The Effects of Hyperoxia on Repeated Sprint Cycling Performance & Muscle Fatigue

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Abstract

Objectives: Hyperoxia (> 21% oxygen) can evoke performance improvements in aerobic and anaerobic exercise. The aims of the current study were to determine the effects of breathing hyperoxic gas (fraction of inspired oxygen [F_iO_2] 1.00) on repeated cycle performance, and to assess the nature and extent of fatigue after intermittent sprinting.

Design & Methods: Testing (n=14 males) comprised two visits to the laboratory. Each session involved 10 x 15s repeated cycle sprints breathing FiO_2 1.00 (hyperoxia) or FiO_2 0.21 (normoxia). Muscle fatigue was measured pre and post sprints using maximal voluntary contraction (MVC), voluntary activation (VA) and potentiated doublet twitch (PTF). Blood lactate (BLa) was taken between sprints.

Paired samples t-tests were used to examine difference between conditions in power output (peak and mean Watts) and BLa. Two-way ANOVA was used to examine fatigue variables pre and post sprints according to condition.

Results: Mean power output was 4% greater in hyperoxia (p<0.01), with no difference in peak power (p>0.05). There was a significant increase in BLa in hyperoxia compared with normoxia (p<0.01) in sprints 4 and 8, as well as meaningful difference in sprints 4-10. There was no significant difference in fatigue factors (MVC, VA and PTF) (p>0.05) in response to the cycling, although a large drop in PTF occurred in both conditions.

Conclusion: Hyperoxia can elicit improvements in mean cycling power, with no significant change in post exercise muscle fatigue. Hyperoxia as a training aid may provide performance enhancing effects during repeated sprint cycling by reducing concurrent muscle fatigue, primarily via peripheral factors.

Keywords: Oxygen, Power, Lactate, Training, Sprinting.

Introduction

Hyperoxia is the inhalation of air with a fraction of inspired oxygen (FiO_2) greater than that of sea level (20.9%). Supplementing high intensity exercise with FiO_2 >0.21 allows the maintenance of performance when fatigue would usually become apparent, both during aerobic and sprint exercise [1,2]. The mechanism behind this attenuation of performance decline are multifactorial and include reduced production of blood lactate (BLa)[1], enhanced clearance of BLa, prevention of muscle oxygen desaturation[3,4] the maintenance of blood pH and enhanced resynthesis of creatine phosphate [5]. These factors are associated with peripheral fatigue; i.e. the exercise induced decrease in muscle force production.

A reduction in neural drive from the motor cortex to muscle appears as a decrease in voluntary muscle activation (VA) during exercise[6]. This 'central fatigue', may also be influenced by the fraction of inspired air. Indeed, research has shown that a reduced cerebral O_2 delivery resulting from hypoxia (FiO_2 0.18) results in curtailment of exercise performance due to fatigue[7]. Thus, whether hyperoxia can alleviate central fatigue in a sport situation is unknown.

Performance decline is likely a combination of both central and peripheral factors and the relative contribution of each depends upon the nature of the task [8]. Peripheral fatigue is likely the limiting factor in short, high intensity exercise, with central fatigue playing a greater role as the exercise bout continues. For example, a single 4 km time trial lasting around 5 minutes was shown to be limited by peripheral fatigue, whilst a 20 km trial (lasting around 32 minutes) was primarily limited by central fatigue[8].

Repeated sprint efforts represent a short term high intensity exercise, which are likely to be limited primarily by peripheral fatigue [9]. However, the extent to which central fatigue contributes to performance decline in repeated sprint performance is equivocal. Racinais *et al.,* [10] determined that the ability to repeat short duration sprints was associated with both central and peripheral factors. The twitch interpolation technique is widely used and is

63 considered a reliable method to estimate the origin of neuromuscular fatigue. Peripheral
64 fatigue is measured by comparing the force responses to electrical stimulation pre and post
65 fatiguing exercise. To determine the contribution of central factors the twitch interpolation
66 technique is used, superimposing single or double twitches on MVC then comparing the
67 superimposed response to the potentiated response obtained from the relaxed muscle

68 Thus, the aims of the current study were to determine the effects of hyperoxia on repeated
69 cycle performance, and to assess the nature and extent of fatigue after sprinting.

