Chapter 5

Is gross efficiency lower at acute simulated altitude than at sea level?

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Abstract

The purpose of this study was to test the assumption that gross efficiency (GE) at sea level is representative of GE at altitude. It was hypothesized that an increased cost of ventilation and heart rate, combined with a higher respiratory exchange ratio (RER), at altitude might result in a decrease in GE. Trained men (n = 16) completed two maximal incremental exercise tests and two GE tests, one at sea level and one at an acute simulated altitude of 1,500 m (hypobaric chamber). GE was determined between min 3:00-6:00 during submaximal exercise at 45%, 55%, and 65% of the altitude specific power output attained at the maximal oxygen uptake ($\dot{V}O_{2\text{max}}$). GE determined at the highest submaximal exercise intensity with a mean RER ≤ 1.0, matched for both conditions, was significantly lower at altitude (20.7 ± 1.1% vs. 21.4 ± 0.8%, $t = 2.9$, $p < 0.05$). In conclusion, these results demonstrate that moderate altitude resulted in a significantly lower GE during cycling exercise than sea level. However, it might be that the lower GE at altitude is caused by the lower absolute exercise intensity.
Introduction

Momentary performance (e.g. velocity) depends on the dynamic balance between power production and power dissipation.¹⁻³ The power production side can be determined as the product of total metabolic energy production (aerobic and anaerobic energy production) and the efficiency of the transfer of energy production to forward propulsion.

The most commonly used definition of efficiency is gross efficiency (GE),³,⁴ the ratio between mechanical power output (PO) and metabolic power input (PI), on which the current version of the energy flow model for cycling¹ and speed skating², used to simulate athletic performance, is based. One of the assumptions underlying the energy flow model is that GE determined at sea level is representative of GE at low to moderate altitude. However, it can be hypothesized that an increased cost of ventilation (VE)⁵⁻⁷ and heart rate (HR),⁶,⁷ in combination with a higher respiratory exchange ratio (RER),⁶⁻⁸ at altitude might potentially result in a decrease in GE.

Multiple studies⁵⁻¹⁰ have investigated the cardiorespiratory response during submaximal and maximal exercise in acute hypoxia. The overall conclusion is that hypoxia does not affect oxygen uptake (VO₂) during exercise at the same absolute submaximal exercise intensity. However, because GE is dependent not solely on VO₂, but also on substrate metabolism (reflected by RER), we cannot be sure that submaximal GE is the same at sea level and altitude. Clark et al.⁵ studied the effect of acute simulated moderate altitude on GE during cycling exercise. Although, VO₂ at the same absolute submaximal exercise intensities (50-250 W) was independent of altitude, GE decreased from a mean value (determined at 50 W, 100 W, 150 W, 200 W, and 250 W) of 17.3 ± 2.4% at 200 m and 1200 m to 16.8 ± 2.2% at 3200 m. However, cadence significantly increased at altitude, which possibly affected the efficiency measurements.⁴ Schuler et al.¹¹ determined GE during cycling exercise at the same absolute exercise intensity at sea level and after one day of altitude exposure. In this study GE was unaffected by altitude exposure, but GE values are extremely high (25.3 ± 0.9% at sea level and 25.2 ± 1.0% after 1 day at 2,340 m).¹¹ Although the subjects in this study were elite cyclists, cycling GE values this high have been debated.¹² Accordingly, no definitive conclusion about the effect of hypoxia on GE can be given. In addition, athletic events are mainly performed at a certain fraction of VO₂max, and thus determining GE at the same relative exercise intensity is of potential interest. As most sporting events are performed at low to moderate altitude (2002 winter Olympics in Salt Lake City at 1,250–2,003 m, 2006 Olympic ski races during the winter Olympics in Torino were held at 1,509 m and 2,800 m, 2012 World Championship speed skating (sprint) Calgary at 1,034 m) it was the purpose of this
study to evaluate the effect of acute hypoxia (1,500 m) on GE determined during submaximal cycling exercise at the same relative exercise intensity.

