Altitude Training in Elite Swimmers for Sea Level Performance
(Altitude Project)

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ABSTRACT

Introduction. This controlled nonrandomized parallel groups trial investigated the effects on performance, \( \dot{V}O_2 \) and hemoglobin mass (tHb\text{mass}) of 4 preparatory in-season training interventions: living and training at moderate altitude for 3 and 4 weeks (Hi-Hi3, Hi-Hi), living high and training high and low (Hi-HiLo, 4 weeks), and living and training at sea level (SL) (Lo-Lo, 4 weeks). Methods. From 61 elite swimmers, 54 met all inclusion criteria and completed time trials over 50 and 400 m crawl (TT50, TT400), and 100 (sprinters) or 200 m (non-sprinters) at best stroke (TT100/TT200). \( \dot{V}O_{2\max} \) and heart rate were measured with an incremental 4x200-m test. Training load was estimated using TRIMPc and session RPE. Initial measures (PRE) were repeated immediately (POST) and once weekly on return to SL (PostW1 to PostW4). tHb\text{mass} was measured in duplicate at PRE and once weekly during the camp with CO rebreathing. Effects were analyzed using mixed linear modeling. Results. TT100 or TT200 was worse or unchanged immediately POST, but improved by \( \sim 3.5\% \) regardless of living or training at SL or altitude following at least 1 week of sea level recovery. Hi-HiLo achieved a greater improvement two (5.3\%) and four weeks (6.3\%) after the camp. Hi-HiLo also improved more in TT400 and TT50 two (4.2\% and 5.2\%, respectively) and four weeks (4.7\% and 5.5\%) from return. This performance improvement was not linked linearly to changes in \( \dot{V}O_{2\max} \) or tHb\text{mass}.

Conclusion. A well-implemented 3- or 4-week training camp may impair performance immediately, but clearly improves performance even in elite swimmers after a period of SL recovery. Hi-HiLo for 4 weeks improves performance in swimming above and beyond altitude and SL controls, through complex mechanisms involving altitude living and SL training effects.

Key words: HYPOXIA, SWIMMING, HEMOGLOBIN MASS, ELITE ATHLETES, OXYGEN UPTAKE
INTRODUCTION

Altitude training (AT) has been a matter of extensive research for half a century and despite some skeptical views (23), it still plays an important role in the preparation of athletes in many countries (37, 47). The global theoretical concept behind this practice is the independent and combined effects of the physiological processes of acclimatization to chronic hypoxia and those derived from training under the additional stress imposed by exercising in a hypoxic environment.

The classical approach (“live high-train high”, Hi-Hi), used since the late 1960’s, involves sea level (SL) resident athletes who travel to, and subsequently live and train at moderate altitude, typically 1,800–2,500 m for a period of 2 to 4 weeks; a similar approach is also practiced by athletes who reside full time at altitude. Despite being used by very many elite swimmers and coaches, there is a remarkable lack of controlled studies on AT in swimming in the scientific literature, and there is no clear evidence that training at natural altitude enhances performance more than training at SL (31).

In a series of studies published in the 1990’s, Levine and Stray-Gundersen provided sound evidence that the “live high-train low” (Hi-Lo) strategy can improve 3-5,000-m running performance in US collegiate athletes (22). This approach was modified to limit the low altitude training sessions to only high intensity workouts, and was subsequently termed “live high–train high and low” (41). Although there was substantial individual variability in the response (6), the improvement in running performance was associated with an increase in red cell mass, the subsequent increase in $\dot{V}O_{2_{max}}$ (the high altitude effect) and the maintenance of high-intensity training velocities and oxygen flux to the muscles (the low altitude effect) (41). This paradigm has now been confirmed repeatedly by multiple investigators (46), in elite endurance athletes
performing different sports including running (41), orienteering (46), and cycling (15). However these studies are difficult to compare with each other directly given the many differences in experimental design including type of athletes being compared, performance measures, control of confounding variables, placebo/nocebo effects, and most importantly, the nature of altitude exposure (i.e., true exposure to terrestrial altitude, or night time exposure to normobaric hypoxic gas) (17). Even though in the last decade the Hi-Lo approach has largely supplanted classical AT in the scientific literature and among many endurance athletes (37), no studies have been conducted using the Hi-Lo strategy in swimmers. Another key unanswered question which is rarely addressed concerns the proper timing of return to SL prior to competition (4).

In view of the disconnect between research evidence and practical use of AT, particularly in elite swimmers, an international group of investigators convened to conduct an international multidisciplinary and collaborative research project (The Altitude Project) to examine the impact of different current AT strategies on performance, technique, and health status of elite swimmers. This article is focused on performance, oxygen uptake kinetics and hemoglobin mass, and will be accompanied by other publications dealing with other aims of this project.

Consequently, this study aimed 1) to test the hypothesis that living at moderate altitude (2,230 m) and training both at moderate and at lower altitude for four weeks (Hi-HiLo) improves SL swimming performance more than living and training at altitude (classical terrestrial AT) for 3 (Hi-Hi3) or 4 weeks (Hi-Hi), or than living and training at low altitude (conventional Lo-Lo sea-level training); 2) to elucidate whether the adaptive mechanisms conform with the “erythropoietic paradigm” (i.e., are mainly hematologic in nature, via the activation of erythropoiesis by induced hypoxia, with subsequent increase in $\dot{V}O_{2\text{max}}$); and 3) to quantify the
eventual effect of the different interventions on performance on return to SL and to track changes during a lengthy period of 4 weeks without concurrent tapering.

METHODS

Study Design

The study was designed as a controlled, nonrandomized, four parallel groups trial, comparing changes in swimming performance, maximal oxygen uptake ($\dot{V}O_{2max}$), oxygen kinetics parameters and total hemoglobin mass ($tHb_{mass}$) after an experimental intervention consisting of training camps in four different conditions: 1) living and training at moderate altitude (2,320 m above SL) for 4 weeks (Hi-Hi); 2) identical intervention for 3 weeks (Hi-Hi3); 3) living at altitude (2,320 m) and training at both moderate and low altitude (690 m) for 4 weeks (Hi-HiLo); and 4) living and training at near SL (190 or 655 m) for 4 weeks (Lo-Lo).

All athletes were studied at the same point in their competitive season as noted below. At the onset of the study, coaches were instructed to conduct a lead-in training program the week before initial testing, in which training load was reduced to avoid excessive fatigue prior to baseline swimming assessments. Then all swimmers and their coaches travelled to Sabadell (190 m) or Madrid (655 m), Spain, where they stayed for 3-5 days for baseline testing. Next, all swimmers allocated to the AT groups travelled to the High Altitude Training Center at Sierra Nevada (2,320 m), Spain, where they lived for 3 or 4 weeks. The two Lo-Lo sub-samples lived and trained in Sabadell ($n = 4$) and the High Performance Center (CAR) at Madrid ($n = 7$). One of the Hi-Hi3 subgroups ($n = 6$) was tested in Granada, Spain (690 m). In all cases, baseline and final testing were conducted at the same location and facility. To minimize instruction bias (placebo/nocebo effects), the group allocation was performed according to previous agreement.
with the team head coaches as to their preferred mode of intervention (i.e., Hi-Hi or Hi-HiLo altitude camps for 3 or 4 weeks or SL camps), and coach and athlete acceptance of its characteristics and procedures. To minimize bias from differences in individual team training approaches, each experimental group was composed of swimmers of at least two different teams and nationalities.

To evaluate the effects on performance and physiological parameters, before the experimental intervention (PRE) all subjects took part in three testing sessions with the following content: 1) two swimming time trials over 50 m (TT50), and 100 or 200 m (TT100 or TT200), 2) one swimming time trial over 400 m (TT400), and 3) one 4x200 m incremental swimming test (T4x200). All measurements were repeated 1-2 days (POST), and 1 week (PostW1), 2 (PostW2), 3 (PostW3), and 4 (PostW4) weeks after completion of the training camp. This way both the immediate effects and the off response of the intervention could be assessed. $tHb_{mass}$ was measured in duplicate (Pre1, Pre2) on two separate days, and the average was adopted as a baseline value (PRE). Measurements were repeated once at the first (W1), second (W2), third (W3), and fourth (W4) week during the intervention period in all altitude groups, and at W4 in the sea-level training group (Lo-Lo). An outline of the study design and testing schedule is shown in Figure 1.