70 It was hypothesised that repeated sprint cycle performance would decrease to a larger degree
71 in the normoxia condition compared with the hyperoxia condition. Second, it was
72 hypothesised that both central and peripheral components of fatigue would be reduced to a
73 greater extent in the normoxia condition.

Methods

Fourteen healthy males were recruited to take part in the study. Participants (1.81 ± 0.04 m, 77.6 ± 11.0 kg, 25.9 ± 7.3 years) were recreational cyclists who had all previously used a cycle ergometer. Participants were accustomed to cycling on a weekly basis, but none had ever competed at any cycling events.

Participants were informed of the procedure and provided informed consent. Ethical approval for the study was granted by the University ethics committee.

This study was a within subjects design with 2 visits to the laboratory in a counter balanced order, in a single blind fashion. Participants completed a series of sprints under two different conditions; hyperoxia (FiO_2 ~ 1.00) or normoxia (FiO_2 ~ 0.21). Visits were separated by at least 48hrs.

Laboratory tests were completed at the same time of the day (±2h). Participants were asked to maintain normal activity and sleep patterns between testing sessions. Participants were requested to refrain from any caffeinated products or eating three hours prior to participation. Participants were asked to refrain from strenuous physical activity 24 hours prior to participating.

Participants undertook the same procedure on both visits; 3x5s Maximal Voluntary Contractions (MVC) then 15 minute relative intensity warm up at 52% of heart rate reserve using the rearranged Karvonen formula [11,12] . This was followed by 10-minute passive recovery, 3x5s MVCs (pre-sprint baseline) and 10x15s cycle sprints with 45 seconds of recovery. Finally, a further set of 3x5s MVCs (post-sprint). Gas administration occurred at the commencement of the first sprint and continued throughout the sprints and the post sprint MVC's.

Hyperoxic and normoxic gas mixtures were administered via a rig of 4 x 200 L Douglas bags connected to a mask and head net (Hans Rudolph, Shawnee, KS, USA). The hyperoxia condition used medical grade oxygen (BOC, Surrey, UK). In each condition, participants wore

100 the mask and breathed from the Douglas bag during the repeated sprints and the last set of
101 MVC's.

102 Prior to starting the protocol, a pre-exercise 20 µl capillary sample was taken from the right
103 ear lobe. Each sample was mixed with haemolysing solution within a 0.5 ml haemolysing
104 solution cup. Further samples were taken during the recovery period of each sprint repetition.
105 All samples were analysed for blood lactate within 24 hours of withdrawal using a Biosen (EKF
106 diagnostics, Cardiff, UK).

107 Muscle fatigue was assessed prior to and after the repeated sprints using electrical stimulation
108 of the right femoral nerve. The right leg was used regardless of dominance due to
109 measurement restraints. The variables obtained to assess muscle performance were;
110 maximal voluntary contraction (MVC), voluntary activation (VA) and potentiated doublet twitch
111 force (PTF). Muscle fatigue was measured within 1 minute of exercise cessation before the
112 decline in force dissipates [13].

113 Knee extensor force (N) during voluntary and stimulated contractions was measured using a
114 calibrated load cell dynamometer (Kin-Com, Chattanooga Group Inc., USA), attached to a
115 custom-built chair. The participant's ankle was strapped to a load cell immediately superior to
116 the right malleoli. Participants were instructed to maximally extend their leg against a static
117 load cell at 90° for 5 seconds. Femoral nerve stimulation was delivered during the middle of
118 each contraction and additionally ~5s after contraction, to determine potentiated quadriceps
119 twitch force and peripheral voluntary activation. PTF was measured as the highest force
120 produced during the three repetitions evoked by a paired pulse stimulus, administered to the
121 resting muscle via the nerve at rest, 5 seconds post the MVC[14]. VA was determined using the
122 interpolated doublet twitch technique and is estimated by the changes in the interpolated
123 doublet twitch relative to the PTF (equation1). The force evoked by the imposed electrical
124 stimulus on top of the MVC is the interpolated doublet twitch (IT).

