

THE EFFECT OF HIGH INTENSITY HYPOXIC TRAINING ON SEA-LEVEL SWIMMING PERFORMANCES

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Abstract

The principal objective of this study was to test the hypothesis that high intensity hypoxic training improves sea-level performances in well-trained swimmers more than the equivalent training in normoxia. **METHODS:** Sixteen well-trained collegiate and Masters swimmers (10 women, 6 men) completed a 5 week training program, consisting of three high intensity training sessions in a flume and supplemental low or moderate intensity sessions in a pool each week. Subjects were matched for gender, performance level and training history, and assigned to either hypoxic (HYPO; FIO₂=15.3%, simulated altitude of 2500m) or normoxic (NORM; FIO₂=20.9%) interval training in a randomized, double-blind, placebo controlled design. All low and moderate intensity training was done in a pool under normoxic conditions. The primary measures of performance were 100m and 400m freestyle time-trials. Laboratory outcomes included maximal O₂ uptake (VO₂max), anaerobic capacity (accumulated O₂ deficit), swimming economy, and hematological parameters. **RESULTS:** Significant (p=0.02 for 100m, p<0.001 for 400m) improvements were found in performance on both the 100m (NORM: -0.7 sec, {95% conf limits +0.2 to -1.7 sec}, -1.2%; HYPO: -0.8 sec {-0.1 to -1.5 sec}, -1.1%) and 400m freestyle (NORM: -3.6 sec {-1.8 to -5.5 sec}, -1.2%; HYPO: -5.3 sec {-2.3 to -8.3 sec}, -1.7%). These percent changes were nearly 5 fold greater than the mean of individual percent differences between two 100m time trials (0.26%) in a similar group of well-trained swimmers. There was no significant difference between groups for either distance (ANOVA interaction p=0.91 for 100m and p=0.36 for 400m). VO₂max was improved significantly (NORM: +0.16 l/min (±0.23), +6.4% (±8.1); HYPO: +0.11 l/min (±0.18), +4.2% (±7.0)). There was no significant difference between groups (P=0.58).

CONCLUSION: Five weeks of high intensity training in a flume improves sea-level swimming performances and VO_2max in well-trained swimmers. There was no significant, additive effect of hypoxic training (15.3% oxygen equivalent to a simulated altitude of 2500m) under the conditions of this experiment.

Key words : hypoxia, swimming, training, aerobic capacity, anaerobic capacity

The effect of altitude training is a function of both altitude acclimatization and hypoxic exercise (18). For endurance sports, the effect of acclimatization predominates and thus “living high – training low” has been demonstrated to be an effective altitude training strategy to improve sea level endurance performance (18,19,32), with hypoxic exercise impairing rather than enhancing the performance advantage of this approach.

However the effectiveness of hypoxic training without altitude acclimatization, referred to as intermittent hypoxic training, remains controversial. One elegant study suggested that hypoxic training could induce local adaptations at the muscle level (increased myoglobin and oxidative enzymes) that would be beneficial for endurance performance (36). More recently, hypoxic training has been shown to enhance the transcription of mRNA for hypoxia inducible factor 1-alpha, though the benefits of this process for exercise performance were not demonstrable (42). At a systemic level, most previous studies in this area focused on endurance athletes and training was predominantly aerobic in nature (24,35,41). However, the results of such training showed no significant effects on aerobic performance markers, such as maximal oxygen uptake (24,35,41).

In contrast, some previous reports (4,24,35) have suggested that intermittent hypoxic training might improve “anaerobic” performance or high intensity power output. This suggestion is supported by the physiological rationale that in hypoxia, an increased reliance on glycolytic metabolism is observed during submaximal exercise intensities (15,25), although maximal anaerobic capacity is apparently not affected (9). Moreover, several studies demonstrated that in order for anaerobic capacity to increase, high intensity training must be done (33,34).

We hypothesized that for sports of relatively high intensity/short duration requiring high rates of “anaerobic” metabolism to generate ATP independent of oxygen availability, the potential advantage of hypoxic exercise, if any, might be maximized. Swimming is a good example of such a sport, with the majority of competitive events lasting less than 2 minutes in duration. In this regard, Ogita and Tabata (27) found a 10% increase in anaerobic capacity, as measured by the accumulated oxygen deficit (AOD) after only 2 weeks of high intensity hypoxic training in 9 competitive Japanese male swimmers. However, no control group was included. Therefore, the question remains whether this improvement was an effect of the added hypoxic stimulus or solely an effect of the training itself.

In short, several studies suggest promising effects of intermittent hypoxic training for the improvement of performances of relatively high intensity/short duration; however, none of these studies generated conclusive evidence in support of this method, and suffered from several limitations. Therefore, the present study was designed to investigate the effect of high intensity intermittent hypoxic training on well-trained swimmers using a randomized, double-blind, placebo controlled design.

Methods

Subjects

Sixteen competitive swimmers (10 women, 6 men) were recruited from collegiate and Masters swimming teams. Five competed in US Olympic trials, and 11 competed at US National Championships. Twelve were honored as National Collegiate Athletic

Association (NCAA) and/or United States Masters Swimming (USMS)-All-Americans; one was a current multiple world record holder for masters events and one was a former American record holder. Three others were recreational. Individual descriptive characteristics are provided in table 1.

table 1 near here

All subjects were sea-level residents and gave their voluntary written informed consent to a protocol approved by the Institutional Review Boards of the University of Texas Southwestern Medical Center and Presbyterian Hospital of Dallas.

Study design

An outline of the study design is shown in Fig. 1.

Figure 1 near here

After familiarization with testing and training techniques, pool time trials and flume testing were conducted. Subjects were matched for gender, time trial performance and training history into pairs and assigned to either the control (living and training in normoxia ($FIO_2 = 20.9\% \pm 0.1$); $n = 8$; 3 men, 5 women) or experimental group (living in normoxia and high intensity training in hypoxia ($FIO_2 = 15.3\% \pm 0.1$ in N_2); $n = 8$; 3 men, 5 women) by balanced, stratified randomization. By this technique, within each matched pair, there was a 50-50 chance of being assigned to the control or experimental

group. All subjects participated in a 5-week training program, designed to include three high intensity training sessions and at least three low or moderate intensity sessions each week. High intensity training sessions were conducted in a flume, swimming the front crawl stroke only. During these sessions the swimmers wore a specially designed respiratory valve that fixed the inspiratory and expiratory tubes vertically parallel. The valves in the inspiratory and expiratory tubing were placed in an extension of the mouthpiece ensuring a minimal “dead space” of 30 ml (37). The inspiratory tube (length 1.65 m; diameter: 36 mm) was connected to a large reservoir which contained either the hypoxic or the normoxic gas mixture according to a double-blind, placebo controlled design. No member of the research team was aware of the blinding code until all the data were analyzed. The O₂ content of the gas inspired from the reservoir was checked frequently by an independent monitor for safety. All inspired gases were humidified before inhalation. The expired gasses passed through 0.74 m of 36 mm diameter tubing. The flume training sessions were carefully controlled and monitored by the research crew.