Subjects

Sixty-one swimmers, 34 females (F) and 27 males (M), were recruited as subjects for the present study. Sample size was calculated based on a potential increase of $\dot{V}O_{2 \text{max}}$ of 5% (mean ± SD: 3 ± 3.2 ml·kg$^{-1}$·min$^{-1}$) based on previous studies (22), requiring 11 athletes per intervention group ($\beta = 0.80$, $\alpha = 0.05$), thus a total of 44 athletes. The subjects were swimmers from eight
countries (Australia, Brazil, China, Great Britain, Netherlands, Slovenia, Spain, and Tunis). Selection criteria were to have competed internationally during the previous season and/or being pre-selected as a member of their National and/or Olympic teams. Exclusion criteria included residence at altitude greater than 1,000 m in the previous six months, recent illness or injuries preventing normal training and racing, and having low ferritin levels at the beginning of the study (<20 ng·ml$^{-1}$ for females, <30 ng·ml$^{-1}$ for males). All subjects—and their legal guardians in swimmers under 18 years of age—gave their informed written consent to the study that had received approval from the Ethics Committee for Clinical Sport Research of Catalonia. To quantify the competitive level of the subjects, the FINA Point Scoring (FPS) system was used, and a point score (range 0-1,100) was ascribed to each swimmer according to her/his best time in her/his main event, scaled up or down from 1,000 points based on the global 2012 fastest performance in each event. The swimmers were categorized as sprinters or non-sprinters—i.e. specialists in 50-100 m or 200 to 1500 m, respectively—according to their best event. Before data analysis, 1 F was excluded because of low baseline ferritin levels, 1 M and 1 F subjects were excluded from the training camp by their coach, 1 M and 1 F swimmers because they could not perform the POST tests, and 1 M and 1 F subjects because they had to withdraw participation for personal reasons. 54 subjects, 30 F and 24 M, successfully completed the intervention protocol (Table 1). After final group allocation, there were no significant differences among the four experimental groups in terms of performance level, body height or mass, $\dot{V}O_{2\max}$, or tHb$_{mass}$. The swimmers in the Lo-Lo group (19.6 ± 2.9 years) were younger than those in the Hi-HiLo group (mean ± SD: 23.7 ± 3.5 years) ($P = 0.02$) but there were no differences among the other groups.
Evaluation of Performance

Swimming Time Trials. The primary outcome measure of this study was swimming performance, as measured in time trials over three distances: 50 m front crawl (TT50), 400 m front crawl (TT400), and 100 m or 200 m at personal best stroke for sprinters and non-sprinters, respectively (TT100 or TT200). All tests were conducted at the same 50-m indoor pool (temperature: water 26-27°C, air 27-28°C). After a standard competition warm-up swimmers were instructed to achieve the best time possible on each trial, in which they swam alone. Start was given as in a competition and time was manually recorded to the nearest 0.01 s by three experienced timers, one of them the swimmer’s personal coach. The median values were used for analysis. Heart rate (HR) was continuously monitored (CardioSwim, Freelap, Switzerland) and $\dot{V}o_2$ was measured immediately post-exercise for 3 min. The testing schedule at PRE and POST comprised two consecutive days. On day 1, two sessions were performed: T4x200 was administered in the morning session, and TT100 or TT200 in the afternoon session. On day 2, T4x200 was repeated in the morning, and TT50 and TT400 were performed in the afternoon, allowing a recovery period of at least 60 min in between. The testing schedule at POST to PostW4 comprised two afternoon sessions following a light morning training session. On day 1, TT50 and TT400 were administered. At day 2, TT100 or TT200 was performed.

Pool Evaluation. The secondary outcome measure of the study was $\dot{V}o_{2\text{max}}$ measured with a 4x200-m incremental swimming test (T4x200) at the same 50-m indoor pool. An incremental swimming exercise protocol based on a previous protocol in the swimming flume (34) was used. After a ~30 min warm-up, subjects swam three times 200 m front crawl at paced speeds (0.9, 1.0, and 1.1 m·s$^{-1}$ for women, and 1.0, 1.1, and 1.2 m·s$^{-1}$ for men). Swimming pace was controlled...
using a computer programmed to produce audio signals at set intervals. An assistant outside the pool entrained his walking pace to the audio signals, and to marker cones placed at 5-m intervals at the poolside. The assistant carried a pole with an attached thin nylon line and a red ribbon at the end. Subjects were instructed to follow closely the red ribbon moving in front and below the water surface. Swimming laps were also timed each 50 m. Following the submaximal paced swims (results reported separately), after 10 min of passive recovery, subjects completed an all-out 200 m front crawl swim to determine $\dot{V}O_{2\text{max}}$.

Oxygen Uptake Kinetics and $\dot{V}O_{2\text{max}}$. $\dot{V}O_2$ was measured using a telemetric portable gas analyzer (K4 b2, Cosmed, Italy) that was held suspended over the water by an assistant following the swimmer along the pool with minimal intended interference with her or his swimming movements. This equipment was connected to the swimmer by a low resistance respiratory snorkel and valve system (32). Pulmonary $\dot{V}O_2$ values during the T4x200 swimming test were measured breath-by-breath and then time-aligned to the start of exercise and plotted against time. No smoothing procedures were applied to avoid distortion of the underlying signal at the transient phase. $\dot{V}O_2$ data during the maximal 200-m swim were fitted using a nonlinear least-square regression technique implemented in Matlab R2010b (Mathworks, USA).

For the analysis of $\dot{V}O_2$ kinetics, the first two phases of the generally adopted 3-phase model were identified, since the exercise duration and intensity constrained the appearance of the slow component (Phase III) (38). Phase I (cardiodynamic component) was determined as the time from the onset of exercise to a point of sharper increase in $VO_2$, and its duration was computed as a time delay for the primary component ($TD_p$). Phase II (principal component)
Parameters were estimated using a monoexponential model according to the following equation:

\[ \dot{V}O_2(t) = A_0 + A_p \cdot (1 - e^{-(t-TD_p)/\tau_p}) \]  

where \( t \) (s) is the time from the onset of exercise; \( A_0 \) is the baseline amplitude; \( A_p \) is the amplitude of the principal component; \( TD_p \) (s) is the time delay of the first exponential term and equals the duration of phase I (cardiodynamic component); and \( \tau_p \) is the time constant of the principal component. The total amplitude (\( A_{tot} \)) was calculated as \( A_{tot} = A_0 + A_p \). On a preliminary analysis, \( \dot{V}O_{2\text{max}} \) was calculated from the 200-m maximal swim as the asymptotic amplitude of the monoexponential equation (\( A_{tot} \)), and as the last 20-s averaged values (3,450 ± 711 vs. 3,364 ± 713 ml·min\(^{-1}\), respectively; \( P = 0.105 \)). Since both values were not different, \( A_t \) was then chosen to best represent the highest \( \dot{V}O_2 \) attained during the maximal 200-m swim test and, thus, as the swimmer’s \( \dot{V}O_{2\text{max}} \) (30). The reliability of \( \dot{V}O_{2\text{max}} \) measurements was characterized by a typical error (TE) of 3.1% (95% confidence interval, 95% CI: 1.1–5.1%; \( n = 9 \)).

