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ORIGINAL ARTICLE

Influence of moderate hypoxia on tolerance to high-intensity exercise

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Abstract It remains uncertain as how the reduction in systemic oxygen transport limits high-intensity exercise tolerance. 11 participants (5 males; age 35 ± 10 years; peak $\dot{V}O_2$ max 3.5 ± 0.4 L min^{-1}) performed cycle ergometry to the limit of tolerance: (1) a ramp test to determine ventilatory threshold (VT) and peak $\dot{V}O_2$; (2) three to four constant-load tests in order to model the linear P–t^{-1} relationship for estimation of intercept (critical power; CP) and slope (AWC). All tests were performed in a random order under moderate hypoxia ($FiO_2 = 0.15$) and normoxia. The linearity of the P–t^{-1} relationship was retained under hypoxia, with a systematic reduction in CP (220 ± 25 W vs. 190 ± 28 W; $P < 0.01$) but no significant difference in AWC (11.7 ± 5.5 kJ vs. 12.1 ± 4.4 kJ; $P > 0.05$). However, large individual variations in the change of the latter were observed (−36 to +66%). A significant relationship was found between the % change in CP ($r = 0.80$, $P < 0.01$) and both peak $\dot{V}O_2$ (CP: $r = -0.65$, $P < 0.05$) and VT values recorded under normoxia (CP: $r = -0.65$, $P < 0.05$). The present study demonstrates the aerobic nature of the intercept of the P–t^{-1} relationship, i.e. CP. However, the extreme within-individual changes in AWC do not support the original assumption that AWC reflects a finite energy store. Lower hypoxia-induced decrements in CP were observed in aerobically fitter participants. This study also demonstrates the greater ability these participants have to exercise at supra-CP but close to CP workloads under moderate hypoxia.

Keywords Power-duration relationship · Critical power · Anaerobic work capacity · Curvature constant · Hypoxaemia · Performance

Communicated by Guido Ferretti.

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Introduction

Maximal oxygen uptake ($\dot{V}O_2$ max) is a strong determinant of maximal performance. The capacity for the exercising muscles to release energy from non aerobic sources is an additional crucial determinant of exercise tolerance when exercising within the severe intensity domain, i.e. above critical power (CP). CP is the asymptote of the hyperbolic power (P)–time exhaustion (t_{lim}) relationship (di Prampero 1999). Above CP, the anaerobic work capacity (AWC; curvature constant of the hyperbolic relationship) is indeed depleted at a rate proportional to the magnitude of the power requirement [$P = (AWC/t_{lim}) + CP$]. The P–t_{lim} relationship has been described as hyperbolic but may be converted to a linear work versus t_{lim} or P versus inverse of t_{lim} (Fig. 1) allowing CP and AWC to be derived (see Morton and Hodgson 1996 for review).

Critical power reflects a rate of aerobic energy pool reconstitution dictating the maximum power that can be sustained without a progressively increasing non aerobic contribution (Jones et al. 2008; Poole et al. 1988). Work rates above CP, i.e. of severe intensity, result in a non steady state of blood (lactate), blood acid–base balance and concentrations of intramuscular metabolites such as phosphocreatine (PCr), inorganic phosphate (Pi) and hydrogen ion [H^+] (Jones et al. 2008; Poole et al. 1988). Supra-CP

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exercise is characterised by a drift in the $\dot{V}O_2$ response until peak $\dot{V}O_2$ is attained, with exhaustion occurring thereafter (Poole et al. 1988). These physiological disturbances have been associated with the depletion of AWC. AWC is traditionally suggested to be equivalent to a finite energy store comprising small amount of oxygen reserves (myoglobin and venous blood), intramuscular high-energy phosphates (ATP and PCr), and glycogen (Monod and Scherrer 1965). CP has been shown to be enhanced by endurance training (Jenkins and Quigley 1992) and hyperoxia (Ferguson et al. 2010) while AWC is sensitive to creatine supplementation (Miura et al. 1999), high-intensity strength/sprint training (Jenkins and Quigley 1993), glycogen depletion (Miura et al. 2000) and prior supra-CP exercise with limited recovery (Ferguson et al. 2007).

The deleterious effect of a reduction in the inspired fraction of oxygen (FiO_2) has been evidenced for supra-CP exercise performances lasting longer than $\sim$60 s (Calbet et al. 2003b; Weyand et al. 1999). At a given work rate, the lower aerobic energy turnover is compensated by a greater anaerobic energy turnover (Richardson et al. 1998; Richardson et al. 1999a), aggravating the rate at which metabolites associated with fatigue processes accumulate (Fitts 1994; Robergs et al. 2004; Westerblad and Allen 2003). Moderate hypoxia ($FiO_2 = 0.15$) therefore exacerbates the development of peripheral and central fatigue (Amann et al. 2007) leading to an early termination of exercise at a given work rate (Romer et al. 2007).