125 **Eq 1. Determining voluntary activation** [15].

126 $$VA(\%) = \left(1 - \frac{IT}{PTF}\right) \cdot 100$$

127

128 Doublet- twitch electrical stimuli of 200μs pulse width were delivered to the right femoral nerve
129 via surface electrodes (Axelgaard ValuTrode) and a constant current stimulator (DS7AH;
130 Digitimer Ltd., Welwyn Garden City, UK). A signal converter was used to convert the digital
131 signals of the computer to analogue signals of the digitimer with a sampling rate of 2000 Hz
132 (PowerLab/4st – ML760, AD Instruments, UK). The cathode was positioned on the femoral
133 triangle. The anode was positioned 3cm proximal to the base of the patella, whilst the knee
134 was fully flexed[16]. Prior to application of the pads, the area was shaven. The electrode
135 placement was marked with semi-permanent ink to ensure consistent placement between
136 trials.

137

138 Prior to the MVCs, participants completed resting twitch stimuli in order to determine the
139 maximal twitch amplitude and M-wave of the muscle at rest (the resting immediate response
140 to an electrical stimulation). Doublet twitch stimuli were delivered starting at 100mA and
141 increasing to 150mA then increasing in stepwise increments of 25mA, until a plateau occurred
142 in twitch amplitude. To ensure a full and optimal stimulus the last twitch was increased by a
143 further 30%. Offline analysis was enabled with the use of LabChart 7.0 software (AD
144 Instruments, UK).

145

146 Following the warm up and two sets of MVCs each participant undertook 10 repetitions of 15
147 second cycling sprint (Watt Bike, Nottingham, UK) followed by 45 sec static recovery.
148 Participants were instructed to stay seated to isolate leg power. The air brake was set to 10
149 and magnetic brake set to one to allow sufficient resistance to generate peak force, whilst not
150 exceeding peak cadence. During each sprint and recovery period the participants breathed
151 either normoxic or hyperoxic air via the Douglas bag system. Data used for analysis were peak
152 sprinting power (the highest W achieved in each cycle) and mean sprint power (the average

W produced during each 15 sec cycle). An overall peak and an overall mean were also calculated for each participant.

All statistical analysis was performed using the statistical package, SPSS statistics version 25 for windows (SPSS Inc, Chicago, IL, USA).

Paired samples t-tests (Bonferroni corrected) were conducted to examine differences according to condition (hyperoxia / normoxia) for; peak power (W), mean power (W) across each 15 second sprint, and blood lactate (mmol/L) for each sprint. Two-way analysis of variance (ANOVA) were conducted to test the differences between MVC, VA, PT before and after the repeated sprints, according to condition. Alpha was set at $p = 0.05$ for all data analysis. Effect size for individual measures were calculated and reported as Cohen's *d* and interpreted using bounds as 0.2, 0.5, > 0.8, where they are small, medium and large respectively[17].

Results

There was no difference in peak sprinting power (W) between the hyperoxia (753.3 ± 87.8) and normoxia (761.0 ± 97.4) conditions across the 10 sprint repetitions; t (9) = 1.09, p =0.304, ES = -0.08. However, average power was significantly higher (around 25W) in the hyperoxia condition (654.6 ± 86.9) compared with the normoxia condition (629.2 ± 96.2) across the 10 sprint repetitions; t (9) = -4.65, p= 0.001, ES = 0.28 (figure 1).

Mean blood lactate was higher in the hyperoxia condition (9.81 mmol/L), although only by a small margin (0.43 mmol/L) t (9) = 3.36, p=0.008, ES = -0.13. When comparing sprints directly between conditions, it was only after sprints 4 and 8 that this difference reached significance (figure 2).

