Effect of hyperoxic supplemented interval training on endurance performance in trained cyclists

by

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Summary of the Research

Training while inspiring hyperoxic gas (increasing O₂ availability) is a relatively novel strategy which allows athletes to train at a higher intensity and may possibly improve physiological adaptation and subsequent endurance performance. However, research investigating the effects of hyperoxic training is both limited and conflicting. Specifically, no research to date has been conducted on well-trained athletes, or used appropriate training methodologies throughout the hyperoxic interventions that simulate typical training sessions of athletes. The aim of this study was to determine the effect of hyperoxic supplemented interval training on high-intensity endurance performance, and physiological correlates of endurance performance in well-trained cyclists.

A single-blind, randomised control-trial design was adopted. Sixteen well-trained cyclists were randomly assigned to either an experimental group that trained in hyperoxia (HYP) or a sham HYP training group that trained in normoxia (N). Participants were required to visit the laboratory twice per week for four weeks to perform 8 high-intensity interval training sessions in HYP or N. Participants attended the laboratory on four occasions pre-intervention and two visits post intervention for performance (20km time trial) and physiological (incremental step test and Wingate test) assessments.

There were small effects on most physiological measures including VO₂peak (1.9 ± 4.3%) and lactate threshold (0.3 ± 8.3%) in HYP. In most instances physiological changes in N were greater than HYP. With respect to performance, there was a small increase in mean power during the 20km TT in HYP (2.1 ± 3.7%) but this was less than that observed for N (4.9 ± 3.9%; ES: -0.44 ± 0.60). During the 60 s all-out test the peak relative power was slightly reduced after HYP (-0.4 ± 1.3%) but not N (0.3 ± 1.6%; ES: -0.24 ± 0.76), whereas there was a tendency for mean relative power to be increased in N (2.3 ± 3.4%) but not HYP (0.3 ± 1.2%; ES: -0.34 ± 0.49).

In conclusion, 8 sessions of hyperoxic supplemented interval training had minimal effect on endurance and anaerobic performance and physiology in trained endurance cyclists compared to training in normoxia. The use of hyperoxic supplemented training at sea-level appears to be not worthwhile for competitive endurance athletes.
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Background

Training strategies that permit maximisation of workload present the possibility of greater training adaptations and performance enhancement. Inspiring a hyperoxic gas mixture while performing intense exercise allows higher power outputs to be achieved both acutely and chronically (Armstrong, Jacks, Sowash, & Andres, 2000; Perry, Reid, Perry, & Wilson, 2005; Perry, Talanian, Heigenhauser, & Spriet, 2007; Welch, 1987) compared with breathing normoxic air.

While it has been clearly demonstrated that higher power outputs can be maintained with hyperoxic training and plausible mechanisms exist supporting its efficacy, research investigating changes in normoxic performance following hyperoxic training is unclear. For example, Plouts-Snyder et al (1996) found that despite increased training intensity in hyperoxia, no additional improvements in VO$_{2\text{max}}$ were observed following 5 weeks of training in hyperoxia at 70% VO$_{2\text{max}}$ when compared to the same training conducted in normoxia by a control group. Conversely, utilising a crossover design, Perry et al. (2007), observed substantial increases (117%) in time to exhaustion following a 6 week period of hyperoxic training, compared to Normoxia (50%). The authors proposed that the additional stimulus provided by the higher training power outputs in hyperoxia might have increased skeletal muscle mitochondrial oxidative capacity to a greater extent than the equivalent training in normoxia. However, more recently, the same authors (Perry, et al., 2007) failed to demonstrate any substantial improvements in exercise performance, VO$_{2\text{max}}$ or mitochondrial exercise capacity following 4 weeks of hyperoxic training, using a similar training regime to their earlier study.

All previous studies, however, have been conducted with untrained (Perry, et al., 2005) or recreationally active participants (Armstrong, et al., 2000; Perry, et al., 2007), and have prescribed training intensities which are not reflective of those performed by athletes (Perry, et al., 2007; Ploutz-Snyder, et al., 1996). Collectively, this makes it difficult to ascertain if hyperoxic supplemented training may, or may not, be a useful training strategy for well-trained athletes at sea-level. Clearly, further research is warranted to determine the performance and physiological effects of hyperoxic training in endurance athletes using appropriate training intensities and assessment protocols.
Research Aims

The aims of this research were two-fold:

- To determine the effects of hyperoxia supplemented interval training on physiological measures relevant to endurance performance
- To determine the effects of hyperoxia supplemented interval training on 20km TT cycling performance.
Methodology

Study Design
This study employed a single-blind randomised control-trial design. Participants (n=16) were randomly assigned to either an experimental group that trained in hyperoxia (HYP) or a sham-HYP control group that trained in normoxia (N). Participants were required to visit the laboratory twice per week for four weeks to perform high-intensity interval training in HYP or N. Participants attended the laboratory on four other occasions for pre- and post-intervention performance trials and physiological assessments.