**Training and Monitoring**

Individualized training plans were developed by the swimmers’ personal coaches, all of them very experienced in AT and well acquainted with the special characteristics of the environment. They were free to implement their own training program according to their previous experience, and the swimmer’s fitness level and previous and expected individual response to altitude. All coaches emphasized endurance, short-interval speed, strength, and flexibility. Typically, training schedules included a morning and an afternoon pool session, and a dry-land workout generally oriented to strength and flexibility from Monday to Saturday morning. Throughout the entire duration of the training camp, careful resting and exercise HR
and training time monitoring permitted calculation of the “training impulse” for each daily workout. The study was carried out during the first macrocycle (short-course season) of the Olympic year (October-December 2011) prior to the London 2012 Olympic Games. The intervention period comprised a 3- or 4-week mesocycle during the general preparation phase (early/mid October up to early/mid November). The performance follow-up period (POST) comprised part of the specific preparation macrocycle (early/mid November up to early/mid December) and included some mid-season races. Prior to participation an agreement was attained with all coaches that training load would not substantially be reduced and no tapering implemented during the 4-week follow-up phase to allow the full assessment of the training intervention. Prior to all performance tests coaches were asked to reduce training load the day before to minimize the influence of fatigue.

Pool training was monitored using waterproof HR monitors (CardioSwim, Freelap, Switzerland), which recorded beat-by-beat HR and lap times. Beacon transmitters (TX H2O, Freelap, Switzerland) were placed at the ends of the swimming pool so that the HR monitors’ microprocessor units could register the lap times, rest intervals, and 50-m average speed. HR was assessed from R–R intervals, 1-s interpolated, and averaged for 5-s intervals. Dryland training was monitored using beat-by-beat Polar RS800CX monitors (Polar Electro Oy, Finland) with the same 5-s interval averaging. At the end of each training session, data were downloaded, processed and stored using the Freelap Manager and Polar Pro-Trainer-5 software.

Estimation of Training Load. The TRIMP method (2) was used to quantify the internal training load. Additionally, to improve estimation when monitoring interval training sessions, a modified calculation method (TRIMPc) (13) was used:
where TRIMP\textsubscript{c} = cumulative TRIMP (a.u.); and \( n \) = total (cumulative) number of exercise and rest intervals during the training session, each with its corresponding HR ratio. To allow the comparison of the TRIMP\textsubscript{c} values at SL and at 2,320 m above SL we used a correction factor based on the results of the experiments conducted by Wehrlin and Hallén on the changes in \( \dot{V}O\textsubscript{2} \) and other cardiorespiratory and metabolic parameters with increasing altitude in treadmill running (45). Based on their data, for the same absolute load (55\% of \( \dot{V}O\textsubscript{2\max} \)) HR was \(~8\%\) (90\% CI: \(~6\–10\%\)) higher at 2,300 m of altitude compared to near SL (300 m), and maximal HR decreased by \(~2.1\%\) (90\% CI: \(~1.6\–2.6\%\)). To minimize the effect of the elevation in resting HR on TRIMP\textsubscript{c} calculations we used the resting HR values during a variability test (6 min at supine position first time in the morning) measured daily (first 9 days) or twice a week (for the rest of the training camp).

Training Log and Clinical Surveillance. During the training camp, each athlete kept a detailed training log which included 1) a self-administered 10-item questionnaire to assess training intensity within 30 min after each training workout (session or s-RPE); 2) a 10-item fatigue questionnaire (TSF-10), modified from a previous 7-item questionnaire developed for swimmers (1); 3) the Lake Louise Score questionnaire for the assessment of acute mountain sickness symptoms; and 4) changes in health status (illness, injury, clinical symptoms, menstruation, etc.) and well-being, along with the outcome of the consultation with a health professional. Only the results of the two first assessments will be reported here.
Nutrition and Iron Supplementation

During the entire study, high quality sports nutrition was provided to subjects under the supervision of their team nutritionist or physiologists with support from the nutrition staff of the training centers on demand. Subjects were provided *ad libitum* access to water in all times and were strongly recommended to drink as often as possible. One month before starting the study, teams were asked to perform a blood test on all swimmers at their country of residence. Iron supplementation (100 mg of Fe\(^{++}\)* per os daily) was strongly recommended even for those with normal ferritin values, but not imposed. For a few subjects with a suboptimal ferritin level (<50 ng·ml\(^{-1}\)) or past history of iron deficiency or anemic episodes, treatment under prescription and supervision of their medical staff was a requirement for participation. During the lead-in period serum ferritin concentration was analyzed using an electrochemiluminescence immunoassay (Roche Diagnostics GmbH, Mannheim, Germany). One female subject had a low ferritin level (17 ng·ml\(^{-1}\)) and was excluded from the study. The mean value (± SD) was 110 ± 73 ng·ml\(^{-1}\) (range 28 to 380). Ferritin was also monitored weekly in all altitude groups during the intervention period.

Total Hemoglobin Mass

\(tHb_{mass}\) was measured using the optimized CO-rebreathing method, as described by Schmidt and Prommer (39) with some modifications (16, 27). Briefly, the subjects inhaled a bolus of carbon monoxide (1.0 ml CO·kg\(^{-1}\) for males and 0.8 ml·kg\(^{-1}\) for females) followed by 3 L pure oxygen and rebreathed in a closed-system spirometer (SpiCo, Bayreuth, Germany) for 2 min. The volume of CO administered at altitude was adjusted according to barometric pressure. Arterialized blood was sampled from an ear lobe before and after the rebreathing period (at 6 and
8 min) for analyzing carboxyhemoglobin (COHb) using a CO-hemoximeter (OSM3, Radiometer, Denmark). COHb was measured in sextuplicate before and in triplicate at min 6 and min 8 after starting the inhalation period. To calculate the amount of CO not taken up during the inhalation time and the amount exhaled after the test, the remaining CO in the spirometer and the end-tidal CO concentration were determined by using a portable CO-analyzer (Draeger, Luebeck, Germany). In our mobile laboratory, the reliability of this method, determined during this study by test–retest, was characterized by a TE of 1.3% in male athletes and 1.5% in females, averaging 1.35% (95% CI: 0.10–2.65%).

**Training Camp and Placebo/Nocebo Effects**

All training camps were conducted in training centers of international standards, whether at SL or at altitude, where subjects lived and trained as a group for the whole intervention period. In the recruiting phase, coaches were offered to choose among the four different interventions, and were asked to take to the training camp only those swimmers who had positive or neutral expectations regarding the effect of that intervention in their preparation and future performance. To evaluate eventual placebo or nocebo effects of the intervention, two *ad hoc* questionnaires were administered at the beginning and at the end of the intervention period, prior to PRE and POST testing respectively, one for the head coaches and one for the swimmers. On their questionnaire, coaches were asked to state whether (yes, no, or not sure) they believed that the chosen intervention would help (PRE) or had helped (POST) the swimmers to improve their swimming performance, and whether they would choose again the same intervention as at the time of entering the study (POST). On their questionnaire, swimmers were asked to state whether they believed that their training camp would (PRE) or did (POST) help them to improve their swimming performance.
Statistical Analysis

Descriptive data are presented as arithmetic means and standard deviations (± SD). Effects on performance, O₂ kinetics parameters and tHb\textsubscript{mass} are expressed as percent change values (Δ%) and 90% confidence intervals of the mean (± 90% CI). 95% CI are used for other variables as indicated. To assess the relationship between \(\dot{V}O_2\text{max}\), tHb\textsubscript{mass}, and performance indicators, the Pearson’s correlation coefficient (r) and coefficient of determination (r\(^2\)) were used. To assess the effect of the intervention on swimming time trial performance over time, the primary outcome measure of this study, a comparison among all time points was made using percent change from PRE baseline values. We used the linear mixed modeling procedure for repeated measures (Proc Mixed) using the statistical package SAS (version 9.1.3, SAS Institute, Cary, NC) to estimate means for main effects and interaction of group (Hi-Hi, Hi-Hi3, Hi-HiLo, Lo-Lo) and test (PRE, POST, PostW1, PostW2, PostW3, PostW4). Where a significant effect was obtained, a post hoc analysis (Tukey’s test) was performed to identify the source of differences. The same analysis was performed for tHb\textsubscript{mass} measurements, using the mean of Pre1 and Pre2 as baseline values (PRE) and interaction of group and test during the intervention (PRE, W1, W2, W3, W4). Since preliminary analysis revealed differences in TRIMPc among groups, an analysis of covariance (ANCOVA) was carried out using TRIMPc as a covariate for performance in the three time trials. To evaluate the effects of the intervention on \(\dot{V}O_2\text{max}\), a statistical comparison was conducted between the PRE score and the score obtained after the intervention period (POST) using a two-tailed paired t-test in the four experimental groups. Time-trial swimming performance was assessed only once considering the high reliability of these measurements on a previous study with trained swimmers for 100-m (TE%: 1.4%; 95% CI: ± 1.5%) and 400-m (TE%: 1.5%; 95% CI: ± 1.5%) time trials (34), and to avoid undue psycho-
physical stress to the subjects and the risk of underperformance. Precise $P$-values are reported, and significance level was set at $P$ (probability of type I error) $< \alpha = 0.05$.

