THE EFFECTS OF TRAINING IN HYPEROXIA VS NORMOXIA ON SKELETAL MUSCLE ENZYME ACTIVITIES AND EXERCISE PERFORMANCE

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Running Head: Metabolic adaptations to hyperoxic training

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ABSTRACT

Inspiring a hyperoxic gas permits subjects to exercise at higher power outputs while training, but there is controversy as to whether this improves skeletal muscle oxidative capacity, VO\textsubscript{2}max and endurance performance to a greater extent than training in normoxia (N). To determine whether the higher power output during H training leads to a greater increase in these parameters, nine recreationally active subjects were randomly assigned in a single blind fashion to train in H (60% O\textsubscript{2}) or N for 6 wks (3 sessions/wk of 10 x 4 min at 90% VO\textsubscript{2}max). Training HR was maintained during the study by increasing power output. After at least 6 wks of de-training, a second 6 wk training protocol was completed with the other breathing condition. VO\textsubscript{2}max and cycle time to exhaustion at 90% of pre-training VO\textsubscript{2}max were tested in room air pre and post training. Muscle biopsies were sampled pre and post training for citrate synthase (CS), β-hydroxyacyl-CoA dehydrogenase (β-HAD) and mitochondrial aspartate aminotransferase (m-AsAT) activity measurements. Training power outputs were 8% higher (17 W) in H vs N. However, both conditions produced similar improvements in VO\textsubscript{2}max (11-12%), time to exhaustion (~100%), and CS (H, 30%; N, 32%), β-HAD (H, 23%; N, 21%) and m-AsAT (H, 21%; N, 26%) activities. We conclude that the additional training stimulus provided by training in H was not sufficient to produce greater increases in the aerobic capacity of skeletal muscle, and whole body VO\textsubscript{2}max and exercise performance as compared to training in N.

Key Words: citrate synthase, β-hydroxyacyl-CoA dehydrogenase, mitochondrial oxidative capacity, high intensity interval training
INTRODUCTION

Inspiring a hyperoxic gas mixture (H) can improve acute aerobic exercise performance and allows subjects to exercise at higher power outputs for a given heart rate (HR) and with a lower RPE for a given power output as compared to breathing normoxic (N) air (2, 9, 13, 16, 25, 26). The ability to exercise at higher power outputs with H also persists during an entire 5-6 wk training paradigm as compared to N (15, 17). Thus, inspiring H allows individuals to train at higher aerobic intensities, which may provide a greater stimulus for enhancing endurance performance. However, it is not clear whether the additional training stimulus from H results in greater adaptive responses in mitochondrial oxidative capacity or exercise performance, relative to N.

Ploutz-Snyder et al (17) reported that even though higher power outputs (~ 20 W or ~ 12%) were achieved for a given HR while training in H at ~70% of VO2max each week for 5 weeks, no additional increase in VO2max occurred as compared to N. Nevertheless, despite the greater training stimulus with H, cytochrome c oxidase and citrate synthase (CS) activities increased to a greater extent in N relative to H, and β-hydroxyacyl-CoA dehydrogenase (β-HAD) activity only increased in the N group. However, this study used separate groups of subjects for the H and N training conditions and the effects of H on training-induced performance increases were not explored. Hence, it is possible that the differences between experimental conditions may be due to genetic factors that influence the individual adaptive responses to training (24).

Perry et al (15) employed a crossover design to investigate the effects of inspiring H or N at the same exercise HR while training with a high intensity interval training protocol (HIIT) on VO2max and endurance performance at 90% VO2max. They reported that inspiring 60% O2 while performing 6 weeks of HIIT at 90% VO2max allowed subjects to maintain higher power outputs (~18 W or ~8%) than training in N. This resulted in a ~2-fold greater increase in endurance time to exhaustion at 90% of pre-training VO2max following training (H, 5.1 to 11.1 min; N, 5.6 to 8.5
min) despite similar increases in VO₂max for both groups. The authors proposed that the additional stimulus provided by the higher training power outputs in H might have increased skeletal muscle mitochondrial oxidative capacity to a greater extent than training in N.