Training programs were based on those described by Tabata et al. (33,34) and designed to improve both anaerobic and aerobic capacities. They consisted of 10 bouts of 30-s exercise with 15-s rest, 5 bouts of 1-min exercise with 30-s rest and an additional 5 bouts of 30-s exercise with 15-s rest between each bout. Between sets the rest was 2-min. Subjects were vigorously encouraged to complete the sets; when they were able to complete more than 10, 5 and 5 bouts of each set respectively the flume speed was increased by 0.03 – 0.05 m/s.

All low and moderate intensity training was done in a pool under normoxic conditions. Programs were determined by the swimmers' coach. However, because matched subjects were members of the same collegiate or Masters team there was no statistically significant difference in pool training programs between groups.

Evaluation of performance

The primary outcome measure of this study was swimming performance, as measured both in the pool and in the flume. An outline of the testing schedule is included in Fig. 1.

TIME TRIALS: 100 AND 400M FREESTYLE. Pre- and Post-test time trials were conducted in an Olympic sized pool in Dallas between 4:30 – 7:00 PM. The time trials were conducted similar to normal swimming events, starting in groups of 4 - 6. Subjects had about 45 minutes rest between events, swimming the 100 m first. Starts were made from the starting blocks, using a whistle as the starting signal. Finish times were measured in duplicate by stopwatch, with one stopwatch functioning as back-up only. Day-to-day variability in 100m performance was determined in a separate series of time trials and was calculated as the mean \pm 95% confidence limits of the individual percentage difference between the two 100m times. These measurements were conducted 7 months after the training intervention using 13 representative swimmers (5 men, 8 women) from the same collegiate team and of the same level of performance, including 3 members of the primary research cohort. Subjects had 48 hours in between tests.

FLUME TESTING: A supramaximal test was used to determine both maximal oxygen uptake (VO_{2max}) and anaerobic capacity. After a 10 min warm-up, swimmers swam at a

predetermined swimming velocity (based on a familiarization test trial and the time-trial results) until exhaustion, which was designed to occur between 2 and 4 min. Exhaustion was defined as the inability to maintain pace with the water flow velocity (i.e. moving about 2 m back from the initial position). During this test the swimmers wore the same mask as described above, with the inspiratory tube in open connection with room air while the expiratory tube was ultimately connected to a series of Douglas bags.

MAXIMAL OXYGEN UPTAKE: Oxygen uptake was measured simultaneously with the Douglas-bag technique and an online system for breath-by-breath measurements. Douglas bags were considered as standard for all oxygen uptake measurements. Breath-by-breath data served as back up and was used for identification of steady states, plateaus and kinetic patterns in the VO_2 -time curve (data on oxygen uptake kinetics will be presented in a separate manuscript). The online system for breath-by-breath measurements consisted of 4 one-way valves to direct flow, two sample lines for measuring gas fractions and a turbine flow meter (VMM, Interface Associates) for measuring ventilation. Breath-by-breath data was stored on a computer and analyzed using customized software. The Douglas bag gas fractions were analyzed by a mass spectrometer (Marquette MGA 1100), that was calibrated twice a day and confirmed before each test, and used for both Douglas bags and the breath-by-breath system. Ventilatory volume was measured with either a Tissot spirometer or dry gas meter (Rayfield Air Meter 9200). Comparison between these devices immediately prior to the study confirmed their equivalence (slope = 1.0, intercept = 0.0, $r^2 = 1.0$). $\text{VO}_{2\text{max}}$ was defined as the highest VO_2 measured from at least a 30-s Douglas Bag. A plateau in VO_2 during supramaximal swimming was achieved in 100% of these tests documenting the

identification of VO_2max . The observation that VO_2 remained constant or decreased slightly during continued supramaximal exercise was defined as a plateau. In addition heart rate was monitored continuously (Polar CIC Inc., Port Washington NY).

ANAEROBIC POWER AND CAPACITY: Anaerobic power was estimated from the relationship between the total metabolic power output (P_{met}) and swimming velocity cubed (v^3) according to the method of Medbo et al. (22) adapted for swimming (26). Details of the derivation of this relationship are provided in Appendix 1. Oxygen uptake was measured at five 2-min submaximal swimming speeds at intensities ranging from 40 - 80% VO_2max , ensuring that subjects swam within the range of aerobic performance and were able to maintain normal swimming technique even at the lowest velocity. About 10 minutes after the last submaximal swim, subjects performed a supramaximal swim at an individually determined speed to exhaust the subject between 2 and 4 minutes as described above. P_{met} at that speed was predicted by extrapolation of the linear relationship between P_{met} and v^3 . The difference between P_{met} and aerobic power, as calculated from the measurement of oxygen uptake during swimming, gives the power generated by anaerobic metabolic processes. Power was transformed to its energy equivalent and expressed in $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and $\text{ml}\cdot\text{kg}(\text{lbm})^{-1}\cdot\text{min}^{-1}$. Fat Free mass (FFM) was calculated from skinfold measurements (Lange Skinfold Calipers: Cambridge Scientific Industries, INC. Cambridge MD), taken from 8 sites, using customized software employing the equations of Siri (30).

The same procedure, including the individual determination of swimming economy was performed both pre and post training. To allow comparison with previous reports in the literature (Ogita and Tabata, (27)), a secondary calculation of anaerobic

capacity was also made using only the economy measured before training. This approach does not take into account small but measurable day-to-day variations in swimming economy and is reported for comparison purposes only.

SUBMAXIMAL SWIMMING ECONOMY: The 2 minute submaximal swims were used to obtain a measure of swimming economy, defined as the P_{met} that is required to swim at a certain velocity. Swimming economy was determined as the slope of the regression relating P_{met} to v^3 .

Other laboratory measures

Hemoglobin ([Hb]) and hematocrit (HCt) were measured and used to monitor gross hematological trends during training. Blood samples were drawn via venipuncture from an antecubital vein. Subjects were seated in a chair, with the arm resting comfortably on a table at heart level. The whole procedure lasted between 1 and 2 minutes. [Hb] was determined using an Instrumentation Laboratories CO-oximeter. HCt was measured in duplicate via microcapillary centrifuge.