RESULTS

Training Camps

After the intervention period, all coaches ($n = 8, 100\%$) responded to the ad hoc questionnaire that they would have chosen again the same intervention as at the time of entering the study, and that they expected that the chosen intervention would help the swimmers to improve their performance. On their PRE questionnaire, all swimmers stated that (‘yes’) they had chosen to participate in the training camp in the belief that it would help them to improve their swimming performance after the intervention. On their POST questionnaire, their answers to the same question were ‘yes’ ($n = 49; 91\%$), or ‘not sure’ ($n = 5; 9\%$). These last subjects belonged to the Lo-Lo ($n = 1$), Hi-Hi ($n = 2$), and Hi-Hi3 ($n = 2$) groups. No subjects answered ‘no’.

Throughout the camp, daily average TRIMPc was greater in Hi-HiLo ($258 \pm 95$) than in Hi-Hi ($205 \pm 102; P = 0.01$), Hi-Hi3 ($177 \pm 115; P < 0.001$), and Lo-Lo ($209 \pm 100; P = 0.006$). Mean daily TRIMPc values were greater in females than in males ($245$ vs. $185; P < 0.001$). TRIMPc, an objective indicator of internal training load, showed a strong correlation with perceived exertion after training (s-RPE score) ($r = 0.724; P < 0.001$). Mean daily s-RPE scores throughout the camp were greater in Hi-Hi3 ($5.3 \pm 1.8$), than in the other three groups (Hi-Hi: $4.4 \pm 1.9, P < 0.001$; Hi-HiLo: $4.8 \pm 1.5, P = 0.01$; and Lo-Lo: $4.6 \pm 1.8, P < 0.001$). Mean daily TSF-10 scores were also higher ($P < 0.001$) in Hi-Hi3 ($25.0 \pm 7.5$) than in the other three groups (Hi-Hi: $20.2 \pm 6.1$; Hi-HiLo: $18.6 \pm 5.4$; and Lo-Lo: $21.0 \pm 10.0$). (See Figure, Supplemental Digital Content 1, comparing the three training load indicators throughout the camp, http://links.lww.com/MSS/A506).
Primary Analysis: Swimming Performance

The time course of time trial performance over time are presented in Table 2 and Figure 2.

TT50. Immediately after the training camp (POST), TT50 performance remained nearly stable in all groups but deteriorated in Hi-Hi3 and remained slower than at PRE until PostW3 (Figure 2.A). At PostW1 all groups improved performance (Hi-Hi3 could not be tested) compared to PRE: Lo-Lo (-2.0%; ± 1.6%; P < 0.001), Hi-Hi (Δt = -4.0%; ± 0.9%; P < 0.001), Hi-HiLo (Δt = -4.8%; ± 0.4%; P < 0.001). From that time point, the Hi-HiLo group tended to improve sprinting performance, reaching the highest change (Δt = -5.5%; ± 1.0%; P < 0.001 from PRE) at the end of the 4-week follow-up period, and exceeding the change in the Lo-Lo control group at that point (Δt = -3.2%; ± 1.1%; P < 0.001 from PRE) (group x test interaction P = 0.01). The rest of the groups stabilized their performance reaching equally significant changes from PRE values by the end of the study as compared with the Lo-Lo controls (Hi-Hi3: Δt = -3.4%; ± 4.0%; P < 0.001; Hi-Hi: -3.7%; ± 1.2%; P < 0.001).

TT400. Immediately after the training camp (POST), all groups tended to decrease 400-m TT performance, whereas the Hi-HiLo tended to improve and swam faster than the Hi-Hi group (group x test interaction P = 0.03) (Figure 2.B). At PostW1 all groups experienced nearly identical improvement in performance (Δt ~2%), but the Hi-HiLo group continued to improve at PostW2 (Δt = -4.2%; ± 0.9%; P < 0.001), point at which their improvement from PRE was greater compared to all other groups (group x time interaction P < 0.001). By the end of the follow-up period, both the Hi-HiLo (Δt = -4.7%; ± 1.1%; P < 0.001) and the Hi-Hi swimmers (Δt = -3.3%; ± 1.3%; P < 0.001) had improved more than the Lo-Lo controls (Δt = -1.6%; ± 1.0%; P
< 0.001) (group x test interaction \( P = 0.001 \) and 0.03, respectively). Even if the linear mixed model was not able to detect significant differences between both 4-week altitude groups (group x test interaction \( P = 0.23 \)), the size of the effect in the Hi-HiLo group was “most likely” greater than in the Hi-Hi group.

**TT100 or TT200.** Since sprinters and non-sprinters swam 100 and 200 m, respectively, at their best personal stroke, this outcome was considered the most specific performance assessment in terms of distance and stroke, and they are presented combined in Figure 2.C. Immediately after the training camp, all groups performed similarly as compared to PRE, except for Hi-Hi3 (\( \Delta t = +1.9\%; \pm 1.3\%; P = 0.06 \)) that was slower than PRE and got worse as compared to Lo-Lo, Hi-Hi and Hi-HiLo (group x test interaction \( P = 0.006, 0.03 \) and < 0.001, respectively). At PostW1, all groups improved similarly to the Lo-Lo controls (mean \( \Delta t = -2–3.5\% \)), and only the Hi-HiLo group improved more than Hi-Hi3 (group x test interaction \( P = 0.03 \)). From that point, the Hi-HiLo group progressed faster than the rest at PostW2 (\( \Delta t = -5.3\%; \pm 1.4\%; P < 0.001 \)) and PostW4 (\( \Delta t = -6.3\%; \pm 1.2\%; P < 0.001 \)), and by the end of the follow-up period, these improvements were substantially greater compared to Lo-Lo (\( \Delta t = -3.7\%; \pm 1.0\%; P < 0.001 \)), Hi-Hi3 (\( \Delta t = -3.1\%; \pm 0.9\%; P < 0.001 \)) and Hi-Hi (\( \Delta t = -3.4\%; \pm 1.0\%; P < 0.001 \)) (group x test interaction \( P = 0.02, 0.002 \) and < 0.001, respectively).

As mentioned before, the daily average TRIMPc throughout the training camp was greater in Hi-HiLo than in the other groups. To ascertain the eventual effect of training load on performance changes, an additional ANCOVA analysis was carried out using TRIMPc as a covariate for all time trials. A significant group x TRIMPc interaction was observed for TT400 only (\( P = 0.002 \)) and, therefore, main effects were reassessed adjusting for TRIMPc. The results
confirmed the differences between groups at all time points, with the exception of differences between Hi-Hi and Lo-Lo at PostW4 in TT400 that became not significant ($P = 0.08$).

### Oxygen Uptake Kinetics and $\dot{V}O_{2\text{max}}$

Table 3 shows the $\dot{V}O_2$ kinetics parameters during T4x200.

There were no significant changes in $\dot{V}O_{2\text{max}}$ ($\Delta\%$ from PRE, $P > 0.05$) in either Lo-Lo (1.9%; ± 1.5%), Hi-Hi3 (1.5%; ± 2.5%), Hi-Hi (1.1%; ± 2.6%), or Hi-HiLo (1.3%; ± 1.4%), with larger variability in the individual changes in both groups living and training at altitude (see Figure, Supplemental Digital Content 2, showing individual and group mean changes, http://links.lww.com/MSS/A507).