The reduction in the systemic oxygen transport induced by hypoxia systematically affects peak oxygen uptake (peak $\dot{V}O_2$) during constant-load and incremental exercise (Amann et al. 2007; Weyand et al. 1999). Accordingly, lower CP values were reported for the two subjects tested in the single study investigating the effects of hypoxia on the two parameters of the P–t_{lim} relationship (Moritani et al. 1981). Interestingly, one of the two subjects saw AWC increasing with severe hypoxia (FiO_2 <12%). The authors, however, concluded for a AWC independent to the environmental condition to support the initial belief that AWC was reflecting a finite store of anaerobic energy (Moritani et al. 1981).

Anaerobic work capacity was initially thought to be another surrogate of AWC (Hill 1993): its complete exhaustion would cause the exercise to cease (Moritani et al. 1981; Poole et al. 1988). However, recent findings are challenging this capacity-based explanation for task failure (Ferguson et al. 2010). The accumulation of work above CP would lead to a proportional build-up of fatigue-inducing metabolites (e.g., Pi, ADP, H^+, and extracellular K^+) that would reach "critical tolerable limits" (Jones et al. 2008; Vanhatalo et al. 2010). AWC therefore would reflect an ability to exercise under increasing levels of fatigue caused by its own utilisation (Ferguson et al. 2010). Exercise would—coincidentally—end when the accumulated work performed above CP equals AWC (Jones et al. 2008), thus irrespective of the rate of AWC expenditure (Fukuba et al. 2003).

The difficulties associated with the assessment of the "non-aerobic" capacity result in very few robust studies being conducted under hypoxia. Questions remain as to whether the ability to produce work above CP remains unchanged under hypoxia. The purpose of the present study was to establish how moderate hypoxia ($FiO_2 = 0.15$) affects supra-CP cycling performance. According to current views of the energetics of muscular exercise, anaerobic capacity would not change in hypoxia (di Prampero and Ferretti 1999). Thus, if AWC is truly an indicator of anaerobic capacity, it would not be altered by moderate hypoxia. However, recent findings do not necessarily support the original belief that AWC represents a finite energy store depleted at exercise cessation (Ferguson et al. 2010). So, we tested the hypothesis that AWC in hypoxia is an invariant. If the hypothesis is verified, the power–inverse of t_{lim} relationship would be parallel to that in normoxia. CP should be systematically reduced whilst AWC will not be altered by moderate hypoxia.

Methods

Participants

Seventeen active participants were volunteered for this study. Those demonstrating exercise-induced hypoxemia were removed from the sample (Chapman et al. 1999). The final population was composed of five active men and six women (age 35 ± 10 years; peak $\dot{V}O_2$ 3.5 ± 0.4 L min^{-1}; mass 73 ± 9 kg). No differences in the findings were evident when looking at males and females so the data were pooled to solely focus on the hypoxia intervention. All participants were briefed as to the benefits and risks of participation and gave their written informed consent to participate in the study, which was approved by the University Ethics Committee. All were familiarised with the laboratory exercise testing procedures. Participants were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h postprandial, and to avoid strenuous exercise in the 48 h preceding a test session. All were free of cardiac, metabolic or respiratory diseases.

Experimental design

The participants visited the laboratory for two stages of experimentation undertaken in random order under both

normoxia and hypoxia. Stage 1 involved the determination of peak oxygen uptake (peak $\dot{V}O_2$); stage 2 involved three to four constant-load tests to exhaustion to determine CP and AWC (one test per session). All stages were separated by a minimum of 24 h. Volunteers were allowed to self-select pedal frequency during the first testing session and then asked to maintain this cadence within ±5 rpm for all remaining tests. Participants were instructed to remain seated during each test and strong verbal encouragements were provided. The study was completed within 3 weeks for all participants.