Evaluation of training

TRAINING LOGS: Each swimmer kept a detailed training logbook that included duration, distance and intensity of each workout in the pool. Training intensity was estimated by the subjects by giving each training session the qualification low, moderate or highly intense.

TRAINING CHARACTERIZATION: To determine precisely the demands of a typical flume training session, swimming velocity, VO_2 , V_e and heart rate were measured during

training. For the training sessions conducted in the pool, the training log information was used for training characterization.

Statistics

The primary statistical comparison was between the testing sessions before and after the training period. A two-way, repeated measures ANOVA (with main effects of time (pre vs post training) and treatment (HYPO vs NORM)) was used for analysis using SigmaStat 2.03 (SPSS Inc.). A separate two-way ANOVA with Swimmer Type (Collegiate vs Masters) and Treatment (NORM vs HYPO) was used to evaluate the difference between masters and collegiate athletes. The significance level for all comparisons was set at $P < 0.05$. When a significant effect was obtained, a Tukey Test was used for post hoc analysis. A multiple regression analysis was performed to reveal whether the change in performance was related to changes in the physiological variables measured. This analysis was performed according to a stepwise procedure, using forward selection with $VO_2\text{max}$ entered first. The variables included in this analysis were determined a priori based on the equation of the power balance for swimming (see Appendix), and included $VO_2\text{max}$, anaerobic capacity and swimming economy.

Data are presented in the tables as means \pm SD. For performance variables 95% confidence intervals are also included.

Results

Blood values and performance indexes are shown in table 2. At the start of the training period there were no significant differences between the groups in terms of time trial performance, VO_2max , anaerobic capacity or hematological parameters.

Table 2 near here

Training

All subjects completed a minimum of 12 flume training sessions (out of a maximum of 14 sessions). Three subjects missed 2 sessions, 6 subjects missed 1 session.

During both the exercise bouts of 30 sec and 1 min the hypoxic group trained with significantly ($p < 0.02$) reduced oxygen uptakes compared to the normoxic group (respectively 69.0 ($\pm 13.9\%$) of pre-training VO_2max for 30 sec and 75.7 ($\pm 9.2\%$) for 1 min sessions in hypoxia vs. 90.9 ($\pm 10.3\%$) and 93.8 ($\pm 11.5\%$) in normoxia). Furthermore, the power output during training, expressed as percentage of the maximal power output as determined on the pre-test was significantly ($p < 0.01$) lower, by 7.2 ($\pm 7.3\%$), in hypoxia (see table 3). These data indicate that for both groups, although the majority of energy was generated aerobically, a considerable part of the training was anaerobic in nature. However, the percentage of aerobic energy used to swim at the required speed was significantly lower in the hypoxic group compared to the normoxic group (65.2 (± 12.7) vs. 78.1 (± 11.3) and 74.1 (± 6.5) vs. 81.5 (± 8.7) for the 30s and 60s bouts respectively).

Table 3 near here

Both groups trained at very high ventilations (NORM: 105% (± 13.5); HYPO: 111% (± 13.4) of pre-test $V_{e\max}$) and heart rates (NORM: 96.4% (± 1.6); HYPO: 96.5% (± 3.0) of pre-test HR_{\max}), consistent with a maximal effort. No significant differences between groups were found. In summary, high intensity training under hypoxic conditions was accomplished with a substantially reduced oxygen flux, a slightly reduced power output, and consequently required a greater anaerobic contribution compared to normoxic training.

Training outside the flume was similar among the groups as determined from the training log information regarding frequency ($p=0.6$ by unpaired t-test), distance ($p=0.4$) and estimated intensity of the training (see table 4). All out-of-flume training sessions were rated as either 'low' or 'moderate' intensity: between 35 and 46% of the out-of-flume sessions/week were rated as low intensity training sessions with the remainder rated as moderate. Four subjects (2 matched pairs of Masters swimmers) were not able to complete the minimum of three training sessions a week in a pool outside the flume training because of logistical constraints. These subjects did complete all the flume training sessions.

table 4 near here

At the end of the study each subject was asked to guess which intervention they received in order to determine the effectiveness of the double-blind design. Subjects were

not able to guess correctly ($p=0.85$; by chi-square testing with Fisher exact test), indicating the successfulness of the design.

Response to training

HEMATOLOGICAL CHANGES: No significant changes in [Hb] or Hct were observed.

TIME TRIAL PERFORMANCE: Both groups improved significantly ($p=0.02$ for 100m, $p<0.001$ for 400m) in performance on both the 100m (NORM: -0.7 sec, {95% conf limits +0.2 to -1.7 sec}, -1.2%, {95% conf limits +0.6 to -3.0%}; HYPO: -0.8sec {95% conf limits -0.1 to -1.5 sec}, -1.1%, {95% conf limits +0.0 to -2.2%}) and 400m freestyle (NORM: -3.6 sec {95% conf limits -1.8 to -5.5 sec}, -1.2%, {95% conf limits -0.5 to -1.9%}; HYPO: -5.3 sec {95% conf limits -2.3 to -8.3 sec}, -1.7%, {95% conf limits -0.5 to -2.9%}). There was no significant difference between groups for either distance (ANOVA interaction $p=0.91$ for 100m and $p=0.36$ for 400m). This difference was nearly 5 fold greater than the mean of individual differences between two 100m time trials of -0.3% {95% conf limits +0.5 to -1.0%} (figure 2.2 A and B). No significant difference between Masters and collegiate swimmers was observed (ANOVA interaction $p=0.43$ for 100m and $p=0.49$ for 400m).

Figure 2 near here

VO₂MAX, V_EMAX AND HR_{MAX}: Both groups improved VO₂max significantly ($p=0.02$) (NORM: +0.16 l/min (± 0.23), +6.4% (± 8.1); HYPO: +0.11 l/min (± 0.18), +4.2% (± 7.0))

(figure 3.1 A and B). No significant difference between groups could be detected ($p=0.58$).

$V_{e_{max}}$ significantly ($p<0.001$) increased in both groups (NORM: 11.90 l/min, 13.6% (± 12.7); HYPO: 8.81l/min, 10.0% (± 7.6)). There was no significant difference between groups ($p=0.71$). No change in HR_{max} was observed.

ANAEROBIC POWER AND CAPACITY: All individual relationships between submaximal oxygen uptake and swimming velocity cubed were highly linear ($r=0.98$ (± 0.03)). There were no significant differences in anaerobic capacity between ($p=0.79$) or within ($p=0.80$) groups after training (figure 3.2 and table 2).