No significant relationship between relative percent change in $\dot{V}O_{2\text{max}}$ and percent change in TT400 performance was found for the entire group of subjects ($r = -0.01, P = 0.95$) or for the swimmers in each group ($r = -0.22, 0.68, -0.12, and -0.39; P = 0.55, 0.09, 0.69, and 0.19$, respectively for Lo-Lo, Hi-Hi3, Hi-Hi, and Hi-HiLo). Likewise, there was no relationship between change in $\dot{V}O_{2\text{max}}$ and change in TT100 or TT200 performance, neither for all subjects ($r = 0.10, P = 0.50$) nor for the swimmers in each group ($r = 0.27, -0.002, 0.20, and 0.06; P = 0.46, 1.00, 0.52, and 0.84$, respectively for Lo-Lo, Hi-Hi3, Hi-Hi, and Hi-HiLo) (see Figure, Supplemental Digital Content 3, showing the regression plots, http://links.lww.com/MSS/A508).

### Total Hemoglobin Mass

$tHb_{\text{mass}}$ was not different among groups before the training camp (PRE, $P = 0.49$). The magnitude of $tHb_{\text{mass}}$ changes at altitude showed remarkable between- and within-groups
variability (see Figure, Supplemental Digital Content 4, showing individual and mean changes, http://links.lww.com/MSS/A509).

Figure 3 shows the time course of percent changes in tHb\text{mass} during the training camps. In the Hi-Hi group, tHb\text{mass} continuously increased from PRE (766 ± 187 g) for 3 weeks (W1: 779 ± 192 g, \( P = 0.03 \); W2: 796 ± 196 g, \( P < 0.001 \); W3: 810 ± 204 g, \( P < 0.001 \)), and remained nearly unchanged at W4 (815 ± 202 g, \( P < 0.001 \)). In the Hi-Hi3, tHb\text{mass} did not change from PRE (816 ± 205 g) at W1, but increased at W2 (857 ± 223 g, \( P < 0.001 \)) and W3 (851 ± 227 g, \( P = 0.02 \)). In contrast, no significant changes were found in the Hi-HiLo group from PRE (896 ± 167 g) at any point during the training camp. Compared to PRE, increase in tHb\text{mass} was more pronounced in the Hi-Hi group (at W4: 6.2%; CI 90% ± 1.1%; \( P < 0.001 \)) than in the Hi-Hi3 group (at W3: 3.8%; ± 2.3; \( P = 0.08 \); group x test interaction \( P = 0.02 \)), whereas no significant changes were found in the Hi-HiLo group (at W4: 1.3%; ± 1.8; \( P = 0.71 \)). Relative changes (\( \Delta \% \) compared to PRE) in tHb\text{mass} were not associated with changes in \( \dot{V}\text{O}_{2\text{max}} \) neither for all subjects (\( r = 0.01; \ P = 0.96 \)), nor for each experimental group (\( r = -0.31, 0.16, -0.28, \) and 0.30, respectively for Lo-Lo, Hi-Hi3, Hi-Hi, and Hi-HiLo; \( P = 0.3–0.6 \)).

DISCUSSION

To the best of our knowledge, this is the first investigation to show performance improvements after a terrestrial AT intervention using a controlled design in swimmers, and one of the few in truly elite athletes (12, 37). The major findings of the study were that: 1) there were no changes, or in some cases a worsening of performance, immediately following 3-4 weeks of any training strategy in this group of elite swimmers; 2) swimming performance in stroke-specific 100 or 200 m improved significantly by \( \sim3.1–3.7\% \) after 1 to 4 weeks of recovery
following completion of a coach-prescribed training camp conducted at SL or at moderate altitude (2,320 m); 3) when 2 weekly sessions of high intensity training at lower altitude were included (Hi-HiLo strategy), a greater improvement in performance occurred 2 and 4 weeks after the training camp (5.3 and 6.3%, respectively); 4) similarly, the Hi-HiLo intervention also elicited further improvement in both 400- and 50-m stroke-nonspecific swimming performance, evidenced 2 weeks (4.2% and 5.2%, respectively) and 4 weeks (4.7% and 5.5%, respectively) after return to SL; and 5) this substantial delayed performance enhancement was not linked linearly to changes in $\dot{V}O_{2\text{max}}$, oxygen kinetics or tHb$_{max}$, hence could not be attributed exclusively to enhanced oxygen transport capacity.

Effects on Performance

The present results clearly show the potential benefit of conducting a well-implemented training camp whether at altitude or at SL following at least one week of recovery from the training camp exposure. The potential improvement seemed to range from primarily anaerobic (50 m) to predominantly aerobic events (400 m), with no sex differences in the response. Previous analysis of competitive performance in Olympic swimmers estimated that the typical variation in an athlete’s maximum performance in competition was $\sim$0.8–1% (42), and $\sim$2% improvement during the final 3 weeks of preparation before the Sydney 2000 Olympic Games has also been reported (26). In a previous study of performance in training, typical variations in performance times were about 2–4% in international Australian swimmers over a 4-month period (28). Taken together, these reports are in line with our finding of a $\sim$3.5% improvement in performance over a 7 to 8-week period during the general preparation phase of the winter
macrocycle in all groups, with an additional \( \sim 2.7\% \) attributable to the Hi-HiLo group intervention.

Although the vast majority of studies in the AT literature are uncontrolled and underpowered—especially with elite athletes (3, 23, 37)—, there seems to be a growing consensus that when athletes are exposed to a high enough altitude, for a long enough amount of time, and are able to preserve fitness by training hard under normoxic conditions, the majority may improve endurance performance (47). In a recent meta-analytic review, Bonetti and Hopkins (3) concluded that performance changes in studies using the conventional Hi-Hi approach were unclear, whereas changes using the terrestrial Hi-Lo strategy were considered “likely” both for elite and subelite athletes (\( \sim 4\% \)), or a more realistic 1.5% when performance was predicted from uncontrolled studies. These estimations are in line with a recent review by Saunders et al. (37) in which, by using a regression analysis of average performance changes, it was estimated that a 3-week terrestrial altitude training camp would elicit mean performance improvements of \( \sim 1.8\% \) (Hi-Hi) and \( \sim 2.5\% \) (Hi-Lo) (37).

The evidence in swimming is much less compelling. Six uncontrolled studies have tested the Hi-Hi strategy in swimmers. Three of them were entirely negative at either short or long distances from 100 m to 2,000 yd (10, 35). Two others showed modest and statistically unclear improvements in performance of \( \sim 1.6–1.8\% \) (11, 25). In the only study with a SL control group, 10 male and female Korean elite swimmers lived and trained for 21 days at 1,890 m (8). No statistical analysis was reported, but the small increase in performance in 100- and 200-m races (0.1–0.7%) was below the smallest worthwhile enhancement effect of the intervention.

Why should swimming be different from land-based endurance sports regarding AT effects? First, swimming performance is more dependent on economy, i.e. the energy cost of
swimming, than on maximal metabolic power (9); it follows that the benefit of enhanced $\dot{V}O_{2\text{max}}$ could be outweighed by impaired technique and economy (24). Second, the benefit of AT might be more or less potent for swimmers of different events; for example, the energy percent share (phosphagenic–glycolytic–oxidative) at maximal competitive speed (front crawl) ranges from ~38–58–4% in 50 m to ~6–21–73% in 400 m (33), and more than three quarters of all competitive events are completed in less than 2.5 min by athletes of national standard or better. Third, in contrast to most endurance sports, a very large proportion of training is performed in intervals, with a growing emphasis on high intensity and strength training (7).