Equipment

The tests were performed on an electrically braked cycle (Schoberer Rad Messtechnik with 8 strain gauges, SRM, Germany), the zero offset calibration procedure being performed on the SRM Powermeter before each test. Seat and handlebar heights were kept constant over the sessions for each participant. All tests were performed in a tent (Colorado Altitude Training, Colorado, USA) where air was nitrogen enriched to generate the hypoxic condition (FiO_2: 0.15). Participants entered in the tent 5 min before the start of the test, and were blind as to the inspired FiO_2 in the tent (normoxia: $FiO_2 = 0.21$; hypoxia: $FiO_2 \sim 0.15$). Room temperature was set at 20°C with 40–50% relative humidity. Arterial oxygen saturation was non invasively estimated by pulse oximetry (SpO_2) and was recorded within the 15 s preceding the commencement and at the end of each test using a finger probe (Palmsat 2500, NONIN Medical Inc., Plymouth, MI, USA). Heart rate was monitored every second using a telemetric heart rate monitor (Accurex +, Polar Electro Oy, Kempele, Finland). Haemoglobin concentrations (Hb) were also measured from the capillary blood samples taken prior the commencement of exercise (HemoCue B-Haemoglobin Analyser, Angelholm, Switzerland).

During the incremental ramp tests, pulmonary gas exchange was measured continuously using a breath-by-breath open-circuit system (Ergocard®, Medisoft, Dinant, Belgium). The participants wore a nose-clip and breathed through a light weight, low-dead space, low-resistance mouthpiece connected to a Pitot tube for the measurement of flow and integration to volume. The inspired and expired gas volumes were measured and corrected to BTPS condition and gas concentration signals were continuously sampled via a moisture exchange line. The derived $\dot{V}O_2$ and $\dot{V}CO_2$ were corrected to STPD. The analysers were calibrated before each test with dry gases of known concentration, and the Pitot tube was calibrated using a 3L syringe. Respiratory gas exchange variables ($\dot{V}O_2, \dot{V}CO_2, \dot{V}_E$) were calculated, displayed for every breath and then subsequently interpolated to provide one value per second.

Incremental ramp test

The initial power output was 50 W for 5 min and then increased by 5 W every 12 s (equating to 25 W min^{-1}) to the limit of tolerance. Maximum power (*P*max) was derived from the SRM data logger as the highest averaged power over 60 s. The highest 60 s average of the second-by-second $\dot{V}O_2$ data was recorded as peak $\dot{V}O_2$. Peak heart rate (HR) was computed as the highest 1 min value recorded during the test. Breath-by-breath data were converted to 15 s averages for the determination of VT in normoxia. VT was defined as the $\dot{V}O_2$ at which a non-linear increase in carbon dioxide production ($\dot{V}CO_2$) and an increase in minute ventilation ($\dot{V}_E$) and in $\dot{V}_E/\dot{V}O_2$ with no increase in $\dot{V}_E/\dot{V}CO_2$ were evident (Wasserman et al. 1973). Three independent investigators blindly reviewed the plots of each index and made individual determinations of VT. To calculate the power output corresponding to peak $\dot{V}O_2$ (P-$\dot{V}O_2$peak) for each individual, a regression analysis was carried out on the second by second $\dot{V}O_2$ data to determine the *y*-intercept (normoxia: 610 ± 30 ml min^{-1}; hypoxia: 626 ± 56 ml min^{-1}) and the slope or functional gain ($\Delta\dot{V}O_2/\Delta w$; normoxia: 10.02 ± 0.99; hypoxia: 9.48 ± 1.41 ml min^{-1} W^{-1}) of the $\dot{V}O_2$–power output relationship (adjusted r^2 and SEE of 0.92 ± 0.07 and 10.0 ± 2.5 ml min^{-1} in normoxia and 0.96 ± 0.03 and 7.8 ± 2.2 ml min^{-1} in hypoxia).

The *P*–*t* relationship

Participants performed a series of three to four constant-load tests to the limit of tolerance, each at different power output (from 72 to 108% of P-$\dot{V}O_2$peak) chosen to induce exhaustion over a range of t_{lim} of 3–12 min (Poole et al. 1988). All exercise tests were conducting from an initial 50 W baseline of 5 min. For each individual, CP and AWC were estimated as the power intercept and slope, respectively, of the least-squares linear regression of power versus t_{lim}^{-1} (Poole et al. 1988). The coefficients of determination (adjusted r^2) and the standard error of the CP and AWC estimates were calculated to examine the goodness of fit of the data to the models (SPSS, SPSS Inc., Chicago, IL, USA). A fourth test was performed if the standard error of CP and AWC estimates were greater than 5 W and 2 kJ, respectively. Only data from the shortest and longest exercise is presented in the "Results" for ease of reading. Figure 1 illustrates the effect of hypoxia on the power–duration relationship in a representative individual. The power outputs predicted to induce exhaustion in 3 (P_3) and 15 min (P_{15}) were subsequently calculated from the modelling.