Participants
The Participants were well-trained competitive male cyclists. Prior to the start of the study all Participants had been training consistently for at least three months. The study took place during the competitive season. All gave voluntary informed consent as required by the institutional ethics committee.

Procedures and measures
All physiological and performance tests and prescribed training was performed on an electromagnetically controlled cycle ergometer (Veletron RacerMate, USA) in a temperature controlled laboratory (21Deg C, 65% rH) in N.

Incremental exercise test
To determine peak oxygen uptake (VO$_{2peak}$), the lactate threshold (LT) and lactate turnpoint (LTP), an incremental step test was performed. The test commenced at 150W and increased by 30 W every 3 min until volitional exhaustion. At the end of each 3-min stage, capillary blood was sampled from an earlobe for measurement of blood lactate using a hand-held analyser (Lactate Pro, Arkray, Japan). In addition, pulmonary oxygen uptake (VO$_2$) was measured continuously using a breath-by-breath metabolic system (Metamax 3b, Cortex, Leipzig, Germany), and heart rate (HR) continuously measured using a short-range telemetry device (Polar 810, Polar Electro, Kempele, Finland). The VO$_{2peak}$ was defined as the highest 30 s VO$_2$ value, LT was defined as the first increase in blood lactate above baseline levels and the LTP was defined as the second steep increase in B[lac].
Time trial performance
Participants presented to the laboratory 48 hours after completing the incremental step test. Following their 6-minute self-selected warm-up (recorded and repeated post-test) participants commenced the 20km-TT from a stationary start (seated, with no crank movement). Participants remained seated throughout, but were able to select and vary their own gearing and cadence. At 5-minute intervals HR and power were recorded.

Anaerobic Power
Twenty minutes after the 20km time trial, during which participants rested for 5 minutes, cycled at 50% \( VO_2\text{peak} \) for 10 minutes before resting again for 5 min, a 60 s Wingate test was performed. The peak and mean power over the 60 s all-out effort were determined.

Hyperoxic and Normoxic Training
All participants visited the laboratory twice per week for four weeks to perform high-intensity interval training. Interval sessions alternated between 12 x 2-minutes, with 2 minutes recovery and 5 x 5 minutes, with 3 minutes recovery. Participants were simply encouraged to complete as much work as possible during each interval by adjusting the power on the ergometer. The average power, HR and arterial oxygen saturation (\( SaO_2\% \)) was determined during each training session. During the training, participants inspired through a mouthpiece connected to multiple 250L Douglas bags that contained either 21% or 60% humidified O\(_2\). Participants wore a nose clip throughout each training session. A metabolic system (MM3b, Cortex, Leipzig, Germany) was used to verify the fraction of inspired O\(_2\). During recovery between intervals, participants breathed ambient air and were permitted to drink ad-libitum. Participants were not informed if they were breathing HYP or N at any time throughout the intervention.

Participants were requested to maintain other supplementary training throughout the study period which, due to the two maximal laboratory-based interval sessions, consisted mainly of low to moderate intensity cycling. Training diaries were used to record each session so that training volume and intensity could be determined.
**Statistical Analysis**

Simple group statistics are shown as means ± between-subject standard deviations. To make inferences about true (population) values of the effect hypoxia on performance, the uncertainty in the effect was expressed as 90% confidence limits and as likelihoods that the true value of the effect represents substantial change (harm or benefit) (Batterham & Hopkins, 2006). An effect was deemed unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be substantially positive and negative, or beneficial and harmful. An estimate of the smallest worthwhile change in power output was required to make these inferences. For performance measures, we assumed a smallest worthwhile effect of 1%. For all other measures we used 0.2 of baseline between-subject standard deviation. Effect size (ES) for each measure were calculated using Cohen’s $d$ statistic and interpreted using Hopkins' categorisation criteria where: 0.2, 0.6 and $>1.2$ were considered small, medium and large, respectively.
Results

Four participants did not complete the study due to illness. The characteristics and baseline exercise performance of the cyclists who completed the study are shown in Table 1. Overall, pre-training measures were similar between groups.