Methods

Subjects
Sixteen trained non-altitude-acclimatized male cyclists (age 28.4 ± 6.7 years, height 187 ± 7.6 cm, body mass 78.2 ± 6.8 kg, training status 9.6 ± 4.6 h·week⁻¹) participated in this study. The inclusion criterion for participation was a maximal oxygen uptake (\( \dot{V}O_{2\text{max}} \)) > 55 mL·kg⁻¹·min⁻¹. Subjects were asked to avoid strenuous exercise and alcohol in the 24 h before the test. Subjects were requested to avoid caffeine and to consume their last meal at least 3 h prior to the experiments. The goal and the experimental protocol were explained before the first test and subjects provided written informed consent. The local ethics committee approved the protocol.

Experimental design
Subjects were tested on four different occasions within four weeks, with at least 36 h of rest between tests. Two maximal incremental exercise tests and two GE tests were performed, one at sea level and one at a simulated moderate altitude of 1,500 m (hypobaric hypoxia). For logistic reasons, all subjects started with a maximal incremental exercise test and GE test at sea level, followed by both tests at simulated altitude. Tests at simulated altitude were performed in a hypobaric chamber (12 m in length and 3 m in diameter) at the Center for Man in Aviation (Soesterberg, The Netherlands).

Exercise was performed on an electronically braked cycle ergometer (Excalibur Sport, Lode Medical Technology, Groningen, The Netherlands). Subjects maintained a fixed pedaling frequency of 80 rpm. Saddle height and handlebars position were adjusted to individual preferences and were kept constant between tests. Subjects used their own clipless pedals.

Expired air was analyzed breath-by-breath using open circuit spirometry (Cosmed Quark b², Cosmed S. R. L., Rome, Italy). Before the start of each test the gas analyzer was calibrated with two known reference gas mixtures (room air and 16% O₂, 5% CO₂) and the volume transducer was calibrated using a 3 L syringe (Cosmed S. R. L., Rome, Italy). The time to ascend to a simulated altitude of 1,500 m and calibrate the Cosmed was ~10 min. During this period subjects sat on the cycle ergometer. HR was measured with radiotelemetry (Polar Electro OY, Kempele, Finland). One minute after completion of the incremental exercise tests, blood lactate concentration ([La]) was measured in capillary
blood obtained from a fingertip (Lactate Pro, Arkay Inc, Kyoto, Japan). Arterial oxyhemoglobin saturation (SpO₂) was measured continuously during the GE tests using a pulse oximeter (Avant 9600, Nonin Medical, Plymouth, U.S.A.), the average value during the last min of each stage of the GE test was recorded. Room temperature and relative humidity were consistent (22.6 ± 3.5 °C; 55.9 ± 7.1%).

**Maximal incremental exercise test**

The maximal incremental exercise tests were conducted to determine \( \dot{V}O_{2\text{max}} \) and the power output attained at \( \dot{V}O_{2\text{max}} \) (\( \text{PV}\dot{V}O_{2\text{max}} \)) at both sea level and altitude. The maximal incremental exercise tests started with a warm-up of 3 min at 100 W, after which exercise intensity was increased by 25 W·min\(^{-1}\). Tests were ended when pedaling frequency dropped below 70 rpm. \( \dot{V}O_{2\text{max}} \) was determined as the highest value of 30 s averages.

**Gross efficiency test**

The GE test at sea level and altitude started with 2 min at 25 W, after which exercise intensity increased to 45% of the altitude specific \( \text{PV}\dot{V}O_{2\text{max}} \) for 6 min, followed by 6 min at 55% \( \text{PV}\dot{V}O_{2\text{max}} \), and 6 min at 65% \( \text{PV}\dot{V}O_{2\text{max}} \). GE increases with exercise intensity until ~50% peak PO is reached (see Chapter 2).\(^{14}\) By determining efficiency at 45% \( \text{PV}\dot{V}O_{2\text{max}} \), 55% \( \text{PV}\dot{V}O_{2\text{max}} \), and 65% \( \text{PV}\dot{V}O_{2\text{max}} \) each individuals’ highest GE was calculated (see Chapter 3).\(^{15}\) Subjects were instructed to maintain a pedaling frequency of 80 rpm. During the final min of each stage subjects rated their overall feeling of perceived exertion (RPE) on the Category-Ratio Rating of Perceived Exertion scale.\(^{16}\) Expired air (\( \dot{V}O_2 \), carbon dioxide output (\( \dot{V}CO_2 \), \( \dot{V}E \), respiratory frequency (\( R_f \)), and RER) was analyzed continuously during the entire GE test, as was HR. GE was determined using Equation 5.1.