**Time Course of Performance Changes**

A distinguishing feature of the present study was that the follow-up period after the intervention covered a wide time span, up to 4 weeks post-intervention. This approach may provide useful evidence about the proper timing of return from AT for optimal SL performance, which remains largely unknown. If we focus on the stroke/distance specific assessment (TT100/200), it becomes clear that best performances were attained 4 weeks after returning to SL, although the superior benefits of the Hi-HiLo intervention became evident already 2 weeks after the training camp. A similar response was observed in TT400, but TT50 significantly improved on the first week after both 4-week altitude camps. Therefore, based on the present results, 50-m sprinters would likely perform at their best level 1 week after completing an effective AT camp, whereas specialists in longer distances would attain their best performance 2 to 4 weeks after the camp. The present findings are partially in line with the results reported by Levine & Stray-Gundersen with college runners, who observed the effect on performance—in the Hi-Lo group only—immediately after and for 3 weeks on return to SL (22). Observational
studies in swimmers have been less compelling. In an uncontrolled study, Friedmann et al. (11) found an unclear improvement in swimming performance (1.8%) 10 days after returning to SL. Gough et al. (20) analyzed the time course of performance based on official competitive records after two types of 3-week hypoxic exposure; swimming performances were substantially slower one day (~1.5%) and one week (0.9–1.9%) after altitude/hypoxic training compared to controls, and not different from pre-altitude 2 and 3 weeks later. Wachsmuth et al. (44) related \( t \)Hb\(_{\text{mass}} \) changes to competitive performance records over the course of 2 years in various groups of elite swimmers undertaking classical AT for 3 or 4 weeks; they found a non-significant drop in competition performance by ~1–2% on days 1 and 7 after AT camps, and there was an unclear improvement of performance (0.8%) between 25 and 35 days after return in a small group of 4 athletes. It is worth emphasizing that in the present study, both Hi-Hi groups’ performance actually deteriorated immediately after the training camp (i.e., Hi-Hi3 decreased performance in all three tests, and Hi-Hi got worse in TT50 and TT100/200) or one week later (i.e., Hi-Hi was slower in TT50 and TT400). The mechanism(s) underlying the delayed effect of the different interventions is uncertain, although empirical evidence suggests that there is a necessary period for physiological and psychological recovery from the accumulated training stress and fatigue during the camp (29). In any case, the potential short-term detrimental effect of an altitude training camp should be acknowledged.

The Effect of Training

A key factor in the individual response to training, whether at altitude or at SL, is the training load. There is a methodological issue that needs to be addressed. For this study, we used a modification of the training impulse method (TRIM\( \text{Pc} \)) deriving from cumulative exercise and
recovery intervals (see Methods). However, to compare training loads at SL and altitude can be tricky. For example, when exercising at altitude at a given $\dot{V}O_2$ the submaximal HR response is increased (45), and, as a consequence of the reduced aerobic power (~15% at 2,300 m) (45), endurance training with an identical absolute workload always translates to a more intense (greater relative workload) training at the higher altitude. Both components likely contribute importantly to the cardiovascular and metabolic response to exercise and thereby the training response. As one example in swimming, a group of competitive swimmers swum a 400-m time trial at 690 m above SL, and within 3-4 h on arrival to 2,320 m at 92.5% of maximal speed at the previous test; peak HR (~5%) and blood lactate (~6%) were higher at altitude (24). Therefore, the weighting factors derived empirically by Bannister et al. (2) may not be as accurate or comparable when training at altitude. Interestingly, TRIMP data reported by Levine and Stray-Gundersen (22) calculated using Banister’s method, despite the fact that base training was achieved at slower speed and at a lower percentage of sea-level $\dot{V}O_2_{max}$ at moderate altitude, training HR was essentially the same; moreover, during interval training at moderate altitude the athletes accomplished significantly lower training workloads and oxygen flux compared to SL (19% lower $\dot{V}O_2$) whereas training HR was only 5% lower. Taking all these factors into consideration, we calculated that for a given training intensity around the average training HR throughout the entire AT camp in our swimmers (~125 bpm), a HR increase of 8% combined with a reduction of 2.1% in maximal HR would increase TRIMPe values by 23% compared to normoxic conditions. Whether this difference translates into a true difference in training stimulus is unknown.

A few other points about training deserve attention. First, there were no differences in calculated training load between the two Hi-Hi groups compared to the Lo-Lo controls,
empirically supporting the validity of the proposed correction for exercise in hypoxia. Second, the higher training workloads achieved during the 2 weekly low-level workouts in the Hi-HiLo group—i.e., the primary factor in comparing both 4-week altitude interventions—is likely to account for at least part of the differences between the Hi-Hi and Hi-HiLo groups. Third, in contrast, the group perceiving the greatest training effort (s-RPE) and fatigue (TSF-10) throughout the training camp was the Hi-Hi3 group and not the Hi-HiLo (see Figure, Supplemental Digital Content 1, comparing the three training load indicators throughout the camp, http://links.lww.com/MSS/A506); this suggests that training intensity may have been greater in Hi-Hi3, causing a certain degree of overreaching and a worse response in performance capacity immediately after the intervention, particularly in TT50. However, TT400 and TT100/200 performance was remarkably similar to the Hi-Hi group after 1 week, suggesting that the effect of altitude acclimatization—i.e., the primary factor in comparing 3- and 4-week altitude interventions—was commensurate. Fourth, regardless of these differences, when performance data were adjusted for TRIMPC as a covariate, there remained significant differences between groups at all time points suggesting that the differences between groups are not likely to be attributed solely to training. Ultimately, the fact that the Hi-HiLo group achieved a greater training internal load—but not training effort—may be a core element of the Hi-HiLo training paradigm and hence, a main factor likely contributing to the performance enhancement accomplished by this particular group.

Erythropoietic Response

There is now little doubt that altitude exposure, even in elite athletes of various sport types (14), including elite swimmers (20), causes an erythropoietic effect, given an adequate
exposure, in the majority of athletes (19, 22, 36). Consistent with previous reports, we found that tHb\textsubscript{mass} clearly increased in those swimmers living and training at altitude for 3 (3.8%) or 4 weeks (6.2%) (Figure 3). The magnitude, time course, and large variability of the erythropoietic response was in line with a recently published meta-analysis including data of 16 altitude training studies (19). For our study, therefore, it is unlikely that the magnitude and duration of altitude exposure was insufficient to induce a functional acclimatization response, at least for the majority of athletes.

However, in contrast to the Hi-Hi groups, mean tHb\textsubscript{mass} did not change in our Hi-HiLo swimmers (at W4: 1.3; ± 1.8%) who were also exposed to the same degree of sustained hypobaric hypoxia for 4 weeks. The simplest explanation for these contrasting results may be the individual variability in tHb\textsubscript{mass} changes, since half of the subjects actually showed an increase of tHb\textsubscript{mass} over the TE of the measurement (see Figure, Supplemental Digital Content 4, showing individual and mean changes, , http://links.lww.com/MSS/A509). Comparable results were reported by Gore et al. in a small group of 8 elite track cyclists living and training at 2,690 m for 31 days (18). We must consider also that a wide variability in the erythropoietic response to moderate hypoxia has been consistently shown (6, 11, 19), also in swimmers (3.0% in males and 2.7% in females) followed over 2-year period (44). Other mechanisms such as superimposed inflammation, or perhaps medications like NSAIDs which may suppress the erythropoietic response in some athlete should also be considered (43).