Thus, it is possible that the enhanced endurance performance following training in H may be explained by greater skeletal muscle metabolic adaptations as compared to training in N. Therefore, the primary goal of this study was to determine whether the hyperoxia-induced increases in training power outputs would lead to greater increases than training in room air in the maximal activities of markers of mitochondrial oxidative potential; the TCA cycle enzyme citrate synthase, the beta-oxidation enzyme β-HAD, and the NADH malate-aspartate shuttle enzyme aspartate aminotransferase. Additionally, the relative increases in whole body VO₂max and cycling performance time at 90% of pre-training VO₂max following training in hyperoxia and normoxia were also measured to corroborate the skeletal muscle mitochondrial adaptations. The crossover design employed in this study required subjects to complete 6 wk of training in both normoxia and hyperoxia breathing conditions, separated by at least 6 wk of de-training. We employed a high intensity interval training protocol which required subjects to perform repeated short-term bouts of exercise at ~90% VO₂max during each workout.
METHODS

Subjects. Nine subjects (3 females and 6 males) volunteered to participate in this study. Their mean (+ SE) age, height and weight were 23 + 1 yr, 178 + 4 cm, 72.5 + 4.0 kg (Pre N) and 73.6 + 4.1 kg (Pre H) respectively. Their body weight was maintained throughout the entire study. The subjects were not involved in structured training regimens, but participated in some form of aerobic activity two to three times a week (eg. cycling, jogging). Subjects were also advised to refrain from taking any supplements other than multi-vitamins, and none were on medication. The experimental protocol and associated risks were explained both orally and in writing to all subjects before written consent was obtained. The study was approved by the Research Ethics Boards of the University of Guelph and McMaster University.

Study Design. Each subject completed two, 6 wk sessions of cycle ergometer interval training separated by at least 6 wk. In a single blind fashion, subjects were randomly assigned to either normoxia (N, room air – 21% O₂) or hyperoxia (H, 60% O₂) for the first 6 wk session. Following at least 6 wk of detraining, subjects completed a second 6 wk training session under the other condition. All subjects successfully de-trained in that they met the criteria of a return in VO₂max to their initial pre-training level after at least 6 weeks of inactivity. There were also no differences in time to exhaustion between both pre-trials.

VO₂max and Cycle to Exhaustion Protocols. Prior to each 6 wk training period, subjects completed a continuous incremental cycling test to exhaustion on an electromagnetically braked cycle ergometer (Lode Instrument, Groningen, The Netherlands) to determine pulmonary VO₂max using a metabolic measurement system (Vmax Series 229, Sensormedics Corporation, Yorba Linda, CA). All subjects practiced cycling to volitional exhaustion at 90% VO₂max on the Lode cycle to verify the power output and become familiarized with the procedure. At least 48 hr separated the VO₂max trial from the experimental performance trial. During this period, subjects refrained from
consumption of alcohol and caffeine as well as vigorous physical activity. When the subjects arrived at the laboratory, they rested on a bed while a catheter was inserted into an antecubital vein. A resting blood sample was taken and saline drip was used to maintain a patent line. For muscle biopsy sampling, an incision was made over the vastus lateralis muscle of one leg under local anesthesia (2% lidocaine, no epinephrine). With the subject on the bed, a resting muscle biopsy was taken and immediately frozen in liquid N₂ until analysis. Subjects then moved to the ergometer and cycled to volitional exhaustion at 90% VO₂max. Venous blood samples were also taken at 5 min and at exhaustion. Subjects conducted their first training session 1 wk after the 90% VO₂max exhaustion trial.

Post-training, the VO₂max test and exhaustion trial at 90% of the pre-training VO₂max were repeated 2 and 5 d after the final training session, respectively. Subjects were advised to consume the same diet 48 hours prior to each testing trial held before and after training in both H and N periods. This was verified by analyses of dietary records that subjects maintained in each experimental testing week. The three female subjects performed all experimental trials during the follicular phase of their menstrual cycles.

Training Protocol. Subjects trained on a cycle ergometer (Monark 894 E, Vasbro, Sweden) at a power output that elicited ~90% VO₂max 3 d/wk for each 6 wk period. Subjects completed ten exercise intervals lasting 4 min and separated by 2 min of rest in each training session. During the first and second exercise sessions of the first 6 wk period, the power output was adjusted to the highest intensity that each subject could tolerate for a complete set of ten intervals. Each subject’s HR reached a steady state during the final 2 min of intervals 6-10 and this HR averaged 180 bpm (95% of HRₘₐₓ) in both groups and was maintained throughout the remainder of both 6 wk sessions. The power output for each subject was increased in order to maintain training HR constant throughout the exercise intervals and sessions.
During the exercise training intervals, subjects inspired through a mouthpiece from a 1000 L reservoir bag that contained either 21% or 60% O₂. Subjects wore nose-clips throughout the work intervals. The subjects were blinded from the filling of the bag by a curtain. The FIO₂ was confirmed with a portable oxygen analyzer (102 series O₂ analyzer, Vacumed, Ventura, CA). During the rest intervals, subjects remained on the bike and inspired ambient room air and were also allowed to consume water or sports drink ad libitum.