SUBMAXIMAL ECONOMY: The slopes of the regression lines relating P_{met} to v^3 were not significantly different ($p=0.21$) suggesting that submaximal swimming economy was not significantly changed either in hypoxia or normoxia.

PREDICTING SWIMMING PERFORMANCE; MULTIPLE REGRESSION: With the data of VO_{2max} , anaerobic capacity and swimming economy we were able to predict 100 and 400m times with an accuracy of respectively 72% (SEE: 4.1 sec) and 67% (SEE: 19.6 sec). However the change in performance that resulted from the training period could not be predicted with this simple linear 3-variable model ($p=0.26$).

Discussion

The present study showed that high intensity flume training significantly improved swimming performance in a pool over both 100 and 400m. However, this improvement was not enhanced by performing such training under hypoxic conditions.

This conclusion is strengthened by the carefully matched groups containing well-trained swimmers, and the randomized, double-blind, placebo controlled nature of the intervention. Therefore, our hypothesis that intermittent hypoxic training improves swimming performance more than training under normoxic conditions was rejected.

Previous work with intermittent hypoxic training

Several studies investigated the effects of intermittent hypoxic training in relatively untrained subjects (8,17,21). Together these studies showed clearly that intermittent hypoxic training has no beneficial effect over equivalent training at sea level in untrained individuals. In such subjects the effect of training seems to be predominant, overwhelming any additional effect of hypoxia. However, this result might be different in already well-trained athletes in whom the effect of training per se has been maximized.

A number of investigators have reported small studies in competitive athletes examining the effects of hypoxic exercise. For example, Terrados et al. (35) investigated the effect of intermittent hypoxic training in 8 elite cyclists, randomly assigned to either hypobaric hypoxia (2300m) or normoxia (sea level), and found no difference between groups for either work capacity or maximal power output. Similarly, Vallier et al. (41) found no significant differences in VO_2max or maximal power output in 5 elite triathletes following intermittent hypoxic training (4000m), though no control group was included. Most recently, Meeuwssen et al. (24) evaluated the efficacy of intermittent hypoxic training in a larger number of triathletes (n=16). Eight trained in a hypobaric chamber at a simulated altitude of 2500m, whereas 8 fitness-matched controls trained at sea level. Again, no significant differences between groups were found following the first post-test

conducted 2 days after the training period. However, a second test, conducted 9 days after the training period, revealed significant differences between groups in both maximal power output as measured during an incremental maximal cycle ergometer test, and mean and peak power as measured during a Wingate test. No significant differences in VO_2max between groups were found. Unfortunately, the training was not controlled during this intermediate period, limiting the strength of the conclusion.

In summary, previous work in both untrained subjects and well-trained athletes has not demonstrated convincingly an additive effect of hypoxia superimposed on endurance training for improvements in aerobic power, at least during whole body cycling exercise. Based on these limited data several other investigators (4,29,36,45) have speculated that benefits from hypoxic training, if any, are likely to be 'anaerobic' in nature. Unfortunately, these speculations have not been substantiated by experimental evidence. One factor contributing to this lack of support might be that previous studies focused on endurance athletes and training was predominantly aerobic in nature (ranging from 60% to 85% of VO_2max). Several studies in a number of different sports have indicated that for anaerobic capacity to increase, high intensity training must be done (33,34).

In this respect the study of Ogita and Tabata (27) is noteworthy. They studied the effect of 2 weeks of high intensity intermittent hypobaric hypoxic (3000m) training on anaerobic capacity and VO_2max in 9 collegiate swimmers. No significant change in VO_2max was observed. In contrast, anaerobic capacity significantly increased by 10%. However, this study suffered from several limitations. First, no control group was included that conducted similar training in normoxia. Therefore, it is impossible to

determine whether this improvement was an effect of the added hypoxic stimulus or an effect of the training itself. Second, no performance measure was included. Thus it remains unclear if the improvement in anaerobic capacity resulted in faster times in a swimming pool. Third, only 14 minutes of the 2 hour training load were specified, the other 106 minutes of each session remain unclear. Therefore, it is difficult to interpret what the contribution of the sets of high intensity exercise was to the 10% improvement in anaerobic capacity they observed. Finally, during the post-test, AOD was measured at higher water flow rates than during the pre-test. This is different from the present study and most other studies in which AOD was measured.

The present study overcomes most of these limitations by using carefully matched groups, and a randomized, double-blind, placebo controlled intervention. Ultimately the results from this study were consistent with previous observations, and extend this analysis to include swimming; although significant improvements in performance and $VO_2\text{max}$ with both normoxic and hypoxic training were found, no differences between groups could be detected. Moreover, in contrast to the study of Ogita and Tabata (27), no changes in either group were identified for anaerobic capacity.

Anaerobic capacity and training

In order to explain the discrepancy between the study of Ogita and Tabata (27) and the present study it is useful to examine the evidence supporting changes in anaerobic capacity with training. Careful review of previous studies raises serious questions regarding whether anaerobic capacity can actually be improved by training. In other words, is the trained muscle actually able to accumulate more oxygen debt? It is widely

reported that performances in which anaerobic metabolism has an important role can be improved as a result of training (14,23,33). However there are no data that unequivocally support the conclusion that these increases in performance are directly related to improvements in anaerobic metabolism itself. Increases in the number of type II muscle fibers (2) or changes in anaerobic enzyme activities (43,44) are highly unlikely with increased training intensity. High intensity training, as used by athletes, seems to result primarily in aerobic adaptations, because of an increased utilization of high rates of aerobic metabolism during training (10,44). Indeed, several studies indicate that high intensity training results in an increase in oxygen uptake and oxygen transport capacity (14,33), findings that support the results of the present study. It should be noted that the increase in VO_2max was not related to a difference in [Hb] as no change in [Hb] was observed. This lack of increase in [Hb] is likely to be due to the short hypoxic exposure time, and is in accordance to the findings of Emonson et al. (8) and Vallier et al. (41). Considering the specificity of the flume training, the relatively small muscle mass involved in swimming, and the time course of the training period we suggest that the observed increase in VO_2max , can be accounted for by neuromuscular adaptations, possibly accompanied with structural changes in the muscle fibers themselves. It is well documented that adaptations in mitochondrial enzyme activity, mitochondrial density and capillary density can rapidly occur as a result of training (1).

Another mechanism by which high intensity training is suggested to result in improvements in anaerobic performance may be improvements in cellular regulation (14,31), increasing the tolerance for products of anaerobic metabolism and delaying the

onset of fatigue. Indeed, hypoxia is often thought to increase this effect by putting an additional 'stress' on the body (29).

