Impaired interval exercise responses in elite female cyclists at moderate simulated altitude

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Impaired interval exercise responses in elite female cyclists at moderate simulated altitude. J Appl Physiol 89: 1819–1824, 2000.—The effect of hypoxia on the response to interval exercise was determined in eight elite female cyclists during two interval sessions: a sustained 3 × 10-min endurance set (5-min recovery) and a repeat sprint session comprising three sets of 6 × 15-s sprints (work-to-relief ratios were 1:3, 1:2, and 1:1 for the 1st, 2nd, and 3rd sets, respectively, with 3 min between each set). During exercise, cyclists selected their maximum power output and breathed either atmospheric air (normoxia, 20.93% O₂) or a hypoxic gas mix (hypoxia, 17.42% O₂). Power output was lower in hypoxia vs. normoxia throughout the endurance set (244 ± 18 vs. 226 ± 17, 234 ± 18 vs. 221 ± 25, and 235 ± 18 vs. 221 ± 25 W for 1st, 2nd, and 3rd sets, respectively; P < 0.05) but was lower only in the latter stages of the second and third sets of the sprints (452 ± 56 vs. 429 ± 49 and 403 ± 54 vs. 373 ± 43 W, respectively; P < 0.05). Hypoxia lowered blood O₂ saturation during the endurance set (92.9 ± 2.9 vs. 95.4 ± 1.5%; P < 0.05) but not during repeat sprints. We conclude that, when elite cyclists select their maximum exercise intensity, both sustained (10 min) and short-term (15 s) power are impaired during hypoxia, which simulated moderate (∼2,100 m) altitude.

hypoxia; exercise intensity; lactate; power output; cycling; percentage of arterial oxygen hemoglobin saturation from pulse oximetry

ALTIMITUDE TRAINING IS FREQUENTLY used by competitive athletes in a wide range of sports in the belief that it will improve sea-level performance (13, 14, 18). However, the efficacy of such a training technique is equivocal (1). Training in hypoxic conditions may increase the “stimulus adaptation” and thereby magnify the normal sea-level responses to training (21). Conversely, altitude-induced hypoxia may reduce the intensity at which elite athletes can train, resulting in a relative deconditioning (13, 14). It has been proposed that interval training undertaken at even moderate altitude (∼2,500 m) would result in lower absolute work rates and/or speeds, with lower heart rates (HRs) and blood lactate concentrations ([La]bl) compared with those at sea level (13, 14). Indeed, investigations that compared submaximal exercise of the same relative intensity reported lower HR, a reduced training pace, and higher [La]bl for exercise under hypoxic vs. normoxic conditions (21, 25).

The effects of hypoxia on anaerobic performance have not been well studied, particularly in elite athletes, whose performance responses to even mild hypoxia are impaired to a greater extent than those of less well-trained individuals (10). Such an observation is important as elite athletes seek to maximize their training stimulus (14). As such, measures of submaximal work capacity (21, 25) may not provide valid indicators of the metabolic perturbations induced by extreme, high-intensity exercise under hypoxic conditions.

To the best of our knowledge, only two previous investigations have compared the responses of competitive athletes to self-selected, high-intensity training sessions performed at altitude and sea level (13, 14). Therefore, the aims of the present investigation were to document the effects of reduced inspired Po₂ (simulated moderate altitude) on indicators of exercise intensity and performance during interval exercise sessions that involved all-out repeated efforts of both short (15 s) and medium (10 min) duration in a group of elite female road cyclists. The interval sessions selected were repeated sprint sets and a sustained 3 × 10-min endurance set. These interval exercise sessions are commonly used by cyclists in their preparation for major competitions. On the basis of the results of previous studies (10, 13, 14), we hypothesized that acute exposure to moderate simulated altitude would impair performance of maximal sustained endurance efforts but have less of an impact on repeated sprint interval exercise. Additionally, we expected the HR, blood lactate, and rating of perceived exertion (RPE) at

Received 14 October 1999; accepted in final form 15 June 2000

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the self-selected exercise intensities during interval exercise to be unaffected by reduced inspired PO2.