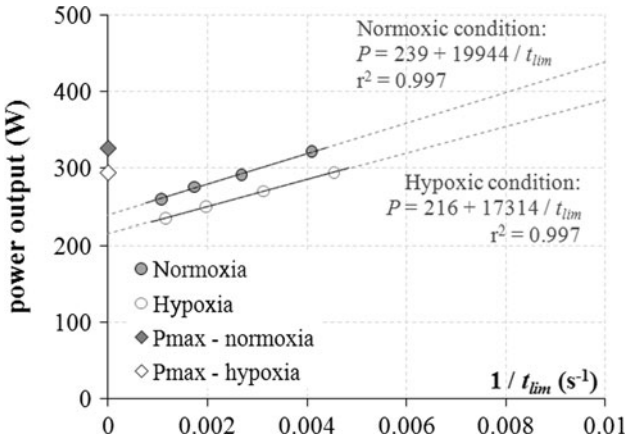

Fig. 1 The linear power–inverse of time relationship during severe-intensity cycling exercise in normoxia (*closed circles*) and hyperoxia (*open circles*) in one participant

Statistical analysis

Data are reported as mean ± SD unless stated otherwise. For each set of data, normal distribution (Shapiro–Wilk test) and homogeneity of variance were checked. Accordingly, a Student *t* test for paired data, and a one- or two-way ANOVA with repeated measures were used to determine the differences between two or more sets of data ($df = 10$). The two P–t_{lim}^{-1} relationships (normoxia vs. hypoxia) were also modelled using a least-squares linear regression with all the data points being included in the model ($df = 64$). Differences between the two slopes and intercepts were tested using a Student *t* test (Glantz 2005). The Bonferroni correction post hoc test was used when F was significant in the ANOVA according to the Greenhouse–Geisser procedure. Relationships were explored using Pearson's product-moment correlation. A 95% level of confidence was accepted for all comparisons.

Results

Incremental ramp test

Table 1 displays the findings from the two exhaustive incremental ramp tests. FiO_2 was 15.2 ± 0.5% prior the exercise performed under hypoxia. SpO_2 under hypoxia was significantly reduced at rest, and at the end of the exercise with greater decrement over time when compared with normoxia ($P > 0.05$). The average (Hb) levels obtained prior the two ramp tests (14.5 ± 1.1 g dL^{-1} and 14.5 ± 1.2 g dL^{-1} for the normoxic and hypoxic conditions, respectively) were not significantly different ($t = 0.64$, $P = 0.95$). Under both conditions, $\dot{V}O_2$ increased as a linear function of power output (adjusted r^2 and SEE of 0.92 ± 0.07 and 10.0 ± 2.5 ml min^{-1} in normoxia and 0.96 ± 0.03 and 7.8 ± 2.2 ml min^{-1} in hypoxia) with a functional gain $\left(\Delta\dot{V}O_2/\Delta w\right)$ unaffected ($t = 1.78$, $P > 0.05$) and a peak $\dot{V}O_2$ reduced under hypoxia ($t = 7.48$, $P < 0.05$). The two sets of peak $\dot{V}O_2$ values were significantly correlated ($r = 0.91$; $P > 0.05$). The reduction in peak $\dot{V}O_2$ (−12.4 ± 5.3%) was not significantly correlated with the peak $\dot{V}O_2$ values recorded under normoxia ($r = 0.29$; $P > 0.05$). The decreases in Pmax ($t = 27.9$, $P < 0.05$), P-$\dot{V}O_2$peak ($t = 5.91$, $P < 0.05$) and peak heart rate ($t = 5.52$, $P < 0.05$) were also significant under the hypoxic condition. VT occurred at 2.14 ± 0.09 L min^{-1} during the incremental test performed under normoxia, which was equivalent to 61 ± 2% of peak $\dot{V}O_2$ on average.

The P–t_{lim} relationship

Stage 2 involved three to four constant-load exercises to exhaustion to determine the CP and AWC. FiO_2 was

Table 1 Incremental ramp test responses under the two conditions

Mean ± SD	Normoxia	Hypoxia	% change	
Pmax (W)	294 ± 29	260 ± 30	−11.9 ± 2.1	*
P-$\dot{V}O_2$peak (W)	288 ± 30	259 ± 42	−10.6 ± 1.6	*
Peak $\dot{V}O_2$ (L min^{-1})	3.50 ± 0.39	3.07 ± 0.45	−12.4 ± 5.3	*
$\Delta\dot{V}O_2/\Delta w$ (mL min^{-1} W^{-1})	10.02 ± 0.99	9.48 ± 1.41	/	n.s.
Peak heart rate (beats min^{-1})	187 ± 11	179 ± 11	−3.9 ± 2.4	*
Baseline $[La]_B$ (mmol L^{-1})	1.19 ± 0.35	1.21 ± 0.26	/	n.s.
Post-ex $[La]_B$ (mmol L^{-1})	7.71 ± 1.56	7.94 ± 1.61	/	n.s.
Resting SpO_2 (%)	98.5 ± 0.7%	92.6 ± 4.2%	−6.4 ± 4.0	*
End exercise SpO_2 (%)	93.1 ± 3.1%	81.5 ± 5.3%	−12.4 ± 7.0	*

n.s. Not significantly different between the two conditions ($P > 0.05$)

