_{2\text{-}min}$) 3.2% ($\dot{V}E_{max}$), 1.6% (Rmax), 1.6% (HR_{max}) and 3.7% for ($[La^-]_{b-max}$). The test–retest CV determined from the two submaximal exercise tests at 300 m were 2.8% ($\dot{V}O_2$), 6.5% ($\dot{V}E$), 3.4% (R), 1.0% (SpO_2), 3.3% (HR) and 19.5% ($[La^-]_b$).

Maximal exercise test

Maximal oxygen uptake

$\dot{V}O_{2\,max}$ decreased from 66.1 ± 4.3 ml kg^{-1} min^{-1} at 300 m to 55.4 ± 3.6 ml kg^{-1} min^{-1} at 2,800 m, corresponding to a 6.3% decrease per 1,000 m ($P < 0.001$; Fig. 3) with individual $\dot{V}O_{2\,max}$ decreases ranging 4.6–7.5% per 1,000 m. Already at 800 m, $\dot{V}O_{2\,max}$ was lower than at 300 m ($P < 0.01$) and the rate of decline between 300 and 1,300 m was not different from the rate of decline between 1,800 and 2,800 m ($P = 0.91$). With increasing altitude, mean $\dot{V}O_2$ was lower at all time points (Fig. 4). TTE, SpO_{2min}, $\dot{V}E_{max}$, $[La^-]_{b-max}$, HR_{max} and R_{max} are presented in Fig. 5. Already at 800 m TTE was shorter than at 300 m ($P < 0.05$) and TTE continued to decline up to 2,800 m with 14.3% per 1,000 m altitude ($P < 0.01$; range 10.3–18.1%). The rate of decline between 300 and 1,300 m was not different from the rate of decline between 1,800 and 2,800 m. SpO_{2min} declined from 89.0 ± 2.9% at 300 m to 76.5 ± 4.0 at 2,800 m ($P < 0.001$). At 300 m, all athletes had SpO_{2min} equal or lower than 92%. The rate of decline in SpO_{2min} between 300 and 1,300 m was not different from the rate of decline between 1,800 and 2,800 m ($P = 0.93$) and the mean SpO_2-altitude relationship was ($r^2 = 0.9961$; $P < 0.0001$) with linear regression and ($r^2 = 0.9932$; $P < 0.0001$) with a quadratic curvelinear regression. $\dot{V}E_{max}$ tended to increase by 2.1% per 1,000 m ($P = 0.07$) and R_{max} increased from 1.12 ± 0.04 at 300 m to 1.30 ± 0.06 at 2,800 m ($P < 0.001$). $[La^-]_{b-max}$ did not change with altitude and HR_{max} decreased by 1.9 beats/min per 1,000 m ($P = 0.01$). The variance (r^2) between the decrease in

$\dot{V}O_{2\,max}$ and the decrease in SpO_2 (at $\dot{V}O_{2\,max}$) was 0.62 if results of all athletes and all altitudes were included (Fig. 7). There was no inter-individual correlation between the individual decrease of SpO_2 at $\dot{V}O_{2\,max}$ and the individual decrease of $\dot{V}O_{2\,max}$.

Submaximal exercise test

Oxygen uptake, arterial oxygen saturation, ventilation, blood lactate, heart rate and respiratory exchange ratio during the submaximal test are presented in Fig. 6. $\dot{V}O_2$ did not change with altitude. There was a tendency for $\dot{V}E$ to increase with altitude ($P = 0.10$) while R increased with altitude ($P < 0.05$). SpO_2 decreased from $96.4 \pm 0.9\%$ at 300 m to $83.4 \pm 2.8\%$ at 2,800 m ($P < 0.001$). The rate of decline in SpO_2 was significantly steeper between 1,800 and 2,800 m than between 300 and 1,300 m ($P < 0.01$). The mean SpO_2-altitude relationship was ($r^2 = 0.9999$; $P < 0.0001$) with a quadratic curvelinear regression and ($r^2 = 0.9511$; $P < 0.0001$) with linear regression. HR increased from 133 ± 5.1 beats/min at 300 m to 150 ± 4.6 beats/min at 2,800 m ($P < 0.001$). At 2,800 m, SpO_2 was 13.4% lower, while HR was 11.9% higher compared with the values at 300 m. The decrease in SpO_2 correlated with the increase in HR ($r = 0.63$). $[La^-]_b$ increased with altitude ($P < 0.001$).