Table 1. Characteristics and baseline measures of the HYP and NC participants. Data are mean ± SD.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>HYP</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Yrs)</td>
<td>25.8 ± 5.7</td>
<td>24.5 ± 9.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 ± 6</td>
<td>176 ± 8</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>75.5 ± 8.2</td>
<td>75.9 ± 13.3</td>
</tr>
</tbody>
</table>

**Incremental step test**

<table>
<thead>
<tr>
<th></th>
<th>HYP</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak Aerobic Power (W)</td>
<td>340 ± 36</td>
<td>350 ± 25</td>
</tr>
<tr>
<td>Lactate Threshold Power (W)</td>
<td>230 ± 25</td>
<td>240 ± 10</td>
</tr>
<tr>
<td>Lactate Turnpoint (W)</td>
<td>260 ± 24</td>
<td>270 ± 11</td>
</tr>
<tr>
<td>Peak VO₂ (L/min⁻¹)</td>
<td>4.3 ± 0.8</td>
<td>4.6 ± 0.8</td>
</tr>
</tbody>
</table>

**Wingate 60 s test**

<table>
<thead>
<tr>
<th></th>
<th>HYP</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak Relative Power (W/kg⁻¹)</td>
<td>9.2 ± 1.4</td>
<td>9.4 ± 1.5</td>
</tr>
<tr>
<td>Mean Relative power (W/kg⁻¹)</td>
<td>5.8 ± 0.3</td>
<td>5.9 ± 0.3</td>
</tr>
</tbody>
</table>

**20km TT**

<table>
<thead>
<tr>
<th></th>
<th>HYP</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (min:ss)</td>
<td>31:56 ± 1:34</td>
<td>32:00 ± 2:03</td>
</tr>
<tr>
<td>Mean Power (W)</td>
<td>279 ± 37</td>
<td>276 ± 43</td>
</tr>
</tbody>
</table>

Adherence to intervention training in both HYP and N groups was 100%. The non-prescribed training hours (12.5 ± 1.7 vs. 10.9 ± 1.6 hrs), kilometers per week (381 ± 50 vs. 353 ± 47 km.wk⁻¹) and speed (29.1 ± 2.6 vs. 29.1± 1.9 km/h⁻¹) during the intervention period were similar for HYP and N respectively. However, the mean power output during intervention training was greater for HYP (100.5 ± 2.1 %MAP) compared to N (93.8 ± 3.7; Figure 1). The SaO₂% was substantially lower in N compared to HYP (92.7 ± 0.9 % vs. 97.8 ± 0.3%).
Figure 1. Mean relative power output, expressed as a percent of pre-intervention maximal aerobic power (MAP), during each interval training session in HYP and N.

Table 2 shows the mean changes in physiological and performance measures for the HYP group relative to the N group condition and statistics for the difference in the changes. HYP produced minimal effects on most physiological measures including VO$_{2peak}$ (1.9 ± 4.3%) and lactate threshold (0.3 ± 8.3%). In most instances physiological changes in N were greater than HYP, though confident limits were wide (Table 2). With respect to performance, there was a small increase in mean power during the 20km TT in HYP (2.1 ± 3.7%) but this was less than that observed for N (4.9 ± 3.9%; ES: -0.44 ± 0.60). During the 60 s all-out test the peak relative power was slightly reduced after HYP (-0.4 ± 1.3%) but not N (0.3 ± 1.6%; ES: -0.24 ± 0.76), whereas there was a tendency for mean relative power to be increased in N (2.3 ± 3.4%) but not HYP (0.3 ± 1.2%; ES: -0.34 ± 0.49).
Table 2. Mean changes in performance in hyperoxia (HYP) and normoxia (N) groups, and chances that the true difference between HYP and N is substantial.

<table>
<thead>
<tr>
<th>Measure</th>
<th>HYP mean ± SD</th>
<th>N mean ± SD</th>
<th>Difference; ±90%CL</th>
<th>Effect Sizes</th>
<th>Practical inferenceb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incremental Step Test</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Peak Aerobic Power (W)</td>
<td>0.3 ± 5.7</td>
<td>5.7 ± 4.4</td>
<td>-5.5; ± 5.4</td>
<td>0.61 ± 0.60</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>Lactate Threshold Power (W)</td>
<td>0.3 ± 8.3</td>
<td>4.0 ± 6.3</td>
<td>-3.7; ± 7.8</td>
<td>-0.45 ± 0.92</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>Lactate Turnpoint Power (W)</td>
<td>0.2 ± 7.3</td>
<td>3.6 ± 5.6</td>
<td>-3.4; ± 6.9</td>
<td>0.46 ± 0.92</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>Peak VO₂ (L/min⁻¹)</td>
<td>1.9 ± 4.3</td>
<td>4.7 ± 5.8</td>
<td>-2.8; ±4.9</td>
<td>-0.41 ± 0.61</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>Wingate 60 s Test</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Relative Power (W/kg⁻¹)</td>
<td>-0.4 ± 1.3</td>
<td>0.3 ± 1.6</td>
<td>-0.9; ± 1.9</td>
<td>-0.24 ± 0.76</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>Mean Relative power (W/kg⁻¹)</td>
<td>0.3 ± 1.2</td>
<td>2.3 ± 3.4</td>
<td>-2.0; ± 2.8</td>
<td>-0.34 ± 0.49</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>20km-TT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time (min:ss)</td>
<td>-0.7 ± 2.8</td>
<td>-2.0 ± 2.3</td>
<td>-1.3; ± 2.4</td>
<td>-0.30 ± 0.39</td>
<td>Unlikely, probably not</td>
</tr>
<tr>
<td>Mean Power (W)</td>
<td>2.1 ± 3.7</td>
<td>4.9 ± 3.9</td>
<td>-2.8; ± 3.6</td>
<td>-0.44 ± 0.60</td>
<td>Unlikely, probably not</td>
</tr>
</tbody>
</table>