* Significantly different between the two conditions ($P < 0.05$)

Table 2 Constant workload exercise under the two conditions

Mean ± SEM	Short trial		Long trial		
	Normoxia	Hypoxia	Normoxia	Hypoxia	
Power output (W)	285 ± 34	249 ± 33	236 ± 27	208 ± 29	**
Power output (%*P*-$\dot{V}O_2$peak	99 ± 6	97 ± 6	82 ± 5	81 ± 5	n.s.
t_{lim} (s)	175 ± 45	217 ± 74	636 ± 100	621 ± 132	n.s.
Resting SpO_2 (%)	98 ± 2	92 ± 4	98 ± 1	94 ± 3	**
End exercise SpO_2 (%)	93 ± 5	81 ± 4	93 ± 3	81 ± 5	**
Peak HR (beat min^{-1})	180 ± 17	173 ± 13	177 ± 12	178 ± 12	n.s.

n.s. Not significantly different between the two conditions ($P > 0.05$)

** Significantly different between the two conditions ($P < 0.01$)

14.9 ± 0.1% prior the exercises performed under hypoxia. Table 2 presents the average SpO_2 values recorded at rest and at end exercise as well as values of power output (W, %*P*-$\dot{V}O_2$peak) and times to exhaustion (t_{lim}) for the shortest and longest constant workload tests. Peak heart rates are also included in the table. SpO_2 under hypoxia was significantly reduced at rest and at end exercise ($P < 0.05$), with a significant greater decrement over time ($P < 0.05$). The decrement was similar between the short versus long test ($P > 0.05$). Peak heart rate values recorded during the t_{lim} were not significantly different between the two conditions nor between the shortest and longest exercise ($P > 0.05$).

The ranges of t_{lim} used to model the P–t_{lim}^{-1} relationship were not significantly different between the two conditions (short: $t = 1.79$, $P > 0.05$; long: $t = 0.558$, $P > 0.05$). To obtain similar ranges of t_{lim}, the absolute power output had to be lowered under hypoxia (short: $t = 11.1$, $P > 0.05$; long: $t = 10.3$, $P > 0.05$) but when expressed in % of *P*-$\dot{V}O_2$peak, no significant difference in P was obtained between the two conditions (short: $t = 0.72$, $P > 0.05$; long: $t = 0.95$, $P > 0.05$). Table 3 displays the findings from the modelling. No significant difference between the standards of estimates (SE) of CP ($t = 0.74$, $P > 0.05$) and AWC ($t = 0.48$, $P > 0.05$), as well as the adjusted r^2 ($t = 0.02$, $P > 0.05$) were found between the two conditions. Overall, hypoxia altered exercise tolerance over the entire P–t_{lim} relationship for four subjects and for t_{lim} greater than 76 ± 35 s for the remaining seven subjects. All actual recorded t_{lim} used to model the P–t_{lim} relationship were longer than 2 min and were therefore affected by the hypoxic condition. Both P_3 ($t = 8.91$, $P < 0.01$) and P_{15} ($t = 10.92$, $P < 0.01$) estimated from the modelling were significantly lowered in hypoxia.

When pooling all the data point together, the intercept (CP) of the P–t_{lim}^{-1} relationship was significantly lower under hypoxia ($t = -5.69$; $P < 0.05$) while the slope (AWC) was not significantly different to the value obtained under normoxia ($t = -0.907$; $P > 0.05$). CP was systematically reduced under hypoxia with individual changes ranging from −6 to −25% ($t = 8.23$, $P < 0.05$). A strong correlation was obtained between the two sets of CP ($r = 0.90$, $P < 0.05$; Fig. 2). The percent change in CP (−14.0 ± 6.2%) and peak $\dot{V}O_2$ (−12.0 ± 1.4%) were not significantly different ($t = -2.38$, $P < 0.05$) but were not significantly correlated ($r = -0.07$, $P > 0.05$). Similarly, the percent change in CP and *P*-$\dot{V}O_2$peak (−10.6 ± 1.6%) were not significantly different ($t = -1.86$, $P < 0.05$) and not significantly correlated ($r = 0.55$, $P > 0.05$).