Subjects were advised to maintain only light levels of aerobic physical exercise and refrain from lower body resistance training throughout the entire study (training and detraining sections). This was verified by activity logs that subjects maintained throughout the study. At the end of the entire study, subjects were asked to respond to a questionnaire indicating which condition they believed they were assigned to in each training period.

**Analyses.** A small piece of frozen wet muscle (6-10 mg) was removed under liquid N₂ for the spectrophotometric determination of CS, β-HAD and mitochondrial aspartate aminotransferase (m-AsAT) maximal activities (37°C) as described previously (3, 20). An aliquot of this muscle homogenate was then extracted with 0.5 M perchloric acid (HClO₄) containing 1 mM EDTA and neutralized with 2.2 M KHCO₃, and used for the enzymatic spectrophotometric determination of total creatine (3). CS, β-HAD and m-AsAT enzyme activities were normalized to the highest total creatine (Cr) content measured in the four biopsies from a given subject (one per experimental trial). There was no change in the total Cr content in samples obtained before and after training, as previously reported (4, 7).

Venous whole blood was collected in heparinized tubes, and 200 µl was immediately deproteinized with 1 ml of 0.6% (wt/vol) HClO₄. The supernatant was stored at -20°C and analyzed for lactate, glucose and glycerol (3). A second portion of blood was immediately centrifuged and
500 µl of the plasma supernatant was stored at -20°C and analyzed for free fatty acids (FFA) with a colorimetric assay (Wako NEFA C test kit, Wako Chemicals, Richmond, VA).

Statistics. A paired t-test was used to compare differences between Pre N and Pre H trials for performance time, VO₂max, body weight and enzyme activities in order to determine if subjects had successfully de-trained between the 6 week training sessions. A two-way ANOVA with repeated measures was used to test for differences in training power output and heart rate across the 6 wk of training in both N and H conditions, and between Pre and Post-training trials for performance time, VO₂max and muscle enzyme activities. In order to test for training effects in the blood measurements, a three-way ANOVA was used to compare resting and 5 min time points between Pre and Post trials in both N and H. A two-way ANOVA with repeated measures was used for the exhaustion blood measures since this time varied for each subject. When a significant F-ratio was obtained, post hoc analyses were completed using a Student-Newman-Keuls test. The level of significance was established at P < 0.05 for all statistics. Results are expressed as mean ± standard error (SE).
RESULTS

Training. Training heart rates were well maintained during 6 wk of training and were not different between N and H (Fig. 1A). Power outputs in H were greater than N by an average of ~17 W (~8%) throughout the training periods (Main condition effect, P<0.001, Fig. 1B). Power outputs increased (20-25%) to a similar extent during training in N and H (Main time effect, P<0.001). There were no differences in body mass prior to the two training periods. In spite of attempts to blind the subjects to the training condition, they were not blinded as eight out of nine subjects successfully identified which condition they were assigned to in each training period.

VO₂max and Cycling to Exhaustion. There were no statistical differences in the Pre values between N and H for VO₂max or cycling performance. This suggests that the subjects were successfully de-trained during the crossover between both training periods. VO₂max increased significantly (P < 0.01) in both N (12%) and H (11%) (Fig. 2). Time to exhaustion while cycling at 90% of the pre-training VO₂max increased ~2-fold following training in both N and H (P < 0.001) (Fig. 3). There was no statistical difference between the magnitude of increase in both N and H.

Muscle enzyme activities. The maximal activities of CS, β–HAD and m-AsAT were similar prior to training and increased to a similar extent following training in N (CS, 32%; β–HAD, 21%; m-AsAT, 26%) and H (CS, 30%; β–HAD, 23%; m-AsAT 21%) (P < 0.05) (Fig. 4).

Blood metabolites. All venous blood metabolites were similar in the two pre training cycle to exhaustion trials. Venous whole blood lactate, glucose, glycerol and plasma free fatty acids were similar before and after training at rest and following 5 min of exercise (Table 1). Venous blood lactate and glycerol were higher at exhaustion following training (P < 0.05). There were no statistical differences between N and H at any time points for any blood metabolites.
DISCUSSION

The present study demonstrated that inspiring 60% O$_2$ allowed subjects to exercise at a higher power output than in room air when training at the same HR. It was expected that the higher power outputs in H would provide an even greater stimulus for training adaptations. However, there were no differences between conditions in the magnitude of increases measured in mitochondrial enzyme capacities, VO$_2$max and cycle time to exhaustion at 90% of the pre-training VO$_2$max. This suggests that the higher training power output in H relative to N (8%) was not sufficient to induce greater training adaptations in H above those achieved in N.