METHODS

Subjects and preliminary testing. Eight elite female road cyclists [mass, 58.8 ± 3.7 kg; peak O2 uptake (VO2peak), 3.59 ± 0.24 l/min], who were all members of the Australian National team, participated in this study, which was conducted during a 2-wk training camp held at the Australian Institute of Sport, Canberra (altitude 585 m). All testing procedures were fully explained to each cyclist, and their written informed consent was obtained before participation. All testing procedures were approved by the Human Ethics Committee of RMIT University and the Ethics Committee of the Australian Sports Commission. At the start of the training camp, all cyclists performed an incremental test to volitional fatigue on an electromagnetically braked cycle ergometer (Lode, Groningen, The Netherlands) modified with racing handle bars and each rider's own clip-on pedals. VO2peak was defined as the highest O2 uptake (VO2) attained during two consecutive 30-s sampling periods. VO2, CO2 production, minute ventilation (VE), and respiratory exchange ratio were determined every 30 s during the test via an open-circuit indirect calorimetry system (9). Before each maximal test and all subsequently described exercise sessions, the O2 and CO2 analyzers were calibrated by using three alpha-grade gasses (BOC Gases Australia, Canberra, Australia) that spanned the physiological range.

Study design. Cyclists were randomly divided into two groups. All laboratory exercise sessions, which were conducted double-blind, were performed with the subjects breathing either atmospheric air (normoxia, 20.93% O2) or a hypoxic gas mix (hypoxia, 17.42% O2) that simulated an altitude of 2,100 m at the barometric pressure (~710 mmHg) in Canberra. On the first and third testing days (day 1 and day 5), the endurance exercise set was performed, whereas, on the second and fourth testing days (day 3 and day 7), cyclists undertook the repeat sprint set. The endurance set consisted of 3 × 10-min maximum work bouts with 5-min active recovery during which the cyclists pedaled at ~100 W. The repeat sprint set consisted of three sets of 6 × 15-s all-out sprints with a 3-min recovery period between each set. The first set utilized a 1:3 work-to-rest ratio (15-s sprint, 45-s rest), the second a 1:2 work-to-rest ratio, and the final set a 1:1 work-to-rest ratio. For all exercise sessions, cyclists inspired air through a two-way Hans Rudolph valve (model R2700, Hans Rudolph, Kansas City, MO) attached via 2 m of respiratory tubing (50 mm ID, Hans Rudolph) to an ~2,000-liter aluminiun Mylar bag (Scholle Industries, Adelaide, South Australia, Australia). During normoxia trials, the bag contained atmospheric air (PO2 = 20.93% × 710 = 149 Torr), whereas during hypoxia it contained air enriched with nitrogen (PO2 = 0.1700 × 710 = 121 Torr). Air in the respiratory tubing immediately before the R2700 valve was analyzed every 10 min using an Ametek S-3A O2 analyzer (Applied Electrochemistry, Sunnyvale, CA) calibrated with a 16.2% alpha-grade gas (BOC Gases Australia). Cyclists were allowed to remove the mouthpiece after 1 min, during the recovery period, and between work bouts for both interval exercise sessions; this permitted cyclists to drink water and clean out any accumulated saliva. One minute before the start of the repeat sprint sets and 3 min before the endurance set, the cyclists were again required to breathe the designated air mix through the mouthpiece. Because of limitations imposed by the squad’s training commitments, four athletes were tested simultaneously. Accordingly, it was not possible to collect samples of expired air because there was only one indirect calorimetry system. Subjects performed all interval exercise sessions at their maximum self-selected exercise intensity. Both the endurance and sprint interval exercise sessions were selected for this investigation, based on previous data from our laboratory that showed the similarity between the demands of these sessions and actual road cycling competition (D. T. Martin, unpublished observations). Furthermore, the cyclists involved in this study frequently perform such workouts as part of their normal training program (e.g., 1 workout/wk for 3 mo/yr).

Power output, HR, and RPE. Five subjects rode their own bicycles on a stationary air-braked ergometry system (RX-5, Blackburn, Sydney, Australia) during all laboratory interval exercise sessions. For these subjects, power output was measured by using Schoberer Rad Messtechnik (SRM) power cranks (Ingenieuruburo Schoberer, Jülich, Germany), which were calibrated before each test by using a torquemeter (27). Cyclists were not allowed to view their power output, HR, or cadence during any laboratory exercise session. Data from all interval exercise sessions were subsequently downloaded and analyzed by using the SRM proprietary software. Subjects whose bikes were not fitted with the SRM cranks (n = 3) performed all exercise sessions on the Lode ergometer (linear mode), and power output was determined from the computer display. The accuracy of the Lode ergometer was verified by using a torquemeter (27) before all testing. HR was recorded every 5 s via telemetry (Polar Vantage, Polar Electro, Kempele, Finland). Subjects were asked to provide their RPE at the completion of each repetition of the endurance set or each set of sprints, according to the Borg scale (4).

