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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 × 15 s 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.

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.

1. 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

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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 min was shown to be limited by peripheral fatigue, whilst a 20 km trial (lasting around 32 min) 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 considered a reliable method to estimate the origin of neuromuscular fatigue. Peripheral fatigue is measured by comparing the force responses to electrical stimulation pre and post fatiguing exercise. To determine the contribution of central factors the twitch interpolation technique is used, superimposing single or double twitches on MVC then comparing the superimposed response to the potentiated response obtained from the relaxed muscle.

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

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

2. 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 48 h.

Laboratory tests were completed at the same time of the day (±2 h). 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 h prior to participating.

Participants undertook the same procedure on both visits; 3 × 5 s Maximal Voluntary Contractions (MVC) then 15 min relative intensity warm up at 52% of heart rate reserve using the rearranged Karvonen formula.[11,12] This was followed by 10-min passive recovery, 3 × 5 s MVCs (pre-sprint baseline) and 10 × 15 s cycle sprints with 45 s of recovery. Finally, a further set of 3 × 5 s 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 × 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 the mask and breathed from the Douglas bag during the repeated sprints and the last set of MVC's.

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

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

Knee extensor force (N) during voluntary and stimulated contractions was measured using a calibrated load cell dynamometer (Kin-Com, Chattanooga Group Inc., USA), attached to a custom-built chair. The participant's ankle was strapped to a load cell immediately superior to the right malleoli. Participants were instructed to maximally extend their leg against a static load cell at 90° for 5 s. Femoral nerve stimulation was delivered during the middle of each contraction and additionally 5̃ s after contraction, to determine potentiated quadriceps twitch force and peripheral voluntary activation. PTF was measured as the highest force produced during the three repetitions evoked by a paired pulse stimulus, administered to the resting muscle via the nerve at rest, 5 s post the MVC.[14] VA was determined using the interpolated doublet twitch technique and is estimated by the changes in the interpolated doublet twitch relative to the PTF (Eq. (1)). The force evoked by the imposed electrical stimulus on top of the MVC is the interpolated doublet twitch (IT).

Eq. (1). Determining voluntary activation.[15]

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

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

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

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

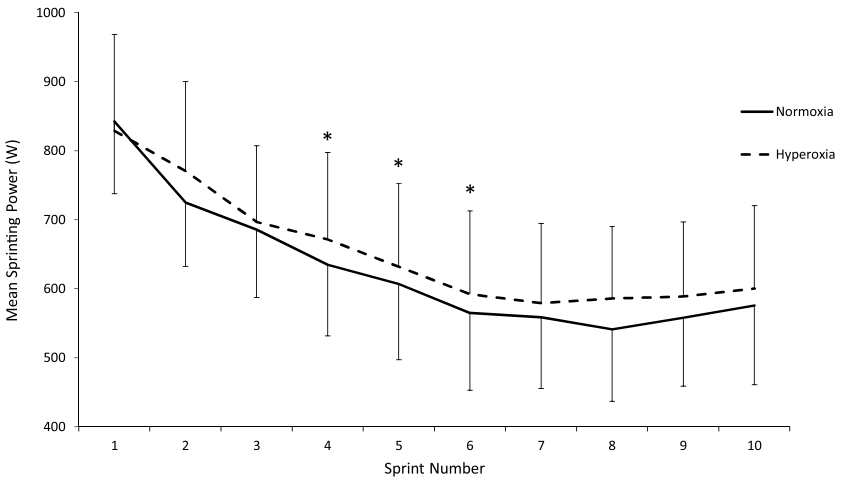

Fig. 1. Mean sprinting power across 10 sprints (n = 14). * significant difference between conditions (Hyperoxia and Normoxia) ($p < 0.05$).

ing each 15 s 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 s 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]

3. 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 25 W) 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 (Fig. 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 (Fig. 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 × 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.