Table 3 Modelling of the P–t_{lim}^{-1} relationship under the two conditions

Mean ± SEM	Normoxia	Hypoxia	% change	
CP (W)	220 ± 25	190 ± 28	−14.0 ± 6.2	*
CP (%*P*max)	82 ± 9%	71 ± 11%	−11.4 ± 4.6	*
CP (%*P*-VO_2peak)	76 ± 5%	73 ± 5%	/	n.s.
AWC (kJ)	11.7 ± 5.5	12.1 ± 4.4	/	n.s.
SE of CP (W)	3.3 ± 2.3	3.8 ± 1.4	/	n.s.
SE of AWC (J)	993 ± 618	1,095 ± 371	/	n.s.
Adjusted r^2	0.95 ± 0.08	0.95 ± 0.07	/	n.s.
P_3 (W)	285 ± 42	257 ± 37	−9.6 ± 4.3	*
P_{15} (W)	233 ± 27	203 ± 29	−13.0 ± 4.5	*

n.s. Not significantly different between the two conditions ($P > 0.05$)

* Significantly different between the two conditions ($P < 0.05$)

Mean AWC was not significantly affected by the hypoxic condition ($t = 0.43$, $P > 0.05$) although the individual differences varied from a −36% decrement to a 66% increase. Correlations was found significant between the two sets of AWC ($r = 0.71$, $P < 0.05$; Fig. 2). A significant negative relationship was found between the percent change in CP and the percent change in AWC ($r = 0.80$, $P < 0.01$; Fig. 3). The percent change in CP and AWC were both significantly correlated with peak $\dot{V}O_2$ (Fig. 4; CP: $r = -0.65$, $P < 0.05$; AWC: $r = -0.65$, $P < 0.05$) and VT (CP: $r = -0.65$, $P < 0.05$; AWC: $r = -0.65$, $P < 0.05$).

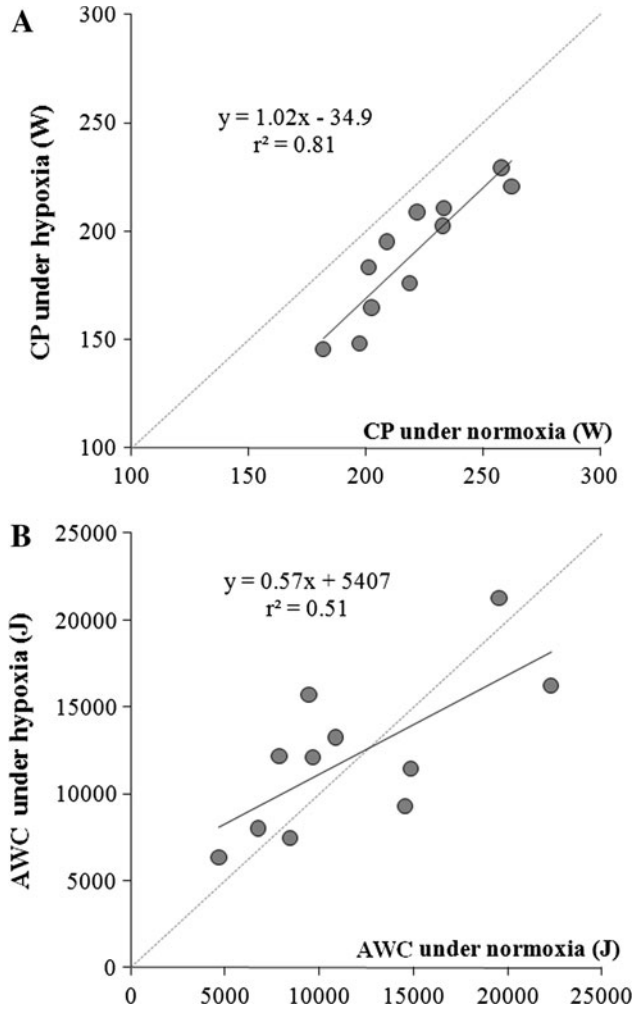

Fig. 2 Hypoxia versus Normoxia values of CP (in W; **a**) and AWC (in J; **b**)

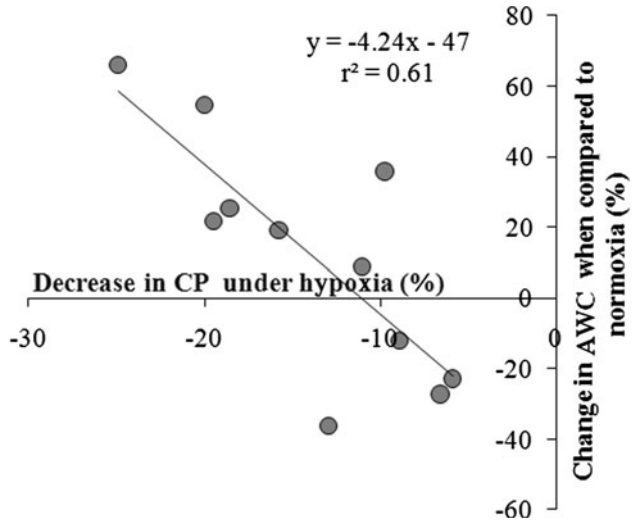

Fig. 3 Relationship between the percent decrease in CP and percent change in AWC when comparing the hypoxia versus normoxia values (%)