Ogita and Tabata (27) speculated that the 10% increase in AOD resulted from an increase in buffering capacity. Indeed, Mizuno et al. (25) and Saltin et al. (28) reported an increase in muscle buffering capacity with altitude acclimatization and related this increase to an improvement in anaerobic capacity. However, because the subjects in these studies both lived and trained at altitude, it is not clear whether the observed increase in muscle buffering capacity was an effect of acclimatization or hypoxic training per se. Evidence in support of the primacy of an acclimatization effect was provided by Gore et al. (13), who reported significant increases in muscle buffering capacity in well-trained triathletes after a 3 week period of "living high" (at a simulated altitude of 3000m) and "training low" in normoxia at sea level. To our knowledge no study has been reported that investigated the effect of IHT on the buffering capacity of human muscle.

It is also not clear whether there really is a greater "stress" or stimulus for adaptation during hypoxic exercise. Certainly, training in hypoxia "feels harder", with increased ventilation, heart rate and lactate during *submaximal* exercise intensities (15). However, in the present study the training protocol was such that all subjects, irrespective of the intervention, trained at a maximal or supramaximal intensity that enabled them to just barely complete the prescribed number of repetitions of each training set regardless of inspired gas mixture. This way both groups trained at similar (very high) relative intensities, which is supported by the heart rate and ventilation data, as well as by the fact that the subjects could not determine whether they trained in hypoxia or normoxia. The only protocol that demonstrated a potential advantage of hypoxic exercise was used by

Terrados et al. (36). By training a small muscle mass (one leg), they enabled the subjects to train at the same absolute work rate in hypoxia compared to normoxia. However, in the present study the hypoxic group trained at a significantly ($p < 0.01$) lower percentage of maximal power output as obtained during the pre-test. This was not an unexpected finding because several studies have found the self-selected power output to decrease when breathing hypoxic gas (5,17). Thus during sustained supramaximal exercise lasting at least one minute, work is reduced proportionate to the reduction in oxygen uptake since maximal anaerobic capacity is apparently not affected by hypoxia (9).

In short, the addition of a hypoxic stress to already maximal exercise intensities does not appear to enlarge the training effect. Hypoxia only shortens the time to exhaustion and/or causes a reduction in maximal power output, and leads to a reduced oxygen flux.

In the light of these concerns the finding of the present study that high intensity training (with or without hypoxia) did not result in improvements in anaerobic capacity in well-trained athletes might not be so surprising after all. Whenever increases in maximal oxygen deficit are measured, they may result from an increase in active muscle mass rather than by improvements in the muscle's capability to accumulate more oxygen debt. Hypoxic training results primarily in reduced power outputs and reduced oxygen flux and therefore does not appear to provide any advantage for a well-trained athlete.

Improvement with high intensity flume training

Regardless of the intervention, significant improvements in 100m and 400m pool swimming performance and VO_2max were found in both groups. Although no direct

measurement of peak power was included in the present study we have strong evidence for an improvement in peak power as a result of the flume training; changes in swimming performance and VO_2max occurred without changes in swimming economy and anaerobic capacity. This outcome is likely to be attributed to the unique characteristics of a swimming flume. A swimming flume does not allow for relaxation in any part of the stroke and makes swimmers heavily aware of all their movements in the water. Subjects reported feelings of exhaustion similar to those experienced in resistance training, suggesting that high intensity flume training may be a specific form of resistance training for swimmers, with consequent increases in maximal power output and swimming speed. We therefore speculate, but can not prove (considering that no control group performing similar training in a pool was included in the present study) that high intensity swim training conducted in a swimming flume might be useful in preparation for swimming events.

Limitations of the present study

A limitation of the present study that might have disguised differences between groups was the absence of a supervised lead-in period prior to the study (18). This ‘training camp effect’ could be especially important in the present study since the training method applied was new to all subjects and highly different from their normal training work. However, carefully matching the subjects created pairs that had very similar preparations at the start of the study, which may account for at least part of the training camp effect (32).

We also can not exclude the possibility that the amount of training done outside of the flume training sessions and the recovery between the sessions could have influenced the outcome of the present study. The collegiate swimmers swam 14 (± 4) hours a week for a weekly distance of 38.1 km (± 11.7) outside of the flume sessions. Therefore, we considered whether this additional training may have led to some overtraining, limiting the improvement from intermittent hypoxic training. However, both groups were well matched for out-of-flume training. Most importantly, the magnitude of improvement was similar in the masters' athletes who had much less out-of-flume training, thus making this possibility unlikely.

Furthermore, in the present study the effective hypoxic stimulus during each training session was only about 20 minutes. This may seem rather low compared to previous studies on intermittent hypoxic training, where the hypoxic stimulus was at least an hour each session. However, the studies of Terrados et al. (36) and Vogt et al. (42) demonstrated that a hypoxic stimulus of half an hour, 3-5 times a week, is enough to establish significant effects, at least at the muscle level. Besides, the combination of high intensity exercise and hypoxia considerably increases the severity of the local hypoxic stimulus because of exaggerated exercise induced hypoxemia (7).

Finally, it is possible that the accumulated oxygen deficit may not be sensitive enough to detect small differences in anaerobic capacity. Indeed, the measurement of anaerobic capacity using the AOD has a number of well recognized limitations; 1) some studies suggest that the relationship between exercise intensity and oxygen uptake may be non-linear (46); 2) assuming a linear relationship between exercise intensity and oxygen uptake, the total mechanical efficiency is considered to be constant, i.e. independent of

exercise intensity. The mechanical efficiency of the whole system is, however, determined by the efficiency of both the aerobic and anaerobic systems, these efficiencies may in fact not be equal (6,12); and 3) the anaerobic contribution to energy production at high submaximal exercise intensities is often not taken into account in the calculation of the energy demand during supramaximal exercise.

However, the high linearity of the PVO_2-v^3 relationships during both pre- ($r^2=0.97$) and post-tests ($r^2=0.99$) justifies the use of this relationship to estimate the AOD according to the method of Medbo et al. (22). Nevertheless, large variations in the change in anaerobic capacity were observed. The mean percentage change was -2.0% (± 51.0) while other training studies that measured AOD according to the method of Medbo et al (22) reported percentages change of $+28$ (33) and $+10\%$ (27) respectively. In contrast to the present study no standard deviation was given in these studies, thus the variability in the change of anaerobic capacity remains open to question. Moreover, these studies used the pre-test regression equations to determine AOD during both pre- and post-test. This way a possible source of variability (i.e. the day-to-day variability in submaximal VO_2 at a certain intensity (v^3)) is ignored, even when no significant changes in swimming economy are observed. Indeed, post-hoc analysis demonstrated that if we apply this strategy to the present data set, the mean percentage change was -2.1% , with a smaller standard deviation (± 32.9). Thus, the observation of a large variability might be disguised by the use of pre-test regression equations for analysis of both pre- and post-test data.