\[
GE = \frac{pO}{pI} \cdot 100
\]

Equation 5.1

The metabolic PI can be calculated by multiplying \( \dot{V}O_2 \) (L·s\(^{-1}\)) with the oxygen equivalent, as suggested by Garby and Astrup.\(^{17}\) Mean \( \dot{V}O_2 \) and RER values were determined during the 3:00-6:00 interval of each 6 min exercise intensity step. The criteria for determining GE were a steady state \( \dot{V}O_2 \) (< 5% difference between the mean \( \dot{V}O_2 \) of min 3:00-4:00 and min 5:00-6:00 expressed relatively to the mean \( \dot{V}O_2 \) over the corresponding 3 min) and RER \( \leq \) 1.0.
Statistics

Data are presented as means ± SD. Visual inspection of Q-Q plots was used to check if the differences were normally distributed (SPSS 17.0, SPSS Inc., Chicago, IL, USA). Differences in \(\dot{V}O_{2\text{max}}, \dot{PVO}_{2\text{max}},\) maximal HR (HR\(_{\text{max}}\)), and maximal \([La]([La]_{\text{max}})\) between sea level and altitude were analyzed using a paired samples t-test. The effect of altitude on \(\dot{V}O_2, \dot{V}CO_2, \text{RER, } \dot{V}E, R_i, \text{ GE, SpO}_2, \text{ HR, and RPE} \) was evaluated by using a repeated measures ANOVA, with two within factors (exercise intensity (45% \(\dot{PVO}_{2\text{max}}, \) 55% \(\dot{PVO}_{2\text{max}}, \) and 65% \(\dot{PVO}_{2\text{max}}\)) and condition (sea level and altitude)). The assumption of sphericity was tested using Mauchly’s test. When the assumption of sphericity was met and significant main effects were found, post-hoc contrasts were performed. Violation of the assumption of sphericity resulted in all cases in a \(\varepsilon < 0.75\), therefore the Greenhouse Geisser correction was applied to the degrees of freedom,\(^\text{18}\) and post-hoc comparisons were tested using the Bonferroni method. The difference in GE, determined at the highest altitude-matched % \(\dot{PVO}_{2\text{max}}\) that resulted in a mean RER \(\leq 1.0\), between sea level and altitude was tested using a paired samples t-test. Differences were considered to be significant if \(p < 0.05\).

Results

Maximal incremental exercise test

\(\dot{V}O_{2\text{max}}, \dot{PVO}_{2\text{max}}\) and HR\(_{\text{max}}\) were all significantly lower at altitude than at sea level (59.0 ± 6.2 mL·kg\(^{-1}\)·min\(^{-1}\) vs. 63.8 ± 5.2 mL·kg\(^{-1}\)·min\(^{-1}\), \(t = 4.49, p < 0.001\); 400 ± 30 W vs. 430 ± 36 W, \(t = 4.84, p < 0.001\); 185 ± 8.5 beats·min\(^{-1}\) vs. 193 ± 9.8 beats·min\(^{-1}\), \(t = 4.98, p < 0.001\)). These absolute decreases correspond to a mean relative decrease of 7.5 ± 6.8% in \(\dot{V}O_{2\text{max}}, \) 6.7 ± 5.5% in \(\dot{PVO}_{2\text{max}}, \) and 4.5 ± 3.5% in HR\(_{\text{max}}\). No significant difference between sea level and altitude was seen in \([La]_{\text{max}}\) (16.0 ± 2.8 mmol·L\(^{-1}\) vs. 15.4 ± 2.8 mmol·L\(^{-1}\), \(t = 0.68, p = 0.51\)).