Arterial O2 saturation. Percentage of arterial O2 hemoglobin saturation was monitored during all interval exercise sessions via a fingertip pulse oximeter (%SpO2) (model US-504, Criticare, Waukesha, WI). These monitors have previously been reported to provide valid and reliable measurements of arterial O2 saturation during intense exercise (15). %SpO2 measurements were taken during the last 30 s of each endurance set and during the final 5 s of each of the six sprints of the three repeat sprint sets.

Blood sampling and analyses. Blood (100 μl) was collected from a fingertip into blood-gas collection capillary tubes (Radiometer, Copenhagen, Denmark) after each work bout during the endurance set or after each set of sprints. Analyses for acidity (pH), [La]bl, and HCO3- concentration ([HCO3-]), were conducted on an automated blood-gas analyzer (ABL 700 series, Radiometer Medical).

Statistical analyses. The effects of simulated altitude exposure during exercise on blood pH, [La]bl, [HCO3-], HR, %SpO2, power output (W), and RPE were analyzed by using a 2 (altitude) × 3 (interval sets) repeated-measures ANOVA. A 2 × 3 × 6 (altitude × sets × interval) three-way ANOVA with repeated measures was performed to further evaluate the effects of hypoxia on each of the repeat sprint intervals. Specific mean comparisons of interest were evaluated by using a priori planned contrasts. Statistical significance was accepted when P < 0.05. All values are presented as means ± SD.

RESULTS

Power output, HR, and RPE. All cyclists completed the prescribed interval exercise sessions. Figure 1A displays the mean power output (W) attained during both sessions, whereas Fig. 1B displays the power output for each consecutive work bout during the repeated sprints. The endurance session was performed at ~85% VO2peak. Power output was lower for the endurance work bouts during hypoxia compared with
normoxia (244 ± 18 vs. 226 ± 17 W, decrease of 6.4 ± 5.3%; *P < 0.05, Fig. 1A). Power output was also reduced for the second and third sets of the repeat sprint sets under simulated altitude (452 ± 56 vs. 429 ± 49 and 403 ± 54 vs. 373 ± 43 W, decrease of 5.0 ± 4.0%; *P < 0.05, Fig. 1A) for normoxia and hypoxia, respectively. Power output declined from the first to second and second to third set of the repeat sprints in both hypoxia and normoxia (Fig. 1A). The only significant interaction was set × interval \([F_{(10,70)} = 14.8, P < 0.01]\), and the main effects were significant for altitude \([F_{(1,7)} = 11.9, P = 0.01]\), sets \([F_{(2,14)} = 54.7, P < 0.01]\), and intervals \([F_{(9,35)} = 11.9, P < 0.01]\). Planned contrasts identified that mean power output was significantly reduced during the repeated sprints on the second and fifth interval of the second set and on the second, third, fifth, and sixth interval of the third set (Fig. 1B). During the endurance sets, HR increased from the first to the second (187 ± 11 and 185 ± 12 vs. 191 ± 10 and 189 ± 12 beats/min; *P < 0.05) and from the first to the final work bout (187 ± 11 and 185 ± 12 vs. 192 ± 10 and 193 ± 11 beats/min; *P < 0.05) for normoxia and hypoxia, respectively. However, there were no differences between treatments. RPE was 16 ± 2 vs. 17 ± 2 units after the first endurance work bout and 17 ± 2 vs. 17 ± 3 units after the first repeat sprint set. Thereafter RPE increased over time, so that by the completion of the final set it had reached ~19 ± 1 units for both treatments (Tables 1 and 2).

Arterial O$_2$ saturation. The %SpO$_2$ was lower under hypoxia than normoxia after each of the three endurance work bouts (95.4 ± 1.5 vs. 92.9 ± 2.9, 96.1 ± 1.5 vs. 93.0 ± 2.6, and 96.1 ± 1.5 vs. 94.2 ± 1.0%; *P < 0.05, Fig. 2). In contrast, there was no difference among %SpO$_2$ during the repeated sprints, either over time (between the first and third set) or between treatments.