**Relationship between Changes in tHb\textsubscript{mass} and \( \dot{V}O_{2\text{max}} \)**

In the present study, changes in tHb\textsubscript{mass} were not associated with changes in \( \dot{V}O_{2\text{max}} \), neither in the whole group of swimmers nor in any of the individual experimental groups. Under
controlled conditions involving athletes training at SL, cross sectional and longitudinal data show that $\dot{V}O_{2\text{max}}$ of elite athletes is closely related to $tHb_{\text{mass}}$ and a random variation of 1 g is associated with a change in $\dot{V}O_{2\text{max}}$ by $\sim$4 ml·min$^{-1}$ (36, 40). However, after real or simulated altitude however, the relationship between the gain in $tHb_{\text{mass}}$ and $\dot{V}O_{2\text{max}}$ has been less robust (36, 40). For example, in runners performing the original Hi-Lo protocol, the correlation for the change in $\dot{V}O_{2\text{max}}$ and red cell volume yielded an $r^2 = 0.14$ (22). These results are in line with those reported in a recent review (36) of 10 recent studies involving four different sports, which estimated a mean $\sim$3% increase in $tHb_{\text{mass}}$ and $\dot{V}O_{2\text{max}}$ and a similarly significant, albeit weak, correlation between both parameters ($r^2 = 0.15$). It should be emphasized that this relationship between changes in $tHb_{\text{mass}}$ and $\dot{V}O_{2\text{max}}$ after altitude exposure is the same order of magnitude as that observed after rhEPO administration (e.g. $r^2 = 0.28$) (36, 40) highlighting that $\dot{V}O_{2\text{max}}$ is a complex parameter that is not exclusively determined by the red cell mass. It is, therefore, not surprising that the present study could not identify a significant linear relationship between $tHb_{\text{mass}}$ and $\dot{V}O_{2\text{max}}$ after any of the training interventions.

**Role of Systemic Oxygen Delivery in Performance**

No significant relationships were found between changes in swimming performance and changes in $\dot{V}O_{2\text{max}}$ in the entire group of swimmers or in any of the experimental groups (see Figure, Supplemental Digital Content 3, showing the regression plots, http://links.lww.com/MSS/A508). This observation highlights the apparent independence of relatively short swimming performances on $\dot{V}O_{2\text{max}}$ (and $tHb_{\text{mass}}$), though this concept is not limited to swimming. For example, in an elegant mechanistic study by Garvican et al. (15), blood
was actually removed to eliminate the effect of the erythropoietic response in female cyclists exposed to simulated (normobaric) hypoxia (Hi-Lo, 26 days, 16 h-day\(^{-1}\), 3,000 m), while undergoing training specifically designed to improve short duration power. Both the “clamped” (phlebotomy to restrain increased \(t\text{Hb}_{\text{mass}}\)) and unclamped groups improved 4-min supramaximal performance (power output above that required to elicit \(\dot{V}O_{2\text{max}}\)) to a similar degree (~4%), despite the fact that by design, and \(t\text{Hb}_{\text{mass}}\) (and consequently peak \(\dot{V}O_{2}\)) increased only in the latter. Like our study, this outcome emphasized the success of the training camp on short-term performance. However performance on a following ride to exhaustion at peak power output was substantially worse in the “clamped” group (38%), which reinforced the role of \(t\text{Hb}_{\text{mass}}\) and \(\dot{V}O_{2\text{max}}\) for repeated high-intensity endurance type efforts. The present study adds to the body of knowledge in this field highlighting the complex interaction among altitude acclimatization effects (such as \(t\text{Hb}_{\text{mass}}\) among others), altitude and SL training effects, \(\dot{V}O_{2\text{max}}\), and performance in events of different sports and different durations/intensities requiring widely divergent metabolic demands (11, 18, 44).

In our study, despite failing to demonstrate an increase in \(t\text{Hb}_{\text{mass}}\) or \(\dot{V}O_{2\text{max}}\), the swimmers in the Hi-HiLo group improved performance more than the altitude controls 2 and 4 weeks after the intervention. A number of possible explanations should be considered. First, swimming, especially in the shorter distances, may not be as dependent on systemic \(O_2\) delivery as endurance running or cycling. Second, there are other factors (e.g. differences in training intensity) that may have played a greater role in improving swimming performances through as yet undetermined mechanisms, e.g. improved muscle efficiency probably at a mitochondrial level, greater muscle buffering, and the ability to tolerate lactic acid production (17), or
improved $O_2$ flux to the exercising muscles (41). This would be particularly true for the differences between Hi-Hi and Hi-HiLo, where the altitude exposure was matched, and the only difference was the twice weekly low altitude training sessions. For the comparison between Lo-Lo and Hi-HiLo, data interpretation is more challenging, and it appears that some other aspect of altitude exposure independent of erythropoiesis must be playing a role. Finally, since the most relevant performance differences occurred after some period of training at SL, we cannot completely exclude the possibility that uncontrolled factors related to the post camp training experience could have influenced the swimming performances.

Collectively, our findings strongly support the involvement of factors not related to systemic $O_2$ delivery in the substantial improvement of performance observed in our Hi-HiLo swimmers, especially compared to the low altitude controls. Therefore, our view is that an “integrative model” of adaptation to hypoxia—not opposed but complementary to the prevailing “erythropoietic/central” or to alternative “nonhematological/peripheral” models—should be considered. This paradigm would recognize 1) the multifactorial nature of physiological adaptations to exposure to mid-term moderate hypoxia and to intensive training, 2) the multi-level control mechanisms of these adaptations (i.e., acting at the systemic, local, and cellular levels in a synergistic or antagonistic manner), and 3) the non-linear behavior of complex, dynamic systems.

Design and Limitations of the Study

From the early steps of this study we were committed to recruit truly elite athletes. Working with such unique individuals in a real-word setting, particularly during an Olympic season, certainly added substantial complexity to our study. Therefore, we were confronted with
the virtual impossibility to conduct a fully controlled experimental design without seriously compromising the ecological validity of the study (i.e., conducted in conditions closely matched to those in which in this unique type of athletic population usually live, train and compete), or limiting its external validity (i.e., the ability to generalize the outcome to the elite swimming population). Regarding subject assignment, we acknowledge that, as subjects were not allocated randomly, selection bias may have occurred. Similar to other sports, a team-coach binomial exists that cannot be separated from different intervention groups as would be required by strict randomization. In an effort to reduce the likelihood of assignment bias, first, we chose to allocate swimmers from at least two different squads and nations in each group and tested that groups were properly matched for performance level and key physiological characteristics ($\dot{V}O_{2\text{max}}$ and $tHb_{\text{max}}$). Finally, we chose to use mixed linear modeling for the primary data analysis because of its greater flexibility to model the variance/covariance structure of repeated measures data sets. This modeling approach proved here to be able to detect and quantify changes despite relatively small subsample sizes, likely owing to: 1) the high reliability of the time trial performance swimming tests (34), in which test-retest reliability matched measures from competition performance in top swimmers; 2) the multiple observations during the follow-up period in the four experimental groups; 3) the large magnitude of the fixed effects in performance observed; and 4) the robustness of the model against loss of statistical power due to missing values in the different assessments (e.g., HR monitoring, time trial tests, $\dot{V}O_{2\text{max}}$ measures, etc.).

An important issue with all training studies is the likelihood of information bias, eliciting the occurrence of placebo, nocebo, and training camp effects (3, 17, 21, 23). Since all camps were conducted in training centers of high international standard, a site-specific training camp effect is less likely, but of course still possible. An effort was made to counterbalance the
information bias by ensuring that all coaches and swimmers were fully informed prior to their involvement about the characteristics of the main intervention, and by stressing the unpredictability of the final outcome of each intervention compared to the others. Only then coaches were offered to choose which type of intervention they and their swimmers would finally be involved in. Finally, the responses to the *ad hoc* questionnaires to swimmers and coaches strongly suggested that nocebo and placebo effects are unlikely to have substantially affected the results of the study.

**Practical Implications for Training and Performance**

Based on the present results with elite athletes, a) after at least a week of recovery, stroke-distance specific swimming performance might be expected to substantially improve as a result of a well-implemented training camp, regardless of whether it is held at altitude or not; and b) a greater benefit can be expected by “living high-training high and low” (Hi-HiLo) for 4 weeks following 2 to 4 weeks of re-acclimatization to SL; this benefit can also expected to be superior in 50- and 400-m performance. However, care should be taken not to generalize these improvements to all swimmers, since substantial individual variability was noted in this as well as other studies including swimmers performing altitude training (6). Another important practical implication of our results is that performance is likely to be unchanged or worsened immediately after a 3- or 4-week intensive training camp regardless of the training environment, and that benefits can only be expected to occur following 1 to 4 weeks after completing the intervention. This delayed response could eventually provide a time window for tapering before competition as has been suggested (29). However, after a training camp at altitude, this outcome should be interpreted with caution since evidence suggests that the combination of the de-acclimatization
response of hematological, ventilatory, and biomechanical factors with return to SL likely interact to determine the best timing for competitive performance (5). Moreover, the potential short-term detrimental effect of an altitude training camp should not be neglected.