Gross efficiency test

Results of the repeated measures ANOVA (i.e. \(F\) values with corresponding degrees of freedom) are reported in Table 5.1. As intended, there was a significant main effect of exercise intensity on \(\dot{V}O_2 (p < 0.001), \dot{V}CO_2 (p < 0.001), \text{RER} (p < 0.001), \dot{V}E (p < 0.001), R_i (p < 0.001), \text{GE} (p < 0.01), \text{SpO}_2 (p < 0.001), \text{HR} (p < 0.001), \) and RPE (\(p < 0.001\)) (Figure 5.1and 5.2). Post-hoc tests showed that for all variables except GE, all combinations were significantly different (\(p < 0.001\)). Only the differences in GE between 45% \(\dot{PVO}_{2\text{max}}\) and 55% \(\dot{PVO}_{2\text{max}}\) (\(p < 0.01\)) and between 45% \(\dot{PVO}_{2\text{max}}\) and 65%
P\(\tilde{V}\)O\(_{2}\)\(_{\text{max}}\) \((p < 0.05)\) were significantly different (Figure 5.1F). A significant effect of condition \((p < 0.05)\) and a significant interaction between exercise intensity and condition was found for \(\tilde{V}\)\(\tilde{O}\)\(_2\) \((p < 0.05; \text{Figure 5.1A})\). Two interaction terms were significant, the contrast that compared \(\tilde{V}\)\(\tilde{O}\)\(_2\) at 45\% P\(\tilde{V}\)O\(_{2}\)\(_{\text{max}}\) and 65\% P\(\tilde{V}\)O\(_{2}\)\(_{\text{max}}\) between sea level and altitude \((p < 0.01)\) and the contrast that compared \(\tilde{V}\)\(\tilde{O}\)\(_2\) at 55\% P\(\tilde{V}\)O\(_{2}\)\(_{\text{max}}\) and 65\% P\(\tilde{V}\)O\(_{2}\)\(_{\text{max}}\) between sea level and altitude \((p < 0.001)\). A significant main effect for condition was found on RER \((p < 0.01; \text{Figure 5.1C})\) and Sp\(\text{O}_2\) \((p < 0.001; \text{Figure 5.2A})\). RER was significantly lower at sea level, and Sp\(\text{O}_2\) was significantly higher at sea level. A significant interaction was found for Sp\(\text{O}_2\) \((p < 0.001)\). All differences between exercise intensities were significantly different between sea level and altitude \((p < 0.05)\); the drop in Sp\(\text{O}_2\) with increasing exercise intensity is larger at altitude than at sea level. No significant main effect of condition was found on \(\tilde{V}\)\(\text{CO}_2\) \((p = 0.54)\), \(\tilde{V}\)\(\text{E}\) \((p = 0.28)\), R\(_f\) \((p = 0.91)\), GE \((p = 0.51)\), HR \((p = 0.56)\), and RPE \((p = 0.44)\).

When GE, determined at the highest altitude-matched individual \% P\(\tilde{V}\)O\(_{2}\)\(_{\text{max}}\) with a mean RER just below 1.0, was compared between sea level \((21.4 \pm 0.8\%)\) and altitude \((20.7 \pm 1.1\%)\), a significant difference was found \((t = 2.87, p < 0.05)\).
### Table 5.1 Results of the repeated measures ANOVA for respiratory variables, peripheral oxygen saturation, heart rate, and rating of perceived exertion.