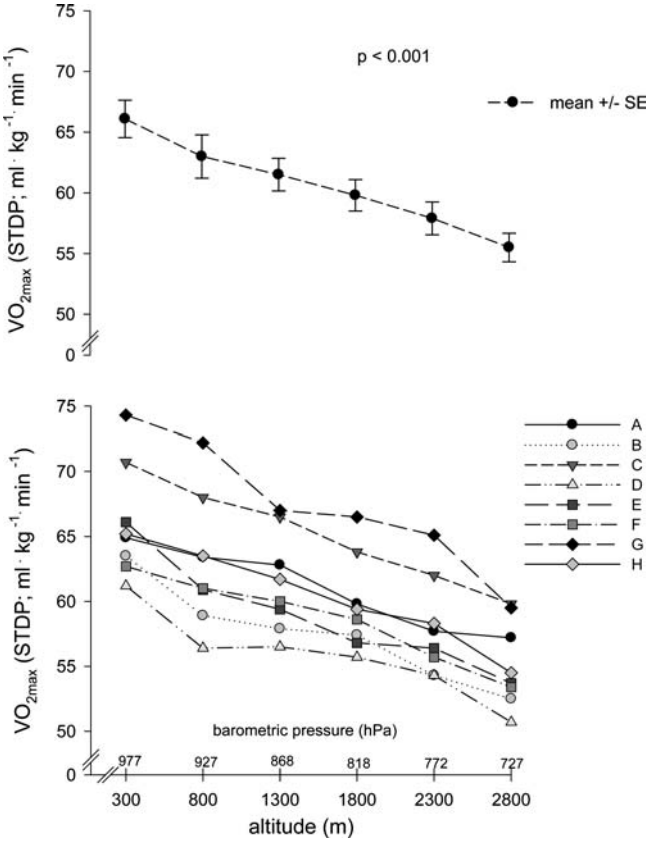

Fig. 3 Effect of acute simulated altitude exposure on $\dot{V}O_{2\,max}$ in eight male, sea level resident, endurance-trained athletes (ETA). The *upper part* of the figure shows means ± standard error (SE), the *lower part* of the figure shows the individual values of the subjects A–H

Discussion

The main finding of this study was that both performance and $\dot{V}O_{2\,max}$ declined significantly from 300 to 800 m and continued to decrease linearly to 2,800 m above sea level. This is the first study to report a linear decrease in performance and $\dot{V}O_{2\,max}$ from sea level (0–300 m) to very low (300–1,000 m), low (1,000–2,000 m) and moderate simulated altitudes (2,000–3,000 m) during acute exposure in ETA.