Monitoring individual training load and adaptation (e.g. resting, exercise and recovery HR response, HR variability, exercise perception and state of fatigue) during and after the altitude camp to avoid excessive overload or detraining, as well as assessing individual peaking performance profile, are strongly recommended before directly applying these rules to individual cases.

Conclusions

Sea level swimming performance of elite swimmers in 100- (sprinters) or 200-m (non-sprinters) time trials was not altered, or in some cases impaired immediately, but improved significantly by ~3.1–3.7% after 1 to 4 weeks of recovery following completion of a coach-prescribed training camp, whether it was conducted at sea level or at moderate altitude (2,320 m). By including 2 weekly sessions of high-intensity training at lower altitude (Hi-HiLo strategy) a greater improvement in performance occurred 2 and 4 weeks after the training camp (5.3% and 6.3%, respectively). Similarly, further improvement in 400- and 50-m freestyle time trial performance was noted 2 weeks (4.2% and 5.2%, respectively) and 4 weeks (4.7% and 5.5%, respectively) following return to sea level after the Hi-HiLo intervention. This delayed performance enhancement was not linked to changes in $\dot{V}O_{2\text{max}}$, $O_2$ kinetics or tHb$_{\text{mass}}$, hence could not be attributed exclusively to enhanced systemic oxygen delivery capacity. We conclude that: a) a well implemented 3- or 4-week training camp, whether conducted at sea level or at moderate altitude, enhances performance in most elite swimmers after a 1- to 4-week delay.
period, with substantial individual variability in the response; b) “living high-training high and low” for 4 weeks has the potential to improve swimming performance above and beyond altitude and sea level controls, through complex mechanisms involving altitude acclimatization and training effects.

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results of the present study do not constitute endorsement by the American College of Sports
Medicine.
REFERENCES


FIGURE CAPTIONS

FIGURE 1. Schematic timeline of the study (see text for explanation).

FIGURE 2. Time course of percent changes in time trial performance in (A) 50 m front crawl (TT50), (B) 400 m front crawl (TT400), and (C) 100 m (sprinters) or 200 m (non-sprinters) at best personal stroke (TT100 or TT200), from pre-training (PRE) to immediately after (POST), and week one to four after the training camp (PostW1 to PostW4). Group means (+ or -) SD are shown. Note that a negative change indicates an improvement in performance. Differences are group x test interactions among groups ($P < 0.05$): Hi-HiLo vs. Lo-Lo ($\#$), Hi-Hi3 (¶), and Hi-Hi (+); Lo-Lo vs. Hi-Hi3 ($\$)$ and Hi-Hi ($\&$); and Hi-Hi vs. Hi-Hi3 (x). "*": non-significant difference when adjusted for TRIMPc (see text for detail). Right brackets are differences at the end of the follow-up period (PostW4).

FIGURE 3. Time course of percent changes in total hemoglobin mass ($tHb_{\text{mass}}$) from pre-training (PRE) to week 3 or 4 (W1 to W3/W4) during the training camp. Symbols and error bars show mean and (+ or -) SD. Significance of differences from the pre-values is indicated in the text. Differences ($P < 0.05$) among groups are: Hi-HiLo vs. Hi-Hi (+) and Hi-Hi3 ($\#$), and Hi-Hi vs. Lo-Lo ($\&$).
SUPPLEMENTAL DIGITAL CONTENT

SDC 1. Figure. Training load throughout the training camp (W1 to W4) expressed as (A) daily average TRIMPc, (B) daily total score of fatigue (TSF-10), and (C) average daily session RPE (s-RPE). Group means and SD error bars are shown. Differences (P < 0.05) between group averages in (A) were: Hi-HiLo vs. Hi-Hi (+), Lo-Lo (#) and Hi-Hi3 (¶); differences in (B) were: Hi-Hi3 vs. Hi-HiLo (¶), Lo-Lo (§) and Hi-Hi (x); differences in (C) were: Hi-Hi3 vs. Hi-Hi (x), Lo-Lo (§) and Hi-HiLo (¶).

SDC 2. VO2max changes after the training camp (POST) from baseline values (PRE). Thin grey lines are individual values, and heavy black lines with symbols and error bars show group mean ± SD. No significant changes were found in any of the groups. Note the larger variability in Hi-Hi3 and Hi-Hi.

SDC 3. Relationship between VO2max and performance changes (PRE vs. POST, %) in (A) TT400 for all subjects, and (B) TT100 (sprinters) or TT200 (non-sprinters). Linear regression lines are displayed for each group (thinner line) and for all subjects (thicker black line). Regression equation and coefficient of determination (r2) for all subjects are indicated. None of these relationships were statistically significant.

SDC 4. Changes in tHbmass from baseline values (PRE) after the training camp (POST) for each experimental group. Thin grey lines are individual values, and heavy black lines with symbols and error bars show group mean ± SD.
Figure 1
Figure 2

(A) 

(B) 

(C)
Figure 3

The graph shows the change in Hb_mass (% change) over time across four weeks (PRE, W1, W2, W3, W4) for different conditions labeled as Hi-Hi, Hi-Hi3, Hi-HiLo, and Lo-Lo. Each condition is represented by a different marker and line style. Significant changes are indicated by symbols: + and #.
Training load throughout the training camp (W1 to W4) expressed as (A) daily average TRIMPc, (B) daily total score of fatigue (TSF-10), and (C) average daily session RPE (s-RPE). Group means and SD error bars are shown. Differences (\(P < 0.05\)) between group averages in (A) were: Hi-HiLo vs. Hi-Hi (+), Lo-Lo (#) and Hi-Hi3 (¶); differences in (B) were: Hi-Hi3 vs. Hi-HiLo (¶), Lo-Lo ($$\$$$) and Hi-Hi (x); differences in (C) were: Hi-Hi3 vs. Hi-Hi (x), Lo-Lo ($$\$$$) and Hi-HiLo (¶).
VO_{2max} changes after the training camp (POST) from baseline values (PRE). Thin grey lines are individual values, and heavy black lines with symbols and error bars show group mean ± SD. No significant changes were found in any of the groups. Note the larger variability in Hi-Hi3 and Hi-Hi.
Relationship between VO\textsubscript{2max} and performance changes (PRE vs. POST, %) in (A) TT400 for all subjects, and (B) TT100 (sprinters) or TT200 (non-sprinters). Linear regression lines are displayed for each group (thinner line) and for all subjects (thicker black line). Regression equation and coefficient of determination \((r^2)\) for all subjects are indicated. None of these relationships were statistically significant.

**A**
\[ y = -0.006x - 2.56 \]
\[ r^2 = 0.0001 \]

**B**
\[ y = 0.068x - 3.53 \]
\[ r^2 = 0.01 \]
Changes in $\Delta tHb_{mass}$ from baseline values (PRE) after the training camp (POST) for each experimental group. Thin grey lines are individual values, and heavy black lines with symbols and error bars show group mean ± SD.
<table>
<thead>
<tr>
<th></th>
<th>Lo-Lo (n = 11)</th>
<th>Hi-Hi3 (n = 15)</th>
<th>Hi-Hi (n = 16)</th>
<th>Hi-HiLo (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Females (8)</td>
<td>Males (3)</td>
<td>Females (8)</td>
<td>Males (7)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>19.6 ± 3.1</td>
<td>19.7 ± 2.7</td>
<td>20.8 ± 4.7</td>
<td>22.6 ± 2.2</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.2 ± 6.8</td>
<td>185.7 ± 5.5</td