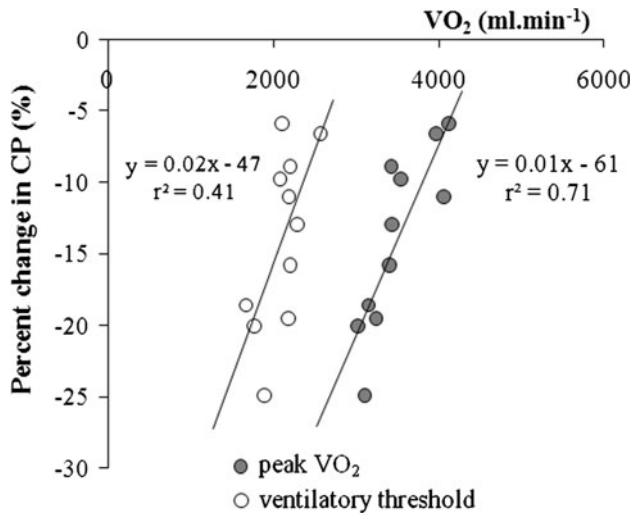

Fig. 4 Percent change in CP (%) as functions of normoxic peak $\dot{V}O_2$ (ml min^{-1}) and VT (ml min^{-1})

Discussion

This is the first study examining exercise tolerance under normoxia and moderate hypoxia (FiO_2 ~0.15) using the P–t_{lim} relationship. A two-parameter modelling was used to estimate both aerobic and non aerobic components of exercise tolerance. The present study demonstrates that a moderate lowering of FiO_2 is sufficient to induce a substantial decrease in the arterial oxygen saturation at rest (S_pO_2 ~93% under hypoxia) and systematically affect CP (−14%) as well as peak $\dot{V}O_2$ (−12%) and Pmax (−12%), and P-$\dot{V}O_2$peak (−11%). In line with the research hypothesis, CP is sensitive to moderate hypoxia. Additionally, AWC remained unchanged by the decrease in FiO_2. The capability to produce supra-CP work during severe-intensity exercise does not seem to be environment-dependent although the wide range of change in some participants (–36 to +66%) demonstrates that AWC is not a finite energy store that becomes depleted at exercise exhaustion.

The levels of SpO_2 when resting under hypoxia were close to those recorded at the end of the exercise performed under normoxia (Tables 1 and 2). With the prior exercise (Hb) levels being similar between the two conditions, the reduction in the arterial content of oxygen (CaO_2) prior the commencement of the tests performed under the normobaric hypoxic condition can be confirmed. Additionally, the drop in SpO_2 during the constant-load exercise was greater under hypoxia, attesting to the bigger hypoxic stress when FiO_2 was lowered (Romer et al. 2007). In the present study, the moderate normobaric hypoxic condition induced a systematic reduction in peak $\dot{V}O_2$ of −12%, as previously reported in the literature for FiO_2 of ~0.15 (Ferretti et al.

1997; Peltonen et al. 2001; Ponsot et al. 2010). The reduction in the systemic oxygen delivery (Calbet et al. 2003a, 2009) and diffusion to the bodily tissues (Richardson et al. 1999b, 1998) could jeopardise the utilisation of oxygen by the mitochondria of skeletal and cardiac muscles (Calbet et al. 2009; Richardson et al. 1999b). Limitations of both O_2 delivery (more likely) and O_2 extraction explains the lower peak $\dot{V}O_2$ recorded under hypoxia, and partly explains the reduction in supra-CP performance under moderate hypoxia.