Linear decrease in $\dot{V}O_{2\,max}$ with altitude

Due to the sigmoidal shape of the oxyhaemoglobin dissociation curve, it has been thought that very mild hypoxia corresponding to an altitude of less than 1,500 m (PaO_2 reduction of 40–50 hPa) will have only a minor effect on the O_2 content of the arterial blood and $\dot{V}O_{2\,max}$. However, in agreement with our results, other studies have reported a reduction in $\dot{V}O_{2\,max}$ at altitudes below 1,000 m in ETA (Gore et al. 1996, 1997; Terrados et al. 1985; Chapman et al. 1999). The most important mechanism for this seems to be "exercise-induced arterial hypoxemia" (EIH), a phenomenon seen even at sea level in ETA with high $\dot{V}O_{2\,max}$ (Chapman et al. 1999; Gore et al. 1996; Terrados et al. 1985). EIH occurred already at sea level in all our athletes, according to the definition of an EIH subject as one with SpO_2 less than or equal to 92% at maximal exercise (Harms and Stager 1995; Powers et al. 1988, 1989; Warren et al. 1991). In another study, mild hypoxia ($FiO_2 = 0.187$) caused a 4% reduction in $\dot{V}O_{2\,max}$ in EIH athletes, while non-EIH athletes experienced no reduction even if there was no difference in hypoxia-induced reduction in SpO_2 and therefore no correlation between ΔSpO_2 and $\Delta\dot{V}O_{2\,max}$ (Chapman et al. 1999). Ferretti et al. (1997) concluded that ETA, contrary to untrained subjects, work at the steep part of the oxygen haemoglobin dissociation curve even at low altitudes. In the present study, we found a difference in the pattern of decline in SpO_2 with altitude during submaximal compared with maximal exercise. Whereas SpO_{2min} declined in a linear manner during maximal exercise, it followed a curvilinear pattern during submaximal exercise. This was judged by visual analyses, curve fitting and the fact that the rate of decline was steeper between 1,800 and 2,800 m than between 300 and 1,300 m during submaximal exercise, but not during maximal exercise. The curvilinear shape during submaximal exercise may reflect the sigmoidal shape of the oxyhaemoglobin dissociation curve, but why is this not seen during maximal exercise? Our hypothesis is that the linear relationship seen during

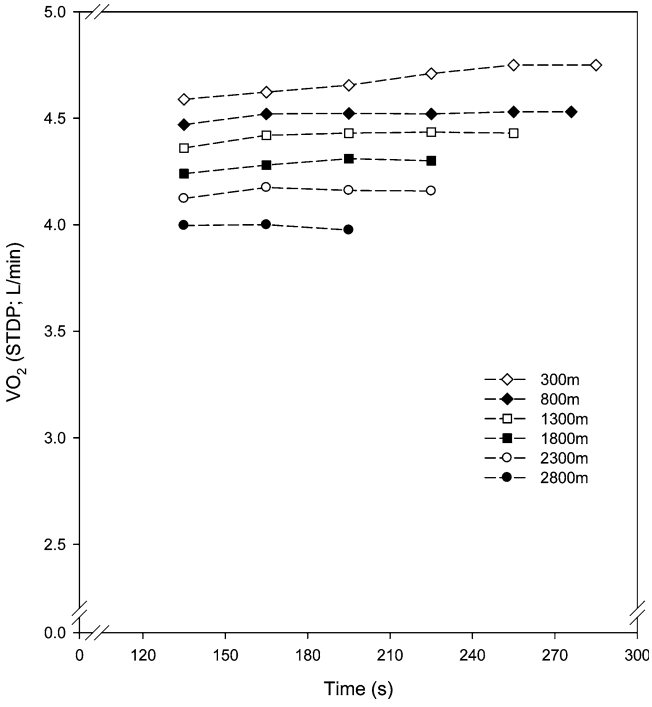

Fig. 4 Mean oxygen uptake of eight male sea level resident athletes at different time points during the constant speed $\dot{V}O_{2\,max}$ test (protocol B: 107% of v$\dot{V}O_{2\,max}$, see text) at different altitudes

maximal exercise is caused by the combined effect of exercise-induced hypoxemia and the right-shifted oxyhaemoglobin curve due to acidification of the blood during maximal exercise (Bohr effect). The Bohr effect is elegantly demonstrated both in humans and horses, by showing that an attenuation in the exercise induced reduction in pH by bicarbonate infusion also attenuate the exercise induced reduction SpO_2 with no change in arterial O_2-tension (Nielsen et al. 2002; Manohar et al. 2004). During submaximal exercise, SpO_2 is neither so low in normoxia nor affected by the Bohr effect and the altitude-induced reduction follows a curvilinear pattern. Untrained subjects, who do not experience EIH, may be less susceptible to both the hypoxia-induced lowering of SpO_{2min} and the Bohr effect during maximal exercise.