Blood acidity, bicarbonate, and lactate. Hypoxia resulted in a lower pH and [HCO$_3^-$] after each set of sprints (pH: 7.30 ± 0.06 vs. 7.26 ± 0.05, 7.24 ± 0.05 vs. 7.20 ± 0.04, and 7.20 ± 0.04 vs. 7.15 ± 0.05; [HCO$_3^-$]: 16.0 ± 2.2 vs. 14.4 ± 1.7, 13.7 ± 1.8 vs. 12.2 ± 1.5, and 11.8 ± 1.3 vs. 10.6 ± 1.5 mM, for sets 1, 2, and 3, respectively, normoxia vs. hypoxia; *P < 0.05, Table 2) but not after the endurance sets (Table 1). Both pH and [HCO$_3^-$] declined progressively over time during both interval exercise sets (see Tables 1 and 2 for specific differences). Accordingly, [La]b rose from the first to the last work bout for both endurance and repeat sprint sets (see Tables 1 and 2), although there were no

![Fig. 1. Average power output (means ± SD) during sustained 3 × 10-min endurance set and the repeat sprint interval exercise session (A) and for each interval of the repeat sprint exercise session (B) at 585 m [fraction of inspired O$_2$ (FIO$_2$) = 0.2093] and a simulated altitude of ~2,100 m (FIO$_2$ = 0.1742). A: power outputs for the 6 sprints in each set of the repeat sprint exercise session are presented as average values for the set. †Decrease. *Significant difference between conditions, *P < 0.05. ‡Significantly different from first set, *P < 0.05. B: P values indicate the main effect of altitude for each set of intervals calculated by using a 2-way ANOVA with repeated measures. ‡Significantly different from 1st interval value, *P < 0.05. n = 8 Subjects.

Table 1. Heart rate, RPE, blood pH, bicarbonate, and lactate responses during the sustained 3 × 10-min endurance set laboratory exercise session at 585 m (FIO$_2$ = 0.2093) and ~2,100 m simulated altitude (FIO$_2$ = 0.1742)

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<th>Interval 1</th>
<th>Interval 2</th>
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<tr>
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<td>585 m</td>
<td>2,100 m</td>
<td>585 m</td>
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<tr>
<td>Heart rate, beats/min</td>
<td>187 ± 11</td>
<td>185 ± 12</td>
<td>191 ± 10*</td>
</tr>
<tr>
<td>RPE (scale 6–20)</td>
<td>16 ± 2</td>
<td>17 ± 2</td>
<td>18 ± 1*</td>
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<tr>
<td>pH</td>
<td>7.33 ± 0.07</td>
<td>7.30 ± 0.05</td>
<td>7.31 ± 0.07</td>
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<tr>
<td>HCO$_3^-$, mM</td>
<td>17.0 ± 2.2</td>
<td>16.1 ± 2.0</td>
<td>16.2 ± 2.4</td>
</tr>
<tr>
<td>[La]b, mmol/l</td>
<td>7.9 ± 3.2</td>
<td>8.9 ± 3.8</td>
<td>8.1 ± 3.0</td>
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</table>

Values are means ± SD for 8 subjects. FIO$_2$, inspired O$_2$ fraction; RPE, rating of perceived exertion; pH, blood acidity; HCO$_3^-$, blood bicarbonate; [La]b, blood lactate concentration. *Significant difference from interval 1, *P < 0.05.
differences in [La]bl for either interval exercise sessions between treatments.

DISCUSSION

The first finding of this study was that maximal self-selected power output was 5–6% lower during hypoxia vs. normoxia when elite cyclists performed both short-duration (15 s) and sustained (10 min) interval exercise sessions. Sprint power output was only significantly reduced by simulated altitude when the work-to-relief ratio was 1:3. It has previously been suggested that, when elite athletes train at altitude, they are unable to sustain the high work rates and/or training velocities necessary to maintain competitive fitness (19). Indeed, Levine and Stray-Gundersen (13, 14) were the first to report that both aerobic "base" training and intense interval training were performed at slower running speeds and lower \( V\dot{O}_2 \) at altitude compared with sea level.