MVC : As expected there was a main effect of time on muscle force, ($F(1,12)=34.47$, $p<0.001$, ES = 4.14) with a decrease in MVC post the sprints (pre 774.4 ± 46.3 vs post 587.9 ± 43.7) Despite a somewhat larger decline in MVC in the hyperoxia trial, there was no statistical difference between conditions (p=0.66,) (table 1). There was no interaction effect for condition x time (p=0.08)

PTF: Again a main effect was found for time ($F(1,12)=53.03$, $p<0.001$, ES = 8.66) with a smaller potentiated doublet twitch production post sprints compared to pre-sprint (pre 459.2 ± 22.2 vs post 290.4 ± 16.5), but not for condition (p=0.86, ES = -0.03). There was no interaction effect reported for condition x time (p=0.31) for PTF.

VA: A main effect was found for condition ($F(1,12)=8.23$, p=0.013, ES = 2.23) with a higher voluntary activation in hyperoxia compared with normoxia (79.1 ± 2.2 vs 74.3 ± 2.1 respectively), but no effect of time (p=0.14). Importantly there was no interaction effect reported for condition x time (p=0.79) for VA.

Discussion

The aim of this study was to identify the effects of hyperoxia on repeated sprint ability and begin to examine its effects on muscle fatigue using interpolated twitch. Though peak cycling power was not different between conditions, average power was higher in the hyperoxia trials, by around 4%, indicating that a higher work output could be maintained in the presence of extra oxygen. This higher mean power was associated with a higher blood lactate level. There was no significant effect of condition on changes in muscle fatigue measures (assessed by MVC and electrical stimulation).

The current study found that breathing hyperoxic air during 15 second repeated sprint efforts led to a higher mean power output, (around 25 Watts), with no significant influence on peak power. Hauser *et al.,*[18] found that mean power was not different between the two conditions in their study (FiO_2=1.00 and FiO_2=0.21). However, their methodology of 3 x 3-minute sprints meant their participants were potentially predominately using a different energy system to that used in a 15 second sprint. The lactic acid system and the aerobic system are the predominate sources of ATP production during maximal 3-minute efforts, whereas the during 15 second sprints the majority of ATP is supplied by the ATP-PC system. Second, Hauser's participants experienced hyperoxia between efforts only. Timing of hyperoxia is potentially key. For example, Sperlich et al [19] also report no difference in mean or peak power across 2 sets of 5 x 30 sec cycle sprints. Although nature of the exercise was more similar to the current study, in contrast, their participants only had supplementary oxygen in the 6-minute recovery period between sets of cycle sprints.

Hyperoxia has been shown to attenuate the onset of fatigue at a peripheral level whilst also maintaining cerebral oxygenation [20]. In the current study, MVC, a global measure of fatigue, dropped by around 20% in the normoxia condition, and by around 28% in the hyperoxia condition. This shows that there was greater (albeit non-significant) muscle fatigue as a result

220 of the higher mean power output in the hyperoxia condition. Interestingly, the change in PTF
221 and %VA were broadly similar across conditions. The drop in voluntary activation (%) was
222 small and less than 1.5% different between conditions. The drop in PTF however was large,
223 although again similar between conditions (34 and 38%, Table 1). These results support the
224 findings of Thomas et al [8], who report performance in shorter efforts is predominately curtailed
225 by peripheral measures.
226
227 The ability to maintain power throughout repeated efforts has been attributed to several factors
228 including the ability to maintain BLa, regulate pH, to maintain neural input[21] and importantly,
229 to replenish phosphocreatine (PC) stores. According to Linossier et al., [1] sprint capacity is not
230 reduced to the same extent in hyperoxia due to the increased rate of cellular metabolic
231 resynthesis of PC, and adenosine triphosphate (ATP). PC resynthesis during 45 seconds of
232 recovery only replenishes ~75% of the stores [22]. Therefore, after sprint 5 in a series such as
233 this one would expect to see a significant performance reduction. A continued depletion of
234 ATP-PC stores and an inadequate resynthesis leads to a reduction in performance until
235 additional energy systems aid in the resynthesis process (lactic acid system). An increase in
236 peak and mean power has been attributed to an enhanced PC resynthesis during repeated
237 high intensity exercise by both Mendez- Villanueva *et al.,*[23] , and Glaister,[22]. Mendez-
238 Villanueva analysed PC recovery rate during 10 x 6s sprints with 30 seconds recovery and
239 found that subsequent sprinting performance (peak power) during a final single sprint was
240 increased corresponding with an 8% higher PC resynthesis.
241
242 Increasing the percentage of inspired oxygen to 100% increases the rate of PC replenishment
243 from a half-life of 25 seconds to 20 seconds[24,25]. Hogan et al.,[24] used a plantar flexion exercise
244 protocol with increasing workload till exhaustion. They found that an increased rate of PC
245 resynthesis aided subsequent performance, and that hyperoxia maintained mean plantar
246 flexion power further into the ramp test protocol (1W increase every 2 minutes by pulley
247 system). This findings was replicated in the current study as mean sprinting power during