In conclusion, five weeks of high intensity training in a flume significantly improved sea-level swimming performances and VO_{2max} in well-trained swimmers.

There was no significant, additive effect of hypoxic training under the conditions of this controlled experiment.

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Appendix 1:

The equation for the power balance for swimming (40) is taken as starting point for determining the relationship between power output (P_o) and swimming velocity

$$P_o - (P_d + P_k) = dE/dt \quad (\text{equation 1})$$

In which P_d is the power to overcome drag, P_k is the power that is lost in giving water a kinetic energy change and dE/dt is the energy expenditure rate. Mechanical power is related to metabolic power according to (16)

$$P_o = P_{\text{met}} * e_m \quad (\text{equation 2})$$

in which P_{met} is the total metabolic power production and e_m is the total mechanical efficiency. Considering that the human body can generate ATP both by aerobic and anaerobic processes, the total metabolic power production equals

$$P_{\text{met}} = P_{\text{aer,met}} + P_{\text{an,met}} \quad (\text{equation 3})$$

Combining equations 1,2 and 3 gives

$$P_{\text{mech}} = P_{\text{aer,met}} e_{m,\text{aer}} + P_{\text{an,met}} e_{m,\text{an}} = P_d + P_k + dE/dt \quad (\text{equation 4})$$

According to Newton's second law there must be a balance between the power production and power losses when swimming at constant velocities, and thus

$$dE/dt = 0 \text{ and } P_o = P_d + P_k \quad (\text{equation 5})$$

Several studies demonstrated that active drag is related to the square of the swimming velocity (38,39). Consequently the power to overcome drag is related to the velocity cubed ($v \cdot v^2$) and a drag factor (K).

$$P_d = K v^3 \quad (\text{equation 6})$$

How P_k is related to velocity is not perfectly clear yet. Introducing the concept of propelling efficiency (e_p) (16,39) helps to solve this problem. Swimming at constant velocity

$$e_p = P_d/P_o = P_d/(P_d + P_k) \quad (\text{equation 7})$$

Assuming e_p to be independent of swimming velocity (which according to our data seems at least reasonable for top-level swimmers), P_o can be calculated according to

$$P_o = P_d/e_p = K * v^3/e_p = K/e_p * v^3 \quad (\text{equation 8})$$

K/e_p is constant and thus P_o is related to velocity cubed. Evidence for this relationship is experimentally generated by Toussaint et al. (39). Combing equation 2 and 8 gives the expression for metabolic energy expenditure

$$P_{\text{met}} = K v^3 / e_m \cdot e_p \quad (\text{equation 9})$$

assuming e_m to be constant the linear relationship between P_{met} and v^3 is established. This relationship is experimentally supported by Toussaint et al. (39) and Ogita et al. (26).

Considering that at low exercise intensities the total mechanical power output is primarily generated by means of aerobic metabolic processes and the measurement of oxygen uptake is a reliable measure of aerobic energy production, the relationship can be transformed into

$$VO_2 = K v^3 \quad (\text{equation 10})$$

which can be transformed to its power equivalent according to (11)

$$P_{VO_2} = 1/60 * 10^3 * [4.1868 * (4.047 + RER)] * VO_2 \quad (\text{equation 11})$$

Where RER is the respiratory exchange ratio.

This linear relationship between P_{VO_2} and v^3 only holds for steady state exercises within the range of aerobic performance (39), i.e. below 80% of $VO_{2\text{max}}$ (1). Now, by measuring VO_2 at several submaximal intensities the linear relationship can be

experimentally established and used for extrapolation to higher exercise intensities in order to determine anaerobic capacity.

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Table 1. *Subject characteristics*

Subject	C/M	Gender	Specialisation	Level	PB	RB* (date)	Age	Height	Body mass	%fat	VO ₂ max
N1	C	F	free, fly	OT, NCAA, Nat.	59.4	-	20	173.5	70.5	17.1	64.6
N2	C	F	IM	OT, NCAA, Nat.	62.5	62.5 ('01)	19	160.3	54.7	12.9	58.4
N3	C	F	breast	Nat.	60.0	-	21	177.7	66.2	13.9	44.1
N4	M	F	breast, free	USMS	-	-	27	166.8	68.2	17.1	54.7
N5	M	M	free	NCAA, OT, Nat. USMS	51.4	51.7 ('01)	30	187.5	81.9	8.5	48.4
N6	M	M	free, fly	NCAA, Nat., Int. USMS, WR Masters	53	-	38	185.8	87.2	13.5	47.8
N7	M	M	free	USMS, Nat.	54	62.3 ('00)	45	176.5	80.6	12.3	40.2
N8	M	F	free	Recr.	-	75 ('01)	31	166	60.5	17.1	43.2
Average							28.9	174.3	71.2	14.1	50.2

Subject	C/M	Gender	Specialisation	Level	PB	RB* (date)	Age	Height	Body mass	%fat	VO ₂ max
H1	C	F	free, fly	Nat.	58.4	59.7 ('01)	19	165.8	63	12.3	53.7
H2	C	F	free	Nat.	60.2	61.2 ('01)	19	176	68.7	18.2	41.5
H3	C	F	breast	NCAA, Nat.	61.7	63.5 ('01)	21	175.3	65	13.8	45.9
H4	M	F	back, fly	NCAA, Nat., Int., AR, OT, USMS	59.2	-	24	181	85.5	15.7	46.3
H5	M	M	back, free	OT, Nat.	52	-	27	185.9	77.3	8.5	56.3
H6	M	M	IM, back	USMS	57	-	39	171.1	80.6	19.1	45.8
H7	M	M	free	Recr.	-	-	54	167.8	75.2	23.8	51.7
H8	M	F	free	Recr.	-	85 ('01)	27	173	62.7	14	41.4
Average							28.8	174.5	72.3	15.7	47.8

N_n, normoxia subject n; H_n, hypoxia subject n; C/M, collegiate or master; PB, personal best; RB, recent best; *, i.e. best time on 100m free style within a year from the start of the study (year between brackets); note that some elite specialists do not have a recent best time in the 100m freestyle; VO₂max, maximal oxygen uptake in ml·kg(lbm)⁻¹·min⁻¹ as measured during the pre-test; AR, American Record; Int., International Championships level; Nat., National Championship level; NCAA, NCAA-All-American; OT, Olympic Trials; Recr., Recreational level; USMS, USMS-All-American; WR Masters, World Record Masters.