b Based on a smallest beneficial or harmful change in performance of 1%

±90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.
Findings

Some (Armstrong, et al., 2000; Perry, et al., 2005; Perry, et al., 2007), but not all (Welch, 1987), studies have reported large gains in physiological measures and performance after HYP training. However, these studies have used untrained or recreationally trained individuals. To our knowledge, this is the first intervention study to investigate physiological adaptations and performance effects of HYP in well-trained cyclists.

The major finding of this study is that four-weeks (8 sessions) of hyperoxia-supplemented interval training had no additive effect on endurance performance over that induced by training in normoxia in trained cyclists, despite our attempts to optimise the nature of training prescribed based on previously successful studies using well-trained athletes (Lindsay, et al., 1996; Stepto, Hawley, Dennis, & Hopkins, 1999; Westgarth-Taylor, et al., 1997; Weston, et al., 1997). Our data is in contrast to several previous studies who reported substantial effects of HYP on performance (Armstrong, et al., 2000; Perry, et al., 2005; Perry, et al., 2007). The only obvious methodological difference between our and previous studies was the subject characteristics; ours being the only study to report the effects of HYP training in well-trained athletes.

We observed only small changes in aerobic physiological measures in HYP, and these limited effects were reflected in the small, unclear enhancement in 20km TT performance (Table 2). Conversely, the greater physiological changes in the sham hyperoxia blinded control group (Table 2), who performed high-intensity interval training in normoxia, are in keeping with previous interval training studies (Lindsay, et al., 1996; Stepto, et al., 1999; Westgarth-Taylor, et al., 1997; Weston, et al., 1997) and provide several plausible mechanisms for the observed performance enhancement.

In the present study, rather than fix the intensity of training sessions, we simply requested each subject to complete as much work as possible in each training session. This approach has been adopted successfully in previous research (Yeo, et al., 2008) and is reflective of many athletes’ approach to training. All training was individually monitored. As anticipated, however, HYP were able to complete each session with a higher mean power output (~6%; Figure 1) which is consistent with previous findings (Perry, et al., 2007; Ploutz-Snyder, et al.,
1996; Welch, 1987). Over the duration of the study (session 1 to 8), the increase in mean power in the N group increased greater than that observed in HYP (Figure 1) which suggests that training in HYP potentially blunted the normal response to training in this group of trained cyclists. Indeed, the attenuated change for most physiological measures in HYP, compared to N, would support this (Table 2).

It is well-known that exercise-induced arterial hypoxemia (EIH) becomes manifest in highly trained participants during maximal exercise (Dempsey, Hanson, & Henderson, 1984; Powers, et al., 1988) but that administration of hyperoxic gas maintains SaO₂% close to resting levels (Ansley, et al., 2007; Grataloup, et al., 2005). In the present study, we measured SaO₂% non-invasively using pulse-oximetry during all training sessions and found that the SaO₂% in HYP remained close to resting levels (~98%), whereas training close to VO₂peak in N caused SaO₂% to drop substantially (92.7 ± 0.3%). This clearly suggests that training in HYP was not as physiologically demanding as N despite the higher power outputs and that this limited the potential change in performance.

We are not the first to suggest that HYP may attenuate some adaptive responses, but that it may augment others (Perry, et al., 2007). For this reason, in addition to measuring endurance performance and aerobic function, we also assessed anaerobic performance. Indeed, given the higher power output that is possible when training in HYP, and the nature of training prescribed, we hypothesised that mean power during a 60 s all-out test would be enhanced as a result of neuromuscular and muscle recruitment related factors. However, contrary to this, changes in these measures were relatively unchanged following the HYP intervention with a tendency for peak power to decrease slightly in HYP (Table 2). These data collectively suggest that HYP training does not enhance short term ‘anaerobic’ performance.

**Limitations**

Due to illness-induced participant dropout, the lower than anticipated sample size could be considered a limitation of the present study. However, based on the observed data, it is of our opinion that further participants would not influence the findings of the present study.
Conclusion

In conclusion, 8 sessions of hyperoxic supplemented high-intensity interval training was ineffective at further augmenting physiological training adaptations and performance in trained endurance athletes compared to training in normoxia. The use of hyperoxic supplemented training at sea-level appears not to be a worthwhile strategy for competitive endurance athletes.
Acknowledgements

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References