Effect of Increased Training Power Outputs in Hyperoxia on the Adaptive Response to HIIT

Many studies have reported that inspiring a hyperoxic gas mixture improves performance during an acute bout of exercise (16, 25, 26). The present study demonstrated that inspiring H allowed all subjects to achieve a higher training power output for a given HR (~17 W or ~8%) as compared to N, and that this effect was sustained throughout 6 weeks of HIIT. This finding supports previous investigations (15, 17) using different training intensities and regimens. However, the present findings of similar increases in both mitochondrial and whole body oxidative capacity following H and N training does not agree with previous work by Ploutz-Snyder et al (17), where two groups of subjects trained for 5 weeks at ~70% VO$_2$max in either H or N. They reported that N resulted in greater increases in CS, β-HAD and cytochrome c oxidase activities relative to H despite similar increases in VO$_2$max for both groups. This study also demonstrated higher power outputs when inspiring H (~12% greater than N). The reason for the differences between the two studies and also between the two experimental conditions in the study by Ploutz-Snyder et al (17) is unclear, but may be due to the fact that they utilized two separate groups of subjects rather than adopting a crossover design. The latter approach would control for genetic influences on the individual responses to training (24).
It is possible that inspiring H actually dampens the metabolic response to training as compared to normoxic training and negates the increased stimulus resulting from the high training power outputs. For example, it has been shown that H can blunt the decrease in phosphocreatine utilization and reduce the accumulation of free ADP and P_i in the muscle while cycling at 70% VO_2max or plantar flexing at moderate to high intensities (8, 10, 21, 22). However, it is not known whether this occurs while cycling at 90% VO_2max as in the present study. Thus, H may enhance some adaptive responses via higher training power outputs but limit others by lessening metabolic stimuli.

The present study also reported an increase of ~100% in performance following training in both H and N conditions. These findings did not confirm a previous report of larger increases in high intensity exercise performance following training in H as compared to N (15), in spite of similar increases in VO_2max. It is difficult to reconcile the results of the two studies since both incorporated similar methodologies and randomly assigned subjects to each condition using a crossover design. Nevertheless, all of the experimental findings in the present study (VO_2max, mitochondrial enzyme measurements and performance) suggest that the greater training power outputs in H were not sufficient to induce greater adaptations relative to training in N. There were also no differences between H and N in venous blood lactate and other blood metabolite responses during the post-training cycle to exhaustion.

Higher training intensity while inspiring a hyperoxic gas

The exact mechanisms that allow subjects to cycle at a higher power output at a given HR while exercising in H are not completely understood. However, two potential mechanisms have been suggested; an effect on either skeletal muscle metabolism or on central motor output. Studies have demonstrated that inspiring 60% O_2 while cycling at 70% VO_2max (21), inspiring 100% O_2 while cycling at 65 or 90% VO_2max (5, 19) or performing repeated bouts of plantar flexion at ~60%
of maximum work rate while inspiring 100% O₂ (8) do not alter phosphocreatine utilization or oxidative metabolism at the onset of exercise. Our laboratory has recently shown that inspiring 60% O₂ during steady state exercise at 70% VO₂max resulted in a small attenuation in muscle glycogen utilization and phosphocreatine degradation, and total lactate production (21, 22). Similarly, Hogan et al (10) demonstrated that H slowed the depletion of phosphocreatine and accumulation of P₁ in calf muscle, particularly at near-maximal power outputs during a test of progressively increasing power outputs. Haseler et al (8) reported that H augmented the overall fall in muscle phosphocreatine during exercise at 60% of maximum work rate. These findings are supported by the observation that H increases myoglobin saturation during maximal exercise (18), indicating that intracellular PO₂ can be increased with H. Thus, it may be possible that H attenuates the development of muscle fatigue during repeated bouts at higher power outputs, such as those used in the present study (4 min intervals at 90% VO₂max), by reducing the reliance on phosphocreatine and glycolysis and enhancing oxidative phosphorylation. This may be a result of improved arterial and skeletal muscle oxygenation when inspiring H, leading to increased muscle oxygen uptake at either the onset of exercise or at steady state. However, attempts to measure increased oxygen uptake with H reported no effect during the first two minutes of moderate, heavy sub-maximal or supra-maximal intensities (11, 12, 27) or on leg VO₂ during steady state exercise at 70% VO₂max (22).