<table>
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<th>Variable</th>
<th>Main effect intensity</th>
<th>Main effect condition</th>
<th>Interaction effect</th>
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<tr>
<td>VO₂</td>
<td>F = 632</td>
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<td>F = 9.6</td>
<td>F = 0.20</td>
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<td></td>
<td>p &lt; 0.001</td>
<td>p &lt; 0.01</td>
<td>p = 0.82</td>
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<tr>
<td>VE</td>
<td>F = 1047</td>
<td>F = 1.27</td>
<td>F = 2.1</td>
</tr>
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<td></td>
<td>p &lt; 0.001</td>
<td>p = 0.28</td>
<td>p = 0.14</td>
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<tr>
<td>Rf</td>
<td>F = 70.1</td>
<td>F = 0.012</td>
<td>F = 0.49</td>
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<td></td>
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<td>p = 0.56</td>
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<tr>
<td>GE</td>
<td>F = 7.74</td>
<td>F = 0.49</td>
<td>F = 0.66</td>
</tr>
<tr>
<td></td>
<td>p &lt; 0.01</td>
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<td>p = 0.54</td>
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<tr>
<td>SpO₂</td>
<td>F = 42.6</td>
<td>F = 34.1</td>
<td>F = 12.6</td>
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<td>p &lt; 0.001</td>
<td>p &lt; 0.001</td>
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<tr>
<td>HR</td>
<td>F = 281</td>
<td>F = 0.37</td>
<td>F = 1.1</td>
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<td></td>
<td>p &lt; 0.001</td>
<td>p = 0.56</td>
<td>p = 0.34</td>
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<tr>
<td>RPE</td>
<td>F = 155</td>
<td>F = 0.63</td>
<td>F = 2.2</td>
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<tr>
<td></td>
<td>p &lt; 0.001</td>
<td>p = 0.44</td>
<td>p = 0.15</td>
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</table>

VO₂, oxygen uptake; VCO₂, carbon dioxide output; RER, respiratory exchange ratio; VE, minute ventilation; Rf, respiratory frequency; GE, gross efficiency; SpO₂, arterial oxyhemoglobin saturation; HR, heart rate; RPE, rating of perceived exertion.
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Figure 5.1 A-C Respiratory data (mean values ± standard deviations) measured during submaximal exercise, at the altitude specific 45%, 55%, and 65% of the power output attained at $\dot{V}O_{2\text{max}}$ ($P\dot{V}O_{2\text{max}}$). A: oxygen uptake ($\dot{V}O_2$) B: carbon dioxide output ($\dot{V}CO_2$) C: respiratory exchange ratio (RER). The filled circles represent the sea level data and the open circles represent the altitude data. 1,2,3Significantly different from 45% $P\dot{V}O_{2\text{max}}$, 55% $P\dot{V}O_{2\text{max}}$, or 65% $P\dot{V}O_{2\text{max}}$, respectively. *Significantly different between sea level and low altitude. **Significant interaction effect.
Figure 5.1 D-F Respiratory data (mean values ± standard deviations) measured during submaximal exercise, at the altitude specific 45%, 55%, and 65% of the power output attained at \( \dot{V}O_{2\text{max}} \) (\( P\dot{V}O_{2\text{max}} \)). D: minute ventilation (\( \dot{V}E \)) E: respiratory frequency (\( R_f \)) F: gross efficiency (GE). The filled circles represent the sea level data and the open circles represent the altitude data. \(^{1,2,3}\)Significantly different from 45% \( P\dot{V}O_{2\text{max}} \), 55% \( P\dot{V}O_{2\text{max}} \), or 65% \( P\dot{V}O_{2\text{max}} \), respectively. \(*\)Significantly different between sea level and low altitude. \( **\)Significant interaction effect.
Figure 5.2 Arterial oxyhemoglobin saturation (SpO₂; A), heart rate (HR; B), and rating of perceived exertion (RPE; C) data measured during submaximal exercise, at the altitude specific 45%, 55%, and 65% of the power output attained at $\dot{V}O_{2\text{max}}$ ($\dot{V}O_{2\text{max}}$). The presented data are mean values ± standard deviations. The filled circles represent the sea level data and the open circles represent the altitude data. $^1,^2,^3$Significantly different from 45% PVO$_{2\text{max}}$, 55% PVO$_{2\text{max}}$, or 65% PVO$_{2\text{max}}$, respectively. *Significantly different between sea level and low altitude.