hyperoxia was similar to that during normoxia until sprint 4 and beyond where the difference became statistically significant. Additionally, sprint 6 was where peak power in the normoxic group appeared to decline at a greater rate than the hypoxia condition. Sprint 6 in a typical series is where it has been documented the reliance on aerobic metabolism increases [22].

Interestingly, the increase in sprint performance as a result of hyperoxia was similar to that seen with creatine supplementation, likely through the same mechanisms of PC resynthesis [22]. Additional to this enhanced rate of resynthesis, hyperoxia has been shown to attenuate the build-up of metabolic by products of lactate, and inorganic phosphate (Pi)[26]. Pi in particular is detrimental to performance via inhibition of muscle afferents. Type III and IV muscle afferents relay exercise induced metabolic changes in the muscles to the central nervous system. The accumulation of metabolic by-products reduces the effectiveness of these afferents, subsequently leading to peripheral fatigue. However, both afferents are positively influenced by hyperoxia, by allowing the electrical feedback to be transmitted efficiently for longer[26], so attenuating fatigue.

Hyperoxia elicits reductions in blood lactate at many workloads [1,27] and although hyperoxia given during recovery periods attenuates lactate accumulation, the effects are more variable. [3]. Maeda et al., gave varying percentages of hyperoxia (30 to 100%) in the recovery between sprints, and found that whilst overall increasing the fraction of oxygen resulted in reduced blood lactate after standardised exercise, the response was dependent on the subjects' fitness. In the current study, higher power outputs seen in the hyperoxia condition were associated with slightly increased lactate levels. Knight *et al.,*[28] suggest that the increase in oxygen kinetics during hyperoxia is enough to attenuate the accumulation of lactate due to the increased diffusion of oxygen into the mitochondria. However, they add that that this attenuation can only last so long, and after a critical point lactate levels will increase exponentially. This could explain the increases in lactate that have been seen in the current study.

Therefore, it is suggested that hyperoxia results in a combination of increased PC resynthesis and a slightly attenuated build-up of blood lactate, leading to a 'maintained' performance compared to the normoxia condition seen in the current study. Acute exposure to additional oxygen appears to enhance repeated sprint performance, however, determining whether chronic physiological adaptation is blunted is crucial before its widespread use as a training tool can be advised.

Limitations

We acknowledge several limitations of this study. Participants were required to avoid strenuous exercise 24hrs prior to testing but it is noted that the effects of training may be evident for 48/72 hrs. To minimise the effects of this, participants were requested to mimic the training three days prior to testing before both visits. Further, no direct measure of fitness ($\dot{V}O_2$ max) was conducted to characterise the study population. Level of fitness is a potential mediator of response to hyperoxia. Additionally, hydration status was not measured prior to testing, but participants were instructed to attend testing in a hydrated state.

Conclusion

Whilst supplementary oxygen does not increase peak power during repeated sprints, participants were able to maintain a higher mean power output (across the 10 sprints). Indices of fatigue (MVC, PFT and VA) changed to a similar extent across conditions in response to the cycling, but the largest drop was in PFT, suggesting fatigue to be predominately peripheral in nature.