Table 2. Hematological parameters and performance indexes

100m time (s)				400m time (s)		
	Pre	Post	%change {95% conf. limits}	Pre	Post	%change {95% conf. limits}
Group	65.39 (\pm 7.64)	64.62 (\pm 7.33)*	-1.2 {-0.3 to -2.1}	305.91 (\pm 33.19)	301.43 (\pm 32.09)*	-1.5 {-0.9 to -2.0}
NORM	64.36 (\pm 5.23)	63.62 (\pm 5.76)	-1.2 {+0.6 to -3.0}	301.08 (\pm 27.12)	297.46 (\pm 26.70)*	-1.2 {-0.5 to -1.9}
HYPO	66.43 (\pm 9.76)	65.62 (\pm 8.93)	-1.1 {+0.0 to -2.2}	310.74 (\pm 39.65)	305.41 (\pm 38.18)*	-1.7 {-0.5 to -2.9}

VO ₂ max (l/min)				Anacap (mlkg(lbm) ⁻¹)		
	Pre	Post	%change	Pre	Post	%change
Group	2.99 (\pm 0.56)	3.12 (\pm 0.50)*	+5.3 (\pm 7.4)	31.06 (\pm 11.87)	27.95 (\pm 13.30)	-2.0 (\pm 51.0)
NORM	3.05 (\pm 0.58)	3.22 (\pm 0.48)*	+6.4 (\pm 8.1)	32.26 (\pm 8.16)	28.16 (\pm 14.33)	-12.1 (\pm 35.2)
HYPO	2.92 (\pm 0.57)	3.03 (\pm 0.53)	+4.2 (\pm 7.0)	29.86 (\pm 15.23)	27.75 (\pm 13.16)	+8.1 (\pm 64.1)

HRmax (bpm)		Vemax (BTSP) (l/min)		Economy (W/v ⁻³)		
	Pre†	Post†	Pre	Post	Pre	Post
Group	184 (\pm 7)	187 (\pm 5)	97.74 (\pm 20.63)	108.09 (\pm 19.94)*	460.5 (\pm 110.4)	435.9 (\pm 92.6)
NORM	183 (\pm 6)	186 (\pm 2)	98.91 (\pm 21.06)	110.80 (\pm 20.42)*	463.6 (\pm 89.9)	433.7 (\pm 84.2)
HYPO	186 (\pm 8)	188 (\pm 6)	96.58 (\pm 21.58)	105.38 (\pm 20.45)*	457.3 (\pm 134.2)	438.1 (\pm 106.2)

Hemoglobin (g/dl)			Hematocrit (%)	
	Pre	Post	Pre	Post
Group	14.5 (\pm 1.3)	14.45 (\pm 1.1)	40 (\pm 2.5)	39.5 (\pm 2.8)
NORM	14.4 (\pm 1.3)	14.2 (\pm 0.9)	39.4 (\pm 2.9)	39.5 (\pm 3.2)
HYPO	14.5 (\pm 1.4)	14.7 (\pm 1.2)	40.5 (\pm 2.1)	39.5 (\pm 2.7)

Values are means (\pm SD); %change, mean of the individual percentage differences; VO₂max, maximal oxygen uptake; Anacap, anaerobic capacity; HRmax, maximal heart rate; Vemax, maximal ventilation.

* Significant F statistic (P<0.05) (Tukey test).

† Due to technical problems heart rate data represents 13 (NORM: 7; HYPO: 6) subjects for pre testing and only 5 (NORM: 3; HYPO: 2) for post testing.

Table 3. *Flume training characteristics*

	%HRmax	%Vemax	%VO ₂ max		%Pmax		%Aer	
	training	training	30sec	60sec	30sec	60sec	30sec	60sec
NORM	96.4 (± 1.6)	105% (± 13.5)	90.8 (± 10.3)	93.8 (± 11.5)	109.1 (± 6.3)	99.9 (± 4.8)	78.1 (± 11.3)	81.5 (± 8.7)
HYPO	96.5 (± 3.0)	111% (± 13.4)	69.0 (± 13.9)*	75.7 (± 9.1)*	102.1 (± 3.2)*	92.5 (± 2.5)*	65.2 (± 12.7)*	74.1 (± 6.5)

Values are means (± SD); %HRmax, percentage of maximal heart rate; %Vemax, percentage of maximal ventilation; %VO₂max, percentage of maximal oxygen uptake; and %Pmax, exercise intensity related to maximal power output as obtained during the pre-test; %Aer, percentage of total energy that is generated by aerobic sources. Both %Hrmax and %Vemax were averaged over both training sessions (training); data for %VO₂max, %Pmax, and %Aer were divided into 30 second (30sec) and 60 second (60sec) intervals.

* significant t statistic (P<0.05) (unpaired t-test).

Table 4. *Out-of-flume training characteristics*

	Week 1				Week 2				Week 3			
	Normoxia		Hypoxia		Normoxia		Hypoxia		Normoxia		Hypoxia	
	M	C	M	C	M	C	M	C	M	C	M	C
Frequency	1.3	6.7	1.5	7.3	1.8	7.3	2.3	8	2	7.7	2	8.3
	(±1.3)	(±0.6)	(±1.7)	(±0.6)	(±1.5)	(±1.2)	(±1.2)	(±2)	(±1.2)	(±0.6)	(±1.4)	(±1.5)
Distance	3.7	35.8	4.2	39.8	4.7	39.6	6.3	44.7	5.1	44.3	5.4	46.7
(km)	(±4.1)	(±6.9)	(±4.9)	(±5.9)	(±4.5)	(±8.9)	(±3.4)	(±11.8)	(±3.4)	(±4.1)	(±3.9)	(±7.9)
Intensity	Low - moderate		Low - moderate		Low - moderate		Low - moderate		Low - moderate		Low - moderate	

	Week 4				Week 5			
	Normoxia		Hypoxia		Normoxia		Hypoxia	
	M	C	M	C	M	C	M	C
Frequency	1.8	5.7	1.5	6.3	1.3	5.7	1.3	5.3
	(±1.5)	(±2)	(±1.7)	(±2.3)	(±1)	(±2.3)	(±1.2)	(±3.1)
Distance	4.4	34.6	4.1	38.1	2.5	33.2	3.7	31.4
(km)	(±4.2)	(±6.1)	(±4.8)	(17.7)	(±1.9)	(±14.4)	(±3.2)	(±19.6)
Intensity	Low - moderate		Low - moderate		Low - moderate		Low - moderate	

Values are means (\pm SD); M, Masters swimmer (n=5; training log data are unavailable for 1 matched pair of Masters swimmers, so data in table are calculated from n=4 Masters athletes); C, Collegiate swimmer (n=3)

Figure legends:

Fig 1. The study consisted of the following phases: A) initial 100 and 400m time trials; B) single initial session for familiarization with testing and training procedures; C) initial laboratory testing; D) 5 wk training period with swimmers divided into 2 groups by balanced randomization; E) repeat time trails; F) repeat series of laboratory testing.