In the present study, $\dot{V}O_{2\,max}$ declined at the same rate between low altitudes (300–1,300 m) as between higher altitudes (1,800–2,800 m) and together with the results from the studies presented in Fig. 8, this supports the hypothesis that the $\dot{V}O_{2\,max}$ decreases linearly beginning at sea level in ETA. Further, it supports the conclusions of Squires and Burkirk (1982) and Gore et al. (1996) who stated that the concepts of threshold altitudes for aerobic impairment as suggested by others (Buskirk et al. 1967; Grover et al. 1986; Robergs et al. 1998) are ambiguous in the case of unacclimatized ETA.

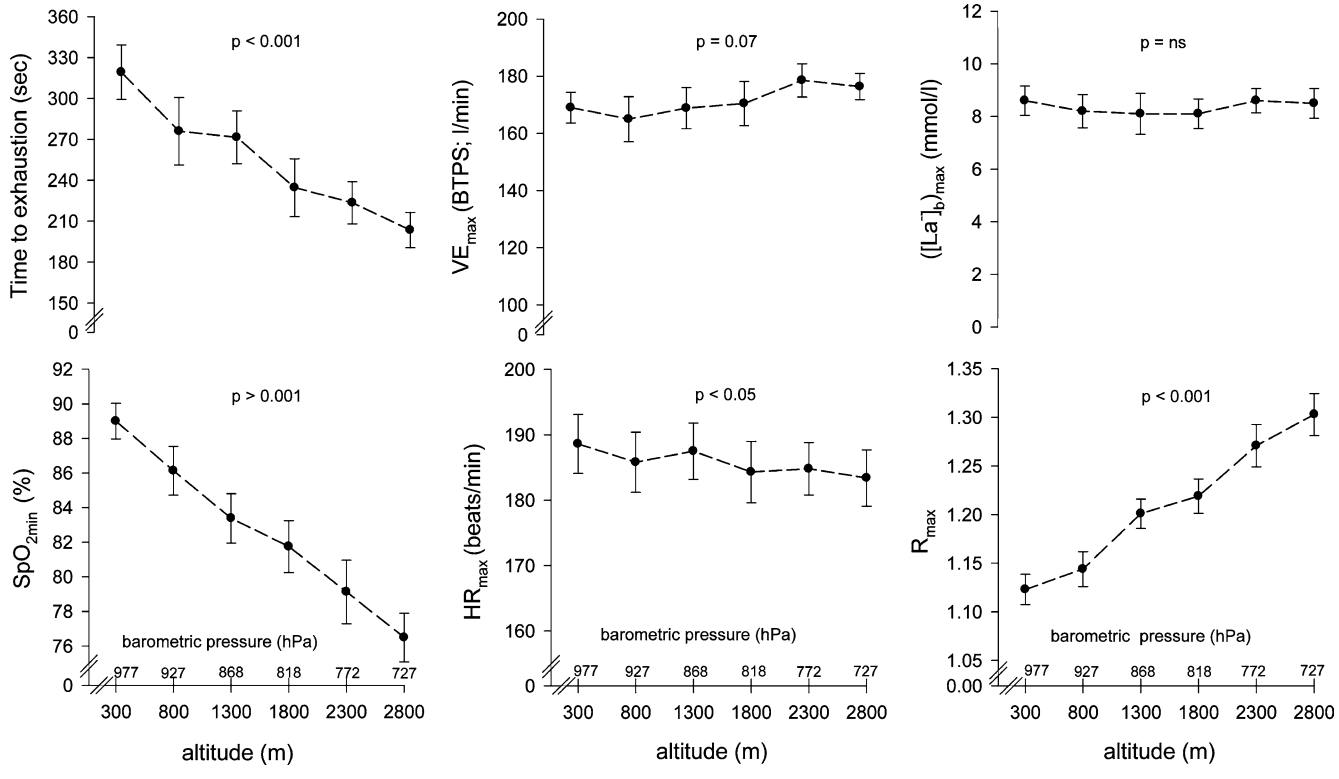

Fig. 5 Effect of acute simulated altitude exposure between 300 and 2,800 m above sea level at maximal exercise on time to exhaustion (TTE), arterial oxygen saturation (SpO_{2min}), ventilation ($\dot{V}E_{max}$), maximal heart rate (HR_{max}), maximal blood lactate concentration ($[La^-]_{b-max}$) and respiratory exchange ratio (R_{max}) in eight male sea level resident ETA. *Symbols* represent means ± standard error (SE)

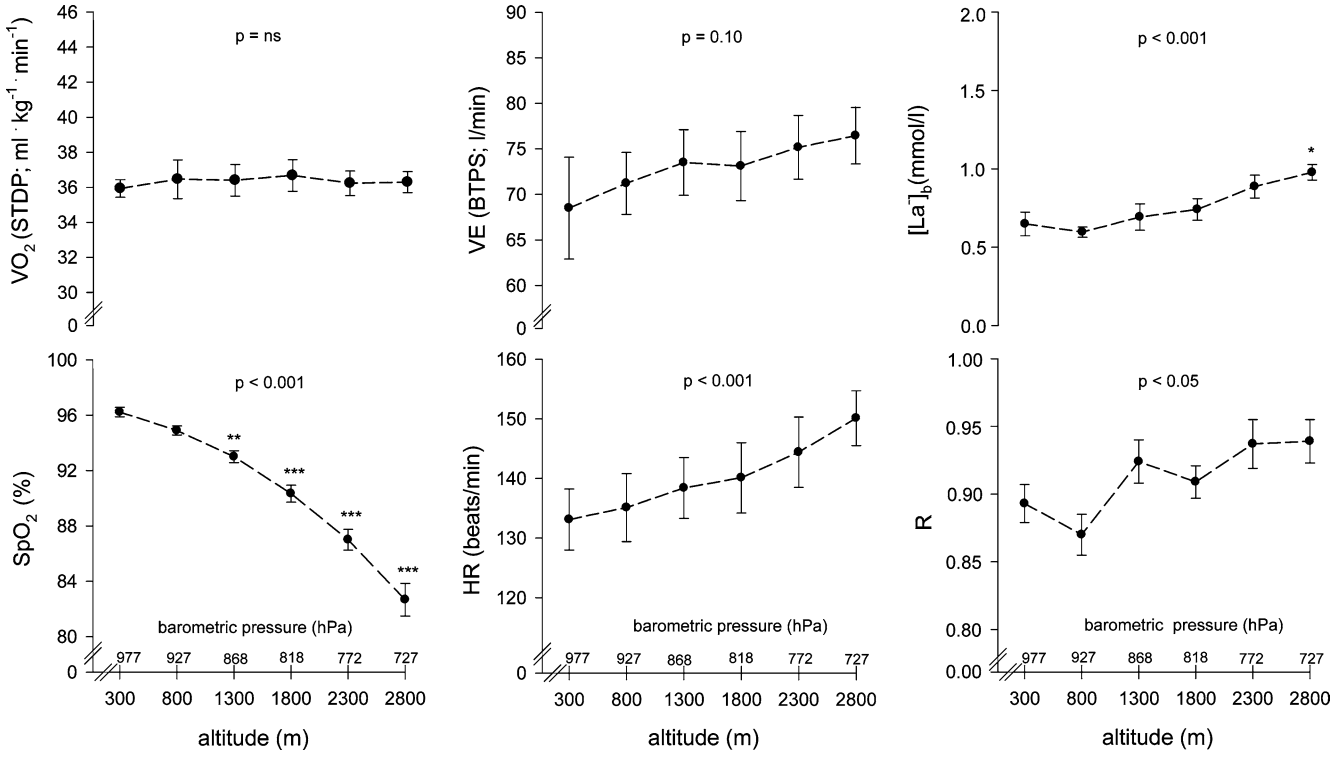

Fig. 6 Effect of acute simulated altitude exposure between 300 and 2,800 m above sea level during the submaximal exercise (55% of $v\dot{V}O_{2max}$) on oxygen uptake ($\dot{V}O_2$), arterial oxygen saturation (SpO_2), ventilation ($\dot{V}E$), heart rate (*HR*), blood lactate ($[La^-]_b$) and respiratory exchange ratio (*R*) in eight male sea level resident ETA. *Symbols* represent means ± standard error (SE)