Practical implications

- Supplementary oxygen given during a single sprint-based cycling session can assist in reducing the extent that performance decreases.
- Peak power output cannot be increased with supplementary oxygen

- Long term effects of the use of oxygen during training are not known and therefore its use as a chronic training tool is not yet advised

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382 **Table 1:** Neuromuscular function of the knee extensors; Maximal Voluntary contraction

383 (MVC), potentiated doublet twitch (PTF) and voluntary activation (VA) (n = 14).

	Normoxia			Hyperoxia		
	Pre-Sprint	Post Sprints	% Difference	Pre-Sprint	Post Sprints	% Difference
MVC (N)	762.6 ± 169.1	611.3 ± 150.6	19.8	786.1 ± 187.0	564.4 ± 204.6	28.2
PTF (N)	452.1 ± 86.0	295.4 ± 64.0	34.6	466.2 ± 95.5	285.4 ± 67.4	38.8
VA (%)	75.8 ± 10.0	72.8 ± 9.7	3.9	81.2 ± 10.3	77.1 ± 9.6	5.0

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389 **Figure 1.** Mean sprinting power across 10 sprints (n = 14). * significant difference

390 between conditions (Hyperoxia and Normoxia) ($p < 0.05$).

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395 **Figure 2**. Mean blood lactate concentration (mmol/L) over 10 sprints (n = 14). *

396 significant difference between condition (Hyperoxia and normoxia) ($p < 0.05$).

Supplementary Table

Table 1. Mean ± SD and Cohens D effect Sizes for three variables in each condition for each of the 10 repeat cycling sprints (n =14).

Sprint No.	*One*	*Two*	*Three*	*Four*	*Five*	*Six*	*Seven*	*Eight*	*Nine*	*Ten*
Mean Power										
Normoxia	842.46 ± 105.17	724.96 ± 92.88	685.49 ± 98.34	634.51 ± 103.25	606.80 ± 110.01	564.59 ± 111.96	558.25 ± 103.31	541.18 ± 104.65	557.90 ± 99.21	575.75 ± 115.21
Hyperoxia	828.89 ± 139.45	770.36 ± 129.76	696.71 ± 110.11	671.37 ± 125.76	632.03 ± 120.82	592.14 ± 120.60	579.30 ± 115.21	585.90 ± 104.63	588.79 ± 107.63	600.38 ± 119.93
ES	-0.11	0.40	0.11	0.32	0.22	0.24	0.19	0.43	0.30	0.21
Peak Power										
Normoxia	958.29 ± 120.03	862.57 ± 109.61	835.79 ± 99.77	776.29 ± 111.79	732.36 ± 113.37	699.14 ± 128.97	680.79 ± 106.48	655.62 ± 115.01	674.42 ± 114.36	734.83 ± 148.52
Hyperoxia	921.27 ± 115.22	870.99 ± 88.89	798.44 ± 96.66	786.48 ± 142.55	730.92 ± 124.85	684.57 ± 150.55	673.17 ± 137.09	678.97 ± 132.18	687.38 ± 124.47	921.27 ± 133.62
ES	-0.31	0.08	-0.38	0.08	-0.01	-0.10	-0.06	0.19	0.11	1.32
Blood lactate										
Normoxia	3.05 ± 1.64	5.02 ± 1.57	7.08 ± 2.86	8.44 ± 2.68	10.14 ± 3.18	10.74 ± 3.28	11.98 ± 3.79	12.47 ± 3.80	12.00 ± 3.50	12.85 ± 3.30
Hyperoxia	2.87 ± 0.59	5.36 ± 1.74	7.48 ± 2.01	9.50 ± 2.33	10.57 ± 3.31	11.34 ± 3.09	12.28 ± 2.75	12.79 ± 2.55	13.10 ± 2.68	12.81 ± 2.93
ES	-0.15	0.21	0.16	0.42	0.14	0.19	0.09	0.10	0.35	-0.01