An important feature of the present study design was that the inspired air mixture was delivered to subjects in a double-blind fashion. In previous studies (13, 14), responses to chronic hypobaric hypoxia (after acclimatization) have been measured in subjects during actual training sessions in which the athletes were aware of their surroundings. Although the investigators in those studies made careful attempts to ensure comparable "training experiences" for both the experimental (altitude) and control (sea level) groups, it is possible that environmental factors could have influenced the subjects’ self-selected exercise intensity. For example, elite athletes use many external (visual feedback, air resistance, cadence/stride rate, how fast they are covering the ground) and internal (sensations of ventilation and HR, subjective ratings of muscular fatigue, sweat rate) cues to judge the intensity of a training session. We chose to study our subjects during maximal exercise in the laboratory setting, as many of the external cues by which they routinely judge an exercise session were effectively eliminated.

We have previously reported that, compared with normobaria (92.66 kPa = 745 mmHg, sea level), the performance of an all-out 5-min work bout was reduced by ~4% in 20 well-trained male and female athletes under mild hypobaric conditions (99.33 kPa = 695 mmHg, ~600-m simulated altitude). In that study (10), the reduction in 5-min work output was associated with both a reduced arterial \( O_2 \) content and \( V\dot{O}_2 \)peak in response to mild hypobaria. In the present investigation, the arterial oxyhemoglobin saturation was reduced throughout the endurance set under hypoxia but did not significantly change in response to the sprint session (Fig. 2). It is likely that the %SpO\(_2\) was unchanged between the normoxic and hypoxic conditions during the repeat sprint session because of a vigorous ventilatory response to these work bouts. The stimulus to ventilate during multiple short bouts of supramaximal exercise would be near maximal from both muscle and joint spindle feedback (20), as well as acidosis (23).

Hogan et al. (12) have previously reported that, during an incremental maximal test performed under hypoxic conditions (17% \( O_2 \)), [La]bl was elevated at moderate-to-high power output (>200 W) compared with normoxia, despite a similar \( V\dot{O}_2 \). [La]bl was reported to be similar at exhaustion under both experimental conditions (~9 mM), leading the authors to propose that "some critical pH level was being reached at different times under each condition" (12). In the present investigation, there was no difference in [La]bl, pH, or \( [HCO_3^-] \) during the endurance exercise session for normoxia or hypoxia. However, the fact that similar values for a variety of measures of blood acidity were

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Table 2. Heart rate, RPE, blood pH, bicarbonate, and lactate responses during the repeat sprint laboratory exercise session at 585 m (\( \text{FiO}_2 = 0.2093 \)) and ~2,100 m simulated altitude (\( \text{FiO}_2 = 0.1742 \))

<table>
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<tr>
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<th>Set 1</th>
<th>Set 2</th>
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<tr>
<td></td>
<td>585 m</td>
<td>2,100 m</td>
<td>585 m</td>
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<tr>
<td>Heart rate, beats/min</td>
<td>177 ± 11</td>
<td>173 ± 14</td>
<td>186 ± 9†</td>
</tr>
<tr>
<td>RPE (scale 6–20)</td>
<td>17 ± 2</td>
<td>17 ± 3</td>
<td>18 ± 2†</td>
</tr>
<tr>
<td>pH</td>
<td>7.30 ± 0.06</td>
<td>7.26 ± 0.05*</td>
<td>7.24 ± 0.05†</td>
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<tr>
<td>( HCO_3^- ), mM</td>
<td>16.0 ± 2.2</td>
<td>14.4 ± 1.7*</td>
<td>13.7 ± 1.8†</td>
</tr>
<tr>
<td>[La]bl, mmol/l</td>
<td>8.5 ± 2.7</td>
<td>10.5 ± 2.3</td>
<td>11.4 ± 2.8†</td>
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Values are means ± SD for 8 subjects. *Significant difference from 585 m, \( P < 0.05 \). †Significant difference from set 1, \( P < 0.05 \).
attained at lower power outputs strongly suggests that subjects might have been limited during exercise by a critical pH value. Others (17, 25) have previously reported that, when exercise is performed in a hypoxic environment, the intracellular pH is reduced by a greater extent than in normoxia for a given lactate efflux (17). During chronic hypoxia (acclimatization to altitude), the “lactate paradox” is observed such that, at any given workload, the initial (high) [La]bl gradually returns to sea-level values for the same absolute power output, despite continued hypoxia (11, 25). Taken collectively, these findings suggest that, at altitude, [La]bl is determined by factors in addition to hypoxemia, as has been recently suggested (5, 8, 16).

Few studies have examined the effect of altitude exposure on the exercise responses of athletes involved in sprint events that utilize primarily the O2-independent energy system during their event. However, acute hypoxic exposure appears to have little effect on O2-independent ATP production (26). Weyand et al. (24) recently reported that the maximal power output of four healthy men during all-out sprints that lasted 15–60 s was similar under hypoxia (13% O2) and normoxia. Similarly, when the work-to-relief ration was 1:3, we also found that 15-s maximal power output was not significantly compromised by hypoxia. Balsom et al. (2) determined the effects of simulated altitude (3,000 m, 562 mmHg) on repeated high-intensity cycle sprints (10 × 6 s with 30-s recovery) when both exercise and recovery were performed under hypoxic conditions. Hypoxia resulted in a drop in power output after the eighth work bout, which was associated with higher [La]bl. These researchers proposed that the lower power output and higher lactate levels in the hypoxic condition were due to a decreased O2 availability and an increased reliance on O2-independent glycolysis for ATP resynthesis (2). In the present study, power output was relatively more depressed in hypoxia than normoxia with shorter repeat sprint work-to-relief ratios (1:2 or 1:1), and, although [La]bl was not different under hypoxia, HCO3 and pH were each lower. Our results are consistent with the hypothesis that, during repeated sprint exercise, there is a mismatch between O2-independent energy release and power output, especially after the first sprint (which depends entirely on substrate-level phosphorylation), and that this mismatch is progressively exacerbated by hypoxia. Indeed, it has previously been reported that, during normoxia, phosphocreatine resynthesis (and thus availability) is important for recovery of power output during repeated sprint exercise (3).

While power output decreased by 5–6% under hypoxic conditions, the similarity in HR (but not necessarily cardiac output) during both treatments suggests that the maximum effort training sets were performed at the same relative intensity for each athlete. Similar HRs at V̇O2 peak at moderate altitude and sea level have been reported previously (6, 7, 19, 21, 22). Whereas no published RPE data from elite athletes during training at both altitude and sea level are available, the results of the present study indicate that, at moderate altitude, elite female cyclists not acclimatized to altitude will automatically adjust their training intensity (W) to a level that ultimately results in a similar HR and/or RPE.

Several limitations of this study should be considered when interpreting our results. The training commitments of the National squad cyclists prevented us from measuring gas exchange during the exercise interval sessions and thus precludes data on O2 kinetics or substrate utilization. Second, the recovery between sets of both endurance and repeat sprint exercise sessions was under normoxic conditions. Recovery in hypoxia may have accentuated the effect of multiple sets of sprints in a cumulative fashion, as reported by Balsom et al. (2). Therefore, our conclusion that a 1:3 work-to-relief ratio during high-intensity repeat sprint interval exercise enables athletes to exercise at sea-level power outputs should be interpreted with caution. Finally, direct arterial puncture rather than pulse oximetry is the optimal method to assess blood O2 saturation, but this methodology would have limited our access to the national level cyclists.

In conclusion, the results of this study show compromised exercise responses of elite female endurance athletes to mild hypoxia (17.42% O2) for both short-duration (15 s) and sustained (10 min) interval sets when subjects were free to select their maximum training power output. While hypoxia resulted in a reduced %SpO2 during the endurance exercise set, it did not significantly change with short-duration repeated sprints. Repeated sprint bouts under hypoxic conditions resulted in reductions in both pH and [HCO3] compared with values for normoxia. However, more commonly employed measures of training intensity, such as [La]bl, HR, and RPEs, were similar under both hypoxic and normoxic conditions. Whether the decrease in training power output of the magnitude observed in the present investigation is sufficient to result in deconditioning in highly trained athletes exercising at moderate (~2,100 m) altitude remains to be established. The maintenance of exercise intensity (power output/speed) and O2 flux is likely to be a critical factor in sustaining competitive performance (14).

We acknowledge the support of James Victor, the National Women’s Road Cycling coach, for assistance with the organization of this study, and members of the 1999 Australian Women’s Road Cycling Squad for enthusiasm and cooperation during the investigation. The technical assistance of Evan Lawton, Rob Shugg, Hamilton Lee, Nathan Townsend, and Tahnee Kinsman is gratefully acknowledged. We also thank the other members of the Department of Physiology at the Australian Institute of Sport for many valuable contributions during the data collection phase of this study. This study was supported by a grant from the Australian Olympic Athlete Program.

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