Fig 2. Percentage change in 400m (Fig. 2.1) and 100m (Fig. 2.2) time trial performances for each individual subject training in A) normoxia, B) hypoxia, and absolute changes for all subjects together (C), before and after the training period. Similar symbols are used for each pair of matched subjects. Parallel dashed lines, 95% confidence interval. Parallel dotted lines, mean \pm 95% confidence interval for individual differences between two 100m time trials in a group of similar athletes, including 3 of the current cohort. *, significant F statistic ($P < 0.05$) (RM ANOVA).

Fig 3. Percentage change in VO₂max (Fig. 3.1) and anaerobic capacity (Fig. 3.2) for each individual subject training in A) normoxia, B) hypoxia, and absolute changes for all subjects together (C), before and after the training period. Horizontal solid line with vertical bar, mean \pm standard deviation. Horizontal dashed line, zero level. *, significant F statistic ($P < 0.05$) (RM ANOVA).

Figure 1:

Figure 1

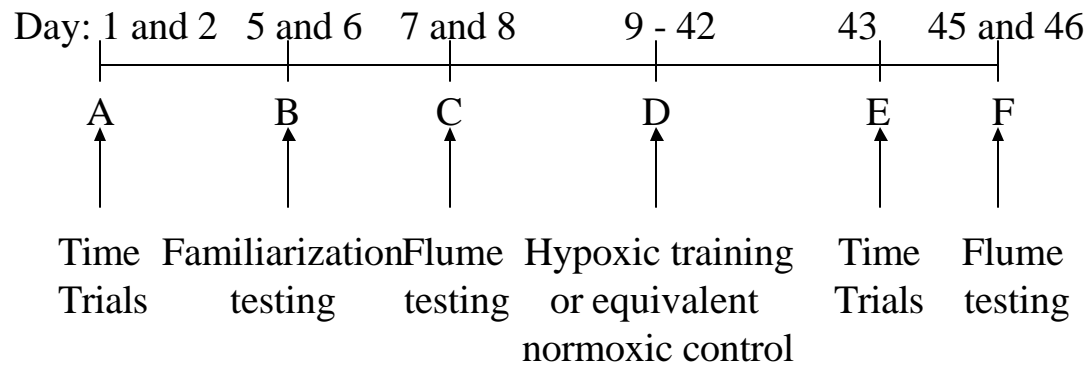


Figure 2:

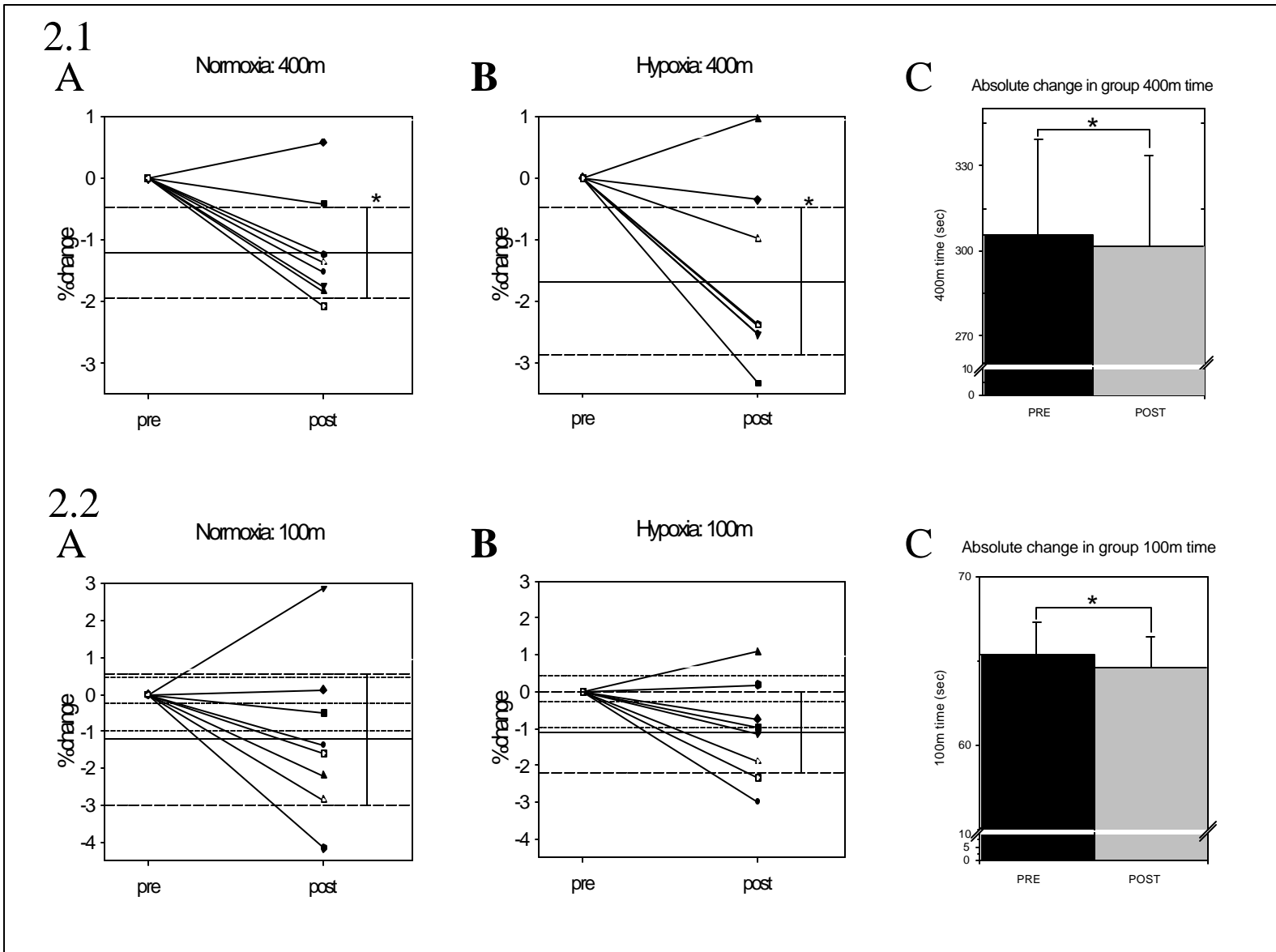


Figure 3:

