Power and peak blood lactate at 5050 m with 10 and 30 s ‘all out’ cycling

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ABSTRACT

Anecdotal observations suggest that the reduction in peak lactate accumulation in blood ([La]b peak) after exhausting exercise, in chronic hypoxia vs. normoxia, may be related to the duration of the exercise protocol, being less pronounced after short supramaximal exercise than after incremental exercise (IE) lasting several minutes. To test this hypothesis, six healthy male Caucasians (age 36.8 ± 7.3, x ± SD) underwent three exercise protocols on a cycle ergometer, at sea level (SL) and after 21 ± 10 days at 5050 m altitude (ALT): (1) 10 s, (2) 30 s ‘all out’ exercise and (3) IE leading to exhaustion in ~20–25 min. ‘Average’ power output ($P_{ave}$) was calculated for 10 or 30 s ‘all out’; maximal power output ($P_{max}$) was determined for IE. Lactate concentration in arterialized capillary blood ([La]b) was measured at rest and at different times during recovery; the highest [La]b during recovery was taken as [La]b peak. No significant differences in $P_{ave}$ were observed between SL and ALT, for either 10 or 30 s ‘all out’ exercise; $P_{max}$ during IE was significantly lower at ALT than at SL. [La]b peak after 10 s ‘all out’ was unaffected by chronic hypoxia (7.0 ± 0.9 at ALT vs. 6.3 ± 1.8 mmol L$^{-1}$ at SL). After 30 s ‘all out’ the [La]b peak decrease, at ALT (10.6 ± 0.6 mmol L$^{-1}$) vs. SL (12.9 ± 1.4 mmol L$^{-1}$), was only ~50% of that observed for IE (6.7 ± 1.6 mmol L$^{-1}$ vs. 11.3 ± 2.8 mmol L$^{-1}$). Muscle power output and blood lactate accumulation during short supramaximal exercise are substantially unaffected by chronic hypoxia.

Keywords chronic hypoxia, lactate paradox, supramaximal exercise.

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METHODS

Six healthy male Caucasian lowlanders (age 36.8 ± 7.3 years, Land ± SD) participated in the study after providing an informed consent. The study was approved by the Ethical Committee of our institution and by the Royal Nepal Academy of Science and Technology, Kathmandu, Nepal. All tests were conducted under close medical supervision. Three of the subjects were experienced mountain climbers, whereas the other three were recreationally active lowlanders, without specific experience in mountain climbing. Heterogeneity of subjects as far as climbing experience should not represent a significant limitation for the present study, because previous investigations (Oelz et al. 1986) showed that ‘anaerobic’ performance is not significantly different in elite mountain climbers and in healthy untrained subjects. Maximal O2 uptake (V\textsubscript{O2max}) divided by body mass (m\textsubscript{b}) of the subjects (during cycloergometric exercise) was 45.3 ± 12.3 mL kg\textsuperscript{-1} min\textsuperscript{-1} at SL before departure for the expedition (see below) and 32.0 ± 8.1 mL kg\textsuperscript{-1} min\textsuperscript{-1} at ALT (P < 0.05). Body mass was 77.0 ± 10.2 kg at SL and 71.0 ± 10.6 at ALT (P < 0.05). Blood haemoglobin concentration was 15.2 ± 0.6 g 100 mL\textsuperscript{-1} at SL and 17.9 ± 0.4 at ALT (P < 0.05).

Sea level experiments were conducted in Milan, Italy (122 m), whereas ALT experiments were conducted after 21 ± 10 days of sojourn at 5050 m (Ex-K2-CNR ‘Pyramid’ Laboratory, set near Lobuche, Khumbu, Nepal). The altitude laboratory was equipped with a stabilized electrical supply powered by a water turbine. Temperature inside the laboratory during the experiments ranged between 17 and 22 °C. At ALT the subjects had free access to drinking water and to a wide variety of palatable food items. After arriving at the ALT laboratory, and before performing the tests, three subjects (i.e. the mountain climbers, see above) spent a few days at ALTs of ~6400 m.

Both at SL and at ALT the subjects underwent three exercise protocols, conducted on the same mechanically braked cycle ergometer (Ergomedic 818E; Monark, Varberg, Sweden): (1) an incremental exercise (IE) (30 W added every 4 min, starting from 60 W at SL and from 30 W at ALT, up to voluntary exhaustion, defined as the inability to maintain the imposed pedalling frequency of 60 r.p.m.; (2) 10 s ‘all out’ and (3) 30 s ‘all out’ exercise (di Prampero & Mognoni 1981, Bar-Or 1987). Each subject was familiarized with the ‘all out’ exercise protocols during specific sessions conducted both at SL and at ALT. Subjects had to stay seated on the saddle throughout the test, and their feet were firmly secured by straps to the pedals. About 10 min after a light warming-up exercise, the subjects, starting from rest, were vigorously encouraged to reach maximal pedalling rate as quickly as possible, and to maintain it throughout the test. Pedalling rate was recorded by an electromechanical switch connected to a paper chart recorder. During the 10 and 30 s ‘all out’ exercise the resistive force (F) against the cycle flywheel was set at 0.735 N kg\textsuperscript{-1} of m\textsubscript{b} measured before performing each test. Work performed during each pedalling cycle (W) was calculated by multiplying F times the distance covered per revolution. Power output (P) was calculated for each pedalling cycle as W divided by the duration of the cycle. ‘Average’ P (\bar{P}) (di Prampero & Mognoni 1981) was then calculated for the 10 or 30 s ‘all out’ exercise periods; \bar{P} was also normalized per kg of m\textsubscript{b} (\bar{P} m\textsubscript{b}\textsuperscript{-1}) measured before performing each test. \bar{P}, as obtained in the present study, can be defined as an ‘average’ maximal power output because it represents the average value obtained during a 10 or 30 s ‘all out’ effort, to be distinguished from the significantly higher ‘instantaneous’ or ‘peak’ values obtained during a single movement of less than 1 s duration (di Prampero & Mognoni 1981). Both at SL and at ALT each subject performed 1–3 repetitions of 10 and 30 s ‘all out’ exercises, and average values were calculated for each subject. Each repetition was performed on a different day. The maximal power during IE (P\textsubscript{max}) was calculated and taken as the highest load sustained for at least 2 min; P\textsubscript{max} was also divided per unit by m\textsubscript{b} (P\textsubscript{max} m\textsubscript{b}\textsuperscript{-1}). At rest before the exercise protocols, and after 1, 3, 5, 10, 30 min and 12 min of inactive recovery after each exercise, 20 μL of arterialized capillary blood were obtained from an earlobe, and lactate concentration ([La\textsubscript{h}]) was determined by an enzymatic method (ESAT 6661 Lactat, Eppendorf, Hamburg, Germany). Lactate concentration measurements were performed on whole blood, which was haemolysed in the test tube before the measurement. Arterialization was achieved by prior application of a hyperaemia-inducing ointment (Trafuril, Ciba-Geigy, Basel, Switzerland). The highest [La\textsubscript{h}] during recovery was taken as [La\textsubscript{h}].

Statistical analysis

The values are given as mean ± standard deviation (\bar{x} ± SD). To determine the statistical significance of differences between two means, a paired Student’s t-test (two-tailed) was performed. To determine the statistical significance of differences amongst three means, a repeated-measures of analysis of variance was performed; a Tukey’s post-hoc test was utilized to discriminate where significant differences occurred. The level of significance was set at P < 0.05. Statistical analyses were performed by utilizing a commercially available software package (Instat, Graph Pad Software, San Diego, CA, USA).
RESULTS

Duration of IE was 26 ± 7 min at SL and 20 ± 4 min at ALT.  

\( \bar{P}, \bar{P}(W), P_{\text{max}}^{\text{b}}(W), \text{and } P_{\text{max}}^{\text{b}}(W) \text{ obtained at SL and ALT are presented in Table 1. } \bar{P} \text{ during 10 s 'all out' was not different at ALT and SL, whereas } \bar{P} \text{ during 30 s 'all out' was slightly (although not significantly) lower at ALT than at SL. } \bar{P}(W), P_{\text{max}}^{\text{b}}(W), \text{and } P_{\text{max}}^{\text{b}}(W) \text{ during IE were not different between SL and ALT. } P_{\text{max}}^{\text{b}}(W) \text{ and } P_{\text{max}}^{\text{b}}(W) \text{ during 10 s 'all out' and during 30 s 'all out' were significantly higher than, respectively, } P_{\text{max}}^{\text{b}}(W) \text{ and } P_{\text{max}}^{\text{b}}(W) \text{ during IE.}

Resting [La]b-values were not significantly different between SL (1.5 ± 0.5 mmol l\(^{-1}\)) and ALT (1.6 ± 0.5). [La]b peak after 10 s 'all out', 30 s 'all out' and IE, at SL and ALT, are given in Figure 1. Both at SL and at ALT [La]b peak occurred on the average at about 6 min into recovery, independently on exercise protocols. At SL, [La]b peak was significantly lower after 10 s 'all out' than after 30 s 'all out' or IE; no significant difference was observed between [La]b peak after 30 s 'all out' and after IE. At ALT [La]b peak was significantly lower after 10 s 'all out' than after 30 s 'all out', as well as after IE than after 30 s 'all out'; no significant difference was observed between [La]b peak after 10 s 'all out' and after IE. [La]b peak after 10 s 'all out' was not significantly different between SL and ALT, whereas after 30 s 'all out' and IE [La]b peak was significantly lower at ALT as compared with SL. The percentage decrease in [La]b peak at ALT compared with SL, was ~18% for 30 s 'all out' and ~41% for IE.

DISCUSSION

Maximal mechanical power output

About 3 weeks of exposure to 5050 m (ALT) did not affect the average mechanical power output (\( \bar{P} \)) (di

\begin{table}[h]
\centering
\begin{tabular}{lcccccc}
\hline
 & 10 s & & & 30 s & \multicolumn{2}{c}{IE} \\
\hline
 & SL & ALT & ALT/SL & SL & ALT & ALT/SL \\
\hline
\( \bar{P} (W) \) & \( \bar{x} \) & 665 & 642 & 0.97 & 566\( ^{\dagger} \) & 515\( ^{\dagger} \) & 0.91 \\
SD & 102 & 76 & & 83 & 45 & & \\
\hline
\( P_{\text{max}} (W) \) & \( \bar{x} \) & 230\( ^{\dagger\dagger} \) & 164\( ^{\dagger\dagger} \) & 0.71 \\
SD & 57 & 51 & & & & & \\
\hline
\( P_{\text{max}}^{\text{b}}(W) \text{ kg}^{-1} \) & \( \bar{x} \) & 8.65 & 9.07 & 1.05 & 7.41\( ^{\dagger} \) & 7.25\( ^{\dagger} \) & 0.98 \\
SD & 0.89 & 1.03 & & 1.06 & 0.75 & & \\
\hline
\hline
\end{tabular}
\caption{Average power output (\( \bar{P} \)) during 10 s and 30 s 'all out' cycle exercise, and maximal power output (\( P_{\text{max}} \)) during incremental cycle exercise (IE) leading to exhaustion in ~20–25 min, at sea level (SL) and after ~3 weeks at 5050 m (ALT). \( \bar{P} \) and \( P_{\text{max}} \) values are expressed per unit of body mass (\( m_{b} \)) (i.e. as \( P_{\text{max}}^{\text{b}}(W) \) and \( P_{\text{max}}^{\text{b}}(W) \)). Data are presented as average values \( (\bar{x}) \) ± standard deviation (SD). ALT/SL indicates the ratio between the values at ALT and at SL. See text for further details.}
\end{table}

Prampero & Mognoni 1981) during 10 s 'all out', exercise, and determined a slight (statistically not significant) decrease of \( \bar{P} \) during 30 s 'all out' exercise. On the other hand, the maximal mechanical power output (\( P_{\text{max}} \)) during an IE leading to exhaustion in ~20–25 min, was significantly (about 30%) lower at ALT than at SL before the expedition. Chronic hypoxic exposure appears inevitably associated with a loss of \( m_{b} \), whose causes are not completely understood (see, e.g. Cerretelli & Hoppeler 1996). In a previous study conducted by our group on subjects staying for ~4 weeks in the same altitude laboratory of the present study (Kayser et al. 1993b), the loss of \( m_{b} \) at ALT was very similar, when expressed as a percentage of the values obtained at SL, to that observed for the muscle volume (estimated from anthropometric measurements) of the lower limbs. Lower limbs muscle volume is one of the main

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determinants of power output during cycle exercise. Thus, in order to correct the influence of muscle mass loss on the observed $\dot{P}$ and $P_{\text{max}}$, decreases at ALT, these variables were also expressed per unit of $m_b$ (i.e. as $\dot{P} \frac{m_b}{s}$ and $P_{\text{max}} \frac{m_b}{s}$). $\dot{P} \frac{m_b}{s}$ after 10 and 30 s ‘all out’ exercise were not different between ALT at SL, whereas $P_{\text{max}} \frac{m_b}{s}$ was still significantly lower at ALT than at SL. Thus, $\sim$3 weeks at ALT did not significantly affect the Caucasian lowlanders who did not present significant changes, compared with SL, in their ‘average’ maximal mechanical power output (di Prampero & Mognoni 1981) determined during 10 and 30 s ‘all out’ cycling exercise. The slight decrease of $\dot{P}$ after 30 s ‘all out’ exercise was likely the result of the reduced muscle mass. On the other hand, $P_{\text{max}}$ and $P_{\text{max}} \frac{m_b}{s}$ during IE were significantly affected at ALT. These results suggest that the capacity of skeletal muscles to generate power during short (10–30 s) supramaximal cycling exercise, in which the contribution of oxidative metabolism to ATP resynthesis is either negligible (10 s) or relatively low (30 s) (see, e.g. Bar-Or 1987), was unaffected by chronic high altitude exposure. By contrast, for exercise of longer duration, in which aerobic processes are predominant, maximal power was significantly reduced in chronic hypoxia.

The unchanged $\dot{P}$ and $P_{\text{max}} \frac{m_b}{s}$ at ALT observed in the present study during 10 and 30 s ‘all out’ exercises should be interpreted in conjunction with previous work conducted in the same ALT laboratory, which showed that the maximal height of a jump off both feet on a jumping platform (Kayser et al. 1993b), considered an index of the ‘instantaneous’ or ‘peak’ anaerobic alactic power (di Prampero & Mognoni 1981), as well as the maximal voluntary isometric contraction of the elbow flexors (Kayser et al. 1993b) are not affected by chronic exposure to 5050 m. Thus, the results of the present study and of that mentioned above (Kayser et al. 1993b) indicate that the capacity of muscle to generate maximum force and power (for duration ranging from a fraction of a second to 10–30 s) is not affected by chronic hypoxia, at least for $\sim$1 month sojourns at 5050 m. The results of the present study are also in agreement with data obtained by di Prampero et al. (1982), who observed no decreases (vs. normoxia or SL) in the ‘average’ maximal mechanical power output during 10 s ‘all out’ cycling exercise carried out in acute and chronic hypoxia (several weeks at 4520 m), and by McLellan et al. (1990), who observed no changes (vs. normoxia) in ‘average’ maximal mechanical power output during 30 s ‘Wingate’ tests carried out in acute hypoxia.

**Peak blood lactate accumulation**

Previous studies observed that peak lactate accumulation in blood ([La]$_b$ peak) after exhausting exercise is reduced in chronic hypoxia compared with acute hypoxia or with normoxia (see, e.g. the reviews by Cerretelli & Hoppeler 1996, Cerretelli et al. 1982, Grassi & Cerretelli 1998, West 1986). Although several hypotheses have been forwarded to explain this finding, the issue does not appear yet settled. A reduced alkali reserve at ALT, attributable to the renal compensation of the respiratory alkalosis, could be responsible for the observed reduced [La]$_b$ peak (Cerretelli et al. 1982, West 1986, Grassi & Cerretelli 1998). Previous experiments performed by our group in the same altitude laboratory of the present study (Kayser et al. 1993a), however, showed that the restoration of a normal alkali reserve by the oral administration of NaHCO$_3$ did not significantly affect [La]$_b$ peak in chronic hypoxia. In the present study [La]$_b$ peak after 10 s ‘all out’ exercise was unaffected by ALT exposure, and [La]$_b$ peak decrease (at ALT vs. SL) after 30 s ‘all out’ was less than half of that observed after IE. The results of the present study confirm previous anecdotal observations by Grassi et al. (1995), and suggest that the reduction of [La]$_b$ peak at ALT is dependent on the duration of the exercise protocol, being more marked for exercise leading to exhaustion in several minutes (see, e.g. Kayser et al. 1993a, Grassi et al. 1996) compared with that observed for shorter supramaximal exercise protocols.

It is well known that blood lactate accumulation during exercise cannot be considered a direct index of lactate production by muscles, because muscles, as well as other tissues and organs, are also consumers of lactate by oxidative metabolism. More recently, the ‘lactate shuttle’ hypothesis has been modified to include a new, intracellular component (Brooks 2000) to the previously demonstrated cell-to-cell and organ-to-organ components (Brooks 1991). In addition, lactate distribution throughout body compartments appears to be regulated by complex mechanisms (see, e.g. reviews by Gladden 1996, Juel 1997). More specifically, lactate transport through the sarcolemma seems to be mainly mediated by a proton-linked monocarboxylate transporter (MCT). Several different MCT isoforms with different functional characteristics have been identified (see, e.g. Halestrap & Price 1999). Undissociated lactic acid passive diffusion represents a smaller portion of lactate exchange through the sarcolemma (Juel 1997). To our knowledge, no studies have investigated the effects of chronic hypoxia on MCT isoforms. If changes in the expression or in the activity of these proteins occur in hypoxia, they would of course affect lactate influx into and efflux out of the blood. Indirect evidence, however, argues against the notion that the ‘lactate paradox’ is attributable to differences in lactate exchange across membranes, in chronic hypoxia vs. normoxia. For example, it was shown (Green et al. 1989, Young et al. 1984) that both muscle and blood lactate concentrations are reduced in chronic hypoxia. Previous work from our
group observed a lower intramuscular hydrogen ion concentration during exhausting exercise in chronic hypoxia, compared with normoxia (Kayser et al. 1993a). Blood lactate kinetics during the recovery after exhausting exercise were found to be unchanged in chronic hypoxia compared with normoxia (Grassi et al. 1995), suggesting that lactate exchange between muscle, blood and other tissues was substantially unaffected by chronic hypoxia. It must also be pointed out that after 3 weeks at 5050 m subjects are in a condition of partially compensated respiratory alkalosis (Kayser et al. 1993a, Grassi et al. 1996), which is compatible with a facilitated active transport and passive diffusion of lactate and H⁺ from muscle to blood.