4. 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 s repeated sprint efforts led to a higher mean power output, (around 25 W), 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 × 3-min sprints meant their participants were potentially predominately using a different energy system to that used in a 15 s sprint. The lactic acid system and the aerobic system are the predominate sources of ATP production during maximal 3-min efforts, whereas the during 15 s 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 × 30 s 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-min 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 of the higher mean power output in the hyperoxia condition. Interestingly, the change in PTF and %VA were broadly similar across conditions. The drop in voluntary activation (%) was small and less than 1.5% different between conditions. The drop in PTF however was large, although again similar between conditions (34 and 38%, Table 1). These results support the findings of Thomas et al,[8] who report performance in shorter efforts is predominately curtailed by peripheral measures.

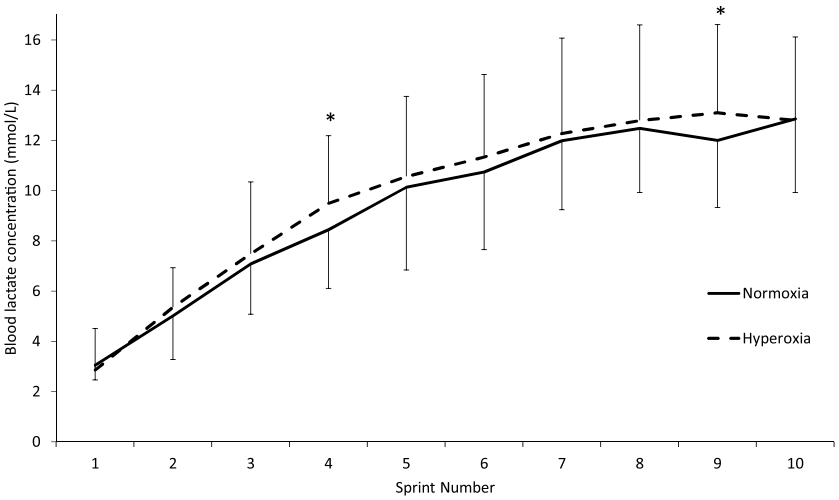

Fig. 2. Mean blood lactate concentration (mmol/L) over 10 sprints (n = 14). * significant difference between condition (Hyperoxia and normoxia) ($p < 0.05$).

Table 1
Neuromuscular function of the knee extensors; Maximal Voluntary Contraction (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

The ability to maintain power throughout repeated efforts has been attributed to several factors including the ability to maintain BLa, regulate pH, to maintain neural input [21] and importantly, to replenish phosphocreatine (PC) stores. According to Linossier et al.,[1] sprint capacity is not reduced to the same extent in hyperoxia due to the increased rate of cellular metabolic resynthesis of PC, and adenosine triphosphate (ATP). PC resynthesis during 45 s of recovery only replenishes ~75% of the stores.[22] Therefore, after sprint 5 in a series such as this one would expect to see a significant performance reduction. A continued depletion of ATP-PC stores and an inadequate resynthesis leads to a reduction in performance until additional energy systems aid in the resynthesis process (lactic acid system). An increase in peak and mean power has been attributed to an enhanced PC resynthesis during repeated high intensity exercise by both Mendez- Villanueva et al.,[23] and Glaister.[22] Mendez- Villanueva analysed PC recovery rate during 10 × 6 s sprints with 30 s recovery and found that subsequent sprinting performance (peak power) during a final single sprint was increased corresponding with an 8% higher PC resynthesis.

Increasing the percentage of inspired oxygen to 100% increases the rate of PC replenishment from a half-life of 25 s–20 s.[24,25] Hogan et al.,[24] used a plantar flexion exercise protocol with increasing workload till exhaustion. They found that an increased rate of PC resynthesis aided subsequent performance, and that hyperoxia maintained mean plantar flexion power further into the ramp test protocol (1 W increase every 2 min by pulley 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 (30–100%) 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.

5. Limitations

We acknowledge several limitations of this study. Participants were required to avoid strenuous exercise 24 h prior to testing but it is noted that the effects of training may be evident for 48/72 h. 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 ($V\dot{O}_2$ 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.

6. 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.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jsams.2019.07.001.

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