Discussion

The purpose of this study was to evaluate the effect of acute hypoxia (1,500 m) on GE, determined during submaximal cycling exercise at the same relative exercise intensity. The main finding was that GE, measured at the highest possible individual relative exercise intensity (matched at sea level and altitude), was significantly lower at altitude than at sea level.

As GE increases with increasing exercise intensity,4,14 GE was determined during submaximal exercise at 45% \( \dot{V}O_{2\text{max}} \), 55% \( \dot{V}O_{2\text{max}} \), and 65% \( \dot{V}O_{2\text{max}} \),15 and the highest valid GE was selected, as GE reaches a stable value at ~50% peak PO (see Chapter 2).14 Our finding of a lower GE at altitude is in contrast with the findings of Schuler et al.,11 who found a similar GE at sea level and after 1 day of altitude exposure. The difference between the present study and the studies of Clark et al.5 and Schuler et al.11 is that GE in the current study was determined at the same relative exercise intensity instead of at the same absolute intensity. As even moderate altitude results in a significant reduction in \( \dot{V}O_{2\text{max}} \) and \( \dot{V}O_{2\text{max}} \), the same absolute exercise intensity would result in an increased relative exercise intensity and therefore a larger homeostatic disturbance. The current study is the first one in which the effect of hypoxia on GE at the same relative submaximal exercise intensity was evaluated, it is therefore hard to compare the current results with the results with former studies. In Figure 5.3, the mean GE data are plotted against absolute PO; this figure suggests that GE is indeed lower at altitude than at sea level. However, we cannot rule out the possibility that GE at altitude is lower because of the lower final absolute PO.

If we assume that the GE is indeed lower under hypoxic conditions, a possible cause could be the significantly higher RER, observed at matched relative workloads, at altitude. A higher RER results in a higher oxygen equivalent, as carbohydrates yield more energy per liter of oxygen than fats,19 and thus in a higher metabolic PI, which decreases GE. Lundby and van Hall20 studied the effect of hypoxia (4,100 m) on substrate utilization at the same absolute and relative exercise intensity. No significant difference in RER between 60 min cycling at 50% \( \dot{V}O_{2\text{max}} \) at sea level and at a simulated altitude of 4,100 m (altitude specific \( \dot{V}O_{2\text{max}} \)) was found. Studying RER at the same absolute exercise intensity resulted in the finding that the 60 min mean RER increased from 0.91 ± 0.01 at sea level to 0.95 ± 0.02 at 4,100 m. Although Lundby and van Hall20 did not find a significant difference in mean RER at the same relative exercise intensity, RER was significantly higher after 15 min of submaximal exercise at the same relative exercise intensity under hypoxic conditions; this difference disappeared during the remaining 45
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min of exercise. Thus, the difference between the results of Lundby and van Hall\textsuperscript{20} and the current results can possibly be explained by the longer exercise duration in the study of Lundby and van Hall\textsuperscript{20}. Friedmann et al.\textsuperscript{21} and Katayama et al.\textsuperscript{8} let their subjects exercise (1 h and 30 min, respectively) at the same relative exercise intensity and found a significantly higher RER under hypoxic conditions (simulated altitude of 2500 and 2000 m, respectively), which supports the findings of the current study. An increase in RER of 0.03 with altitude, which was found in the current study, will only result in a decrease of about 0.2% in GE (e.g. 21.4% to 21.2%), so there must be another physiologic change that causes the decrease in GE, as the mean absolute decrease in GE with acute simulated altitude was 0.77 ± 1.1%.

The current results showed that GE, measured at the highest possible individual relative exercise intensity, was lower at acute simulated altitude than at sea level. However, there is a need for follow-up research, in which GE measurements are performed at both absolute and relative submaximal exercise intensities at sea level and altitude, to make definitive conclusions about the effect of acute and chronic hypoxia on GE, to gain insight into the underlying mechanisms, and to evaluate the effect on performance.
References