On the other hand, previous work has also suggested that central motor output is inversely related to inspired oxygen concentration (14, 23). This suggests that H may lower central neural drive for a given exercise intensity, which may delay the development of neuromuscular fatigue. In support of this, recent evidence suggests that H allows one to achieve a greater power output for a given central motor output. Amann et al (1) reported that inspiring 100% O₂ during a 5 km time trial resulted in a greater central motor output to the contracting quadriceps muscles, which allowed
subjects to maintain higher power outputs and achieve a faster time to complete the distance. While it is clear there is a strong relationship between arterial oxygen concentration and central motor output during exercise, it is uncertain whether this is due to a direct effect on the central nervous system or feedback from the periphery (1, 6). Additional research is required to fully elucidate the interactions between peripheral and central systems in order to explain the ability to exercise at a higher power output with H.

It is important to note that the subjects in the present study began in an untrained state (recreationally active) and trained for six weeks. The training stimulus in the N condition was intense and the higher training power output in H (8% greater than in N) did not appear to be sufficient to increase the already impressive metabolic and performance adaptations that occurred with N. However, it would be interesting to determine if H training would be of benefit for already trained, elite athletes who have reached a plateau in their adaptive response to training. For instance, would training twice a week in H permit athletes to exercise at higher power outputs and increase the adaptive responses and improve performance?

Conclusions. The present study employed a crossover design to examine the effects of 6 wk of high intensity interval training in room air or a hyperoxic (60% O₂) environment on VO₂max, endurance performance at 90% VO₂max and skeletal muscle mitochondrial enzymes in recreationally active subjects. The results demonstrated that training in room air produced robust increases in skeletal muscle oxidative enzyme activities, whole body VO₂max and endurance performance. Subjects were able to exercise at power outputs that were 8% higher than room air when training while breathing 60% O₂. However, this additional training stimulus was not sufficient to produce greater increases in mitochondrial enzyme activities, VO₂max or endurance performance above the already large adaptations afforded by training in room air.
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REFERENCES


1999.


Figure 1. Training heart rates (A) and power outputs (B) throughout 6 weeks of training in N (21% O₂) and H (60% O₂). Values are means + SE for 9 subjects. † P < 0.001, main effect for H being significantly higher than N. *Main effect of training, such that the power output for each week was higher than all prior weeks in each condition (P < 0.001).

Figure 2. VO₂max pre and post 6 weeks of high intensity interval training while breathing N (21% O₂) and H (60% O₂). Values are means + SE for 9 subjects. *P < 0.01, main effect of training such that Post > Pre.

Figure 3. Cycle time to exhaustion at 90% VO₂max pre and post 6 weeks of high intensity interval training while breathing N (21% O₂) and H (60% O₂). Values are means + SE for 9 subjects. *P < 0.001, main effect of training such that Post > Pre.

Figure 4. Maximal activities of CS (A), β-HAD (B) and m-AsAT (C) pre and post 6 weeks of high intensity interval training protocol while breathing N (21% O₂) and H (60% O₂). Values are means + SE for 9 subjects, w.w., wet wt. *P < 0.05, main effect of training such that Post > Pre.
Figure 1

A

![Graph A: Training HR (bpm)]

B

![Graph B: Training Power (W)]
Figure 2

VO₂max (L/min)

- Normoxia
- Hyperoxia

* Indicates significant difference.
Figure 4
Table 1. *Blood metabolites during a cycling trial to exhaustion at 90% VO\textsubscript{2} max pre and post 6 wks of training in hyperoxia (H) and normoxia (N).*

<table>
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<th>Metabolite</th>
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<th>Exh</th>
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<td>0.6 ± 0.1</td>
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<td>Lactate, mM</td>
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<tr>
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<td>Post H</td>
<td>0.6 ± 0.1</td>
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<tr>
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<td>Glucose, mM</td>
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<td>Post N</td>
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<td>Glycerol, µM</td>
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<tr>
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<tr>
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<td>Post N</td>
<td>0.17 ± 0.03</td>
<td>0.27 ± 0.07</td>
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</table>

Values are means ± SE for 9 subjects. Exh, exhaustion. *P < 0.05, greater than pre for same time point, main effect of training.