Thus, the unchanged [La]b peak after 10 s ‘all out’ exercise, and the less marked decrease after 30 s ‘all out’ exercises, make it tempting to hypothesize that skeletal muscle lactate production during supramaximal exercise may not be affected by chronic hypoxia. The decrease in [La]b peak observed at ALT vs. SL after more prolonged exhausting exercise might then be attributable to hypoxia-induced modifications of the complex interactions between lactate production by muscles and lactate uptake by muscles and other organs, possibly mediated by β-adrenergic control of glycogenolysis and glucose–lactate metabolism (Reeves et al. 1992). These factors, on the other hand, might not be relevant in determining blood lactate levels during short supramaximal exercises. Another possibility is that during heavy prolonged exercise in chronic hypoxia, respiratory muscle fatigue may develop (Cibella et al. 1996), which may limit Pmax and, as a consequence, [La]b peak. Another study by Cibella et al. (1999), showing that during prolonged exercise at 5050 m the mechanical power sustained by respiratory muscles may substantially limit Pmax which would be in agreement with this hypothesis. A further possibility is that during a long exercise protocol at ALT, Pmax and therefore [La]b peak, may be reduced because of a decreased central drive to locomotory muscles (central fatigue’, see, e.g. Bigland-Ritchie & Vollestad 1988), aimed at preserving O₂ for the needs of vital organs. This hypothesis would be substantiated by the observation that at 5050 m, during cycloergometric exercise leading to exhaustion in ~5 min, the quadriceps muscles did not show signs of fatigue detectable by electromyography (Kayser et al. 1994), suggesting that fatigue in these conditions was not ‘peripheral’. In short, several factors listed above could be responsible for the reduced [La]b peak observed, at ALT vs. SL, after relatively long exhausting exercise. On the other hand, during short supramaximal exercise the capacity of skeletal muscle to generate mechanical power (as discussed above), and also the contribution of anaerobic glycolysis to the overall energy expenditure may not be impaired in chronic hypoxia. This would also be compatible with biopsy data (see, e.g. the review by Cerretelli & Hoppeler 1996), which demonstrate that the activities of the main regulatory enzymes of the glycolytic pathway are not affected by chronic hypoxic exposure.

**Limitations of the study**

In the present study [La]b peak after 10 s ‘all out’ was not different at ALT vs. SL, in the presence of an unchanged P. The lower [La]b peak at ALT after 30 s ‘all out’, on the other hand, was associated with a (statistically not significant) tendency towards lower P. It is difficult to make cause–effect relationships between power output and blood lactate accumulation. In theory, lactate accumulation could be lower as the consequence of a reduced power output, but it could also be that a reduced contribution by ‘anaerobic’ metabolism determines a reduced power output. Our data do not allow discrimination between the two hypotheses.

Another limitation of the present study was that the number of measurements was not enough to allow a discrimination of possible changes of the investigated variables as a function of acclimatization to altitude. Such changes, if present, could also be different between the mountain climbers and the rest of the group.

**CONCLUSIONS**

‘Average’ mechanical power output generated during 10 and 30 s ‘all out’ bicycle exercises was substantially not different at ALT vs. SL, [La]b peak after 10 s ‘all out’ exercise was not different between ALT and SL, whereas after 30 s ‘all out’ exercise the decrease in [La]b peak at ALT was less than half of that described for IE leading to exhaustion in several minutes. The results of the present study confirm previous preliminary observations by Grassi et al. (1995), and demonstrate that the ALT related reduction of [La]b peak may be dependent on the duration of the exercise protocol, being more marked for exercise leading to voluntary exhaustion in several minutes (see, e.g. Kayser et al. 1993a, Grassi et al. 1996) than during short supramaximal exercise. Muscle power output and blood lactate accumulation during short supramaximal exercise are substantially unaffected by chronic hypoxia.

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