Rate of decline of $\dot{V}O_{2max}$ with altitude

The mean decrease in $\dot{V}O_{2max}$ in the present study was 6.3% per 1,000 m, close to the 7.7% calculated from the studies in Fig. 8. Individual decreases in $\dot{V}O_{2max}$ ranged 4.7–7.5%, a small variation compared with that found in ETA earlier by Gore et al. (1996) (+1 to −12% change from 168 to 748 m above sea level) and Billat et al. (2003) (−8 to −24% from sea level to 2,400 m). Unfortunately, none of these studies or the studies included in Fig. 8 reported test–retest reproducibility. It is therefore not clear how much of the reported variability is methodological variation and how much is biological variation between the subjects.

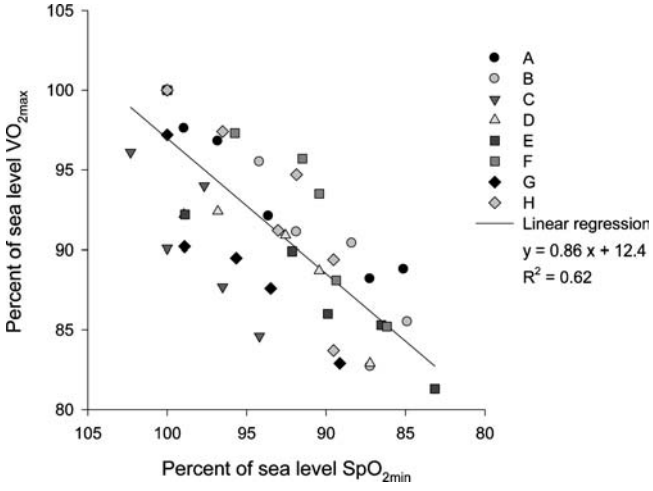

Fig. 7 Normalized $\dot{V}O_{2max}$ in relation to normalized SpO_2 (at the time point of measured $\dot{V}O_{2max}$) in all measured altitudes (300–2,800 m) in eight male, sea level resident ETA (subjects A–H). *Symbols* represent means ± standard error (SE) and regression line. The regression equation, the explained variance (r^2) and the statistical level are given

The decrease in SpO_2 is strongly associated with the decrease in $\dot{V}O_{2max}$ with altitude. According to Ferretti et al. (1997), the decrease in SpO_2 accounts for about 86% of the decrease in $\dot{V}O_{2max}$, which fits with the present study where approximately 70% of the decrease in $\dot{V}O_{2max}$ can be explained by the decrease in SpO_2 at $\dot{V}O_{2max}$. Furthermore, the decrease in $\dot{V}O_{2max}$ of 6.3% per 1,000 m is close to the decrease in SpO_{2min} of 5.5% per 1,000 m and fits the conclusion of Powers et al. (1989) that a reduction of 1% in SpO_2 below 92–93% causes a decrease of ~1% of $\dot{V}O_{2max}$. Hence, the main mechanism for the hypoxia-induced decrease in $\dot{V}O_{2max}$ at low and moderate altitude is the decrease in SpO_2. However, there was no correlation between the individual rate of decrease in SpO_2 at $\dot{V}O_{2max}$, and individual rate of decrease in $\dot{V}O_{2max}$, suggesting that there are other confounding mechanisms involved, such as a reduction in maximal cardiac output ($\dot{Q}_{max}$) (Calbet et al. 2003). In the present study, HR increased during submaximal exercise (with no change in $\dot{V}O_2$) reflecting the reduced oxygen content of the arterial blood, while HR tended to decrease during