*P*max (−12%) as well as the power output associated with t_{lim} of 3 and 15 min were indeed systematically reduced in hypoxia (−10% and −13%). A lowering of FiO_2 altered exercise tolerance over the whole severe-intensity section of the P–t_{lim} relationship in four participants and for t_{lim} greater than 76 ± 35 s in the remaining seven participants. For a given supra-CP power output, the limitation in muscular $\dot{V}O_2$ increases the reliance on the non-aerobic energy turnover for the total energy demand to be met (Hogan et al. 1999). This has been shown to exacerbate the development of peripheral fatigue within the exercising muscles, which contributes to the early termination of supra-CP exercise (Romer et al. 2007) especially at moderate hypoxia (Amann et al. 2007). Interestingly, the percent changes in P_{max}, P_3 and P_{15} are associated with large coefficient of variation (18, 42 and 33%) demonstrating that the capacity to cope with a given reduction in FiO_2 is individual-dependent for constant-load tests as it is during incremental ramp tests (Ponsot et al. 2010; Romer and Dempsey 2006).

It is generally accepted that CP represent the lower limit of the severe intensity domain or the lowest work rate at which maximal $\dot{V}O_2$ would be attained at exhaustion. Closely associated with the maximal lactate steady state (Burnley et al. 2006), CP would be limited by the maximal rate of aerobic energy pool reconstitution (di Prampero 1999; Ferguson et al. 2010). CP was indeed systematically affected by moderate hypoxia (Fig. 2). Interestingly in the present study, moderate hypoxia had a systematic and proportional deleterious effect on both CP (−14%) and peak $\dot{V}O_2$ (−12%) but with no correlation found between their respective within-participant percent change ($r = -0.07$, $P > 0.05$). Similar findings were obtained between CP and P-$\dot{V}O_{2peak}$. This demonstrates that peak $\dot{V}O_2$, determined from a ramp exercise test, is not such a strong putative determinant of CP. The associated peak power may not reflect the actual maximal aerobic power of the participants.

The capability in producing work above CP during severe-intensity exercise is not affected by a decrease in FiO_2 (Table 3). Despite a greater rate of accumulation of fatigue-induced metabolites during the exercise performed under hypoxia, typically exercise ceased when normoxia AWC was totally utilised. Accordingly, at the end point of supra-CP exercise, the accumulated work above CP was reported independent of the rate of AWC expenditure (Fukuba et al. 2003). This is in line with the original hypothesis that AWC would represent a finite store of anaerobic energy (Moritani et al. 1981). However, the changes in AWC with hypoxia were largely individual with changes ranging from −36 to +66%. Similar findings were reported when investigating the sensitivity of the P–t_{lim} relationship to hyperoxia (Vanhatalo et al. 2010). In the present study, some participants therefore did not use fully their supra-CP capacity under normoxia and could produce more "non-aerobic" work under hypoxia whilst others could not access their whole AWC before exhaustion under hypoxia. This represents a novel finding.

Interestingly, AWC tends indeed to be greater in hypoxia when compared with normoxia in participants whose CP decreases by more than ∼12% with hypoxia (Fig. 3). Their reduced ability to cope with hypoxia during the longer constant-load trials widens their severe-intensity domain. The physiological profile of these participants includes lower normoxia-values of peak $\dot{V}O_2$ and VT (Fig. 4). A lower muscular oxidative capacity would explain their lesser ability to exercise over long periods of time and this is exacerbated under lower levels of FiO_2. Recent findings reported hypoxia to induce greater fatigue and disruption of cellular homeostasis in rat hindlimb muscle composed primarily of fibres with low oxidative capacity (Howlett and Hogan 2007). Conversely, participants whose CP were less affected by hypoxia (0 to ∼12%) saw their AWC decreasing slightly in hypoxia resulting in a flattening of the P–t_{lim} relationship (Fig. 3). These participants were generally more aerobically trained (Fig. 4) and demonstrated better capacities in coping with fatigue during the longest trials under hypoxia. To our knowledge, this is the first performance-based study showing a greater ability in aerobically fitter participants to perform under hypoxia. These participants could even accumulate greater supra-CP work while the systemic oxygen transport was reduced. In agreement with these findings, the performance during a 30 s Wingate test in severe acute hypoxia has been shown to be maintained or barely reduced in trained endurance cyclists, owing to the enhancement of the anaerobic energy release (Calbet et al. 2003b).

Conclusion

The present study demonstrates the aerobic nature of the intercept of the P–t^{-1} relationship. Indeed, CP was systematically affected by hypoxia (−3% to −23%). However, the change in CP with moderate hypoxia was not mirrored by changes in peak $\dot{V}O_2$ demonstrating peak $\dot{V}O_2$

determined from a ramp exercise test, is not such a strong putative determinant of CP. The moderate reduction in FiO_2 did not change the average value of the second parameter of the P–t_{lim} relationship although large intra-participant changes were depicted. These findings demonstrate that AWC does not simply reflect a simple source of stored energy. Finally, this study demonstrates the greater ability aerobically fitter participants have to exercise at constant workloads with lesser changes in CP occurring under moderate hypoxia.

Conflict of interest None.

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