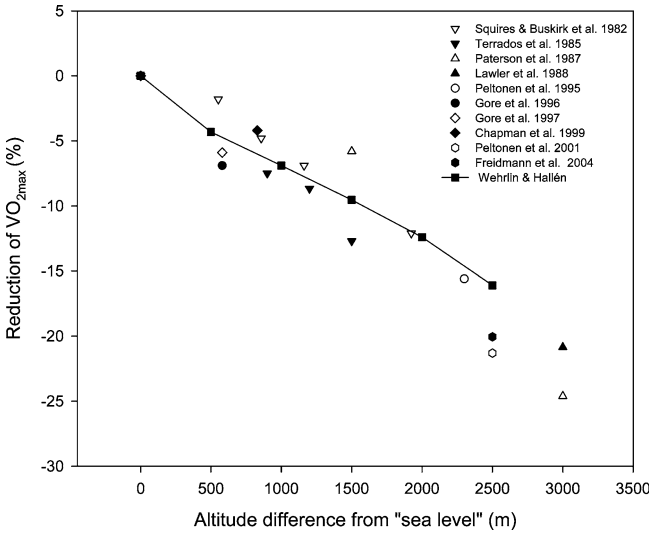

Fig. 8 Percent decrease in $\dot{V}O_{2\,max}$ from sea level values. "Sea level" in these studies is set at 0 m but varies originally from 0 to 362 m. Only studies which tested male unacclimatized ETA with a mean $\dot{V}O_{2\,max} >$ 60 ml kg^{-1} min^{-1} under laboratory conditions at acute hypoxia are included

maximal exercise, supporting the theory of a minor hypoxia-induced decrease in $\dot{Q}_{max}$.

Decrease in performance with increasing altitude

Performance (time to exhaustion) decreased between 300 and 800 m (−9.4%) and continued to decrease by a mean of 14.3% per 1,000 m increasing altitude. Surprisingly, few studies have tested the change in exercise performance at acute exposure to altitudes below 3,000 m in ETA (Gore et al. 1996, 1997; Lawler et al. 1988; Peltonen et al. 1995, 2001) and only two have tested ETA below 1,000 m. Performance was determined either with an incremental (Lawler et al. 1988; Peltonen et al. 2001) or an all out test for a given distance (Gore et al. 1996, 1997; Peltonen et al. 1995). When performance declines during a graded exhaustive protocol or a self-selective workload, it can be argued that $\dot{V}O_{2\,max}$ decreases as a result of reduced workload and muscle recruitment (Peltonen et al. 2001). In the present study, the absolute exercise intensity was the same and constant at all altitudes, and the $\dot{V}O_2$ was different at all time points (Fig. 4). This indicates that the reduced $\dot{V}O_{2\,max}$ in hypoxia is limited by O_2 availability and not due to reduced absolute exercise intensity associated with reduced muscle recruitment (Noakes et al. 2001).

Submaximal exercise intensity

Oxygen consumption at the same absolute intensity did not change with increasing altitude. This finding is in agreement with several previous studies (Fulco et al. 1996, 1998; Knuttgen and Saltin 1973; Rowell and Blackmon 1987; Peltonen et al. 1999), even though some studies reported lower $\dot{V}O_2$ values in hypoxia (Squires and Buskirk 1982; Ibanez et al. 1993). Despite the tendency of $\dot{V}E$ to increase, SpO_2 decreased in a curvilinear manner, reflecting the sigmoidal shape of the oxyhaemoglobin dissociation curve. HR increased with increasing altitude reflecting increased $\dot{Q}$ and the relative increase in HR was very similar to the relative decrease in SpO_2.

Summary

Performance and $\dot{V}O_{2\,max}$ decreased significantly already from 300 to 800 m above sea level and continued to decrease linearly by 14.3 and 6.3%, respectively, per 1,000 m altitude, up to 2,800 m. A linear decrease was also seen in arterial haemoglobin O_2 saturation during maximal exercise, whereas during submaximal exercise, the decrease was curvilinear. The decrease in $\dot{V}O_{2\,max}$ correlated with the reduction in SpO_2 at $\dot{V}O_{2\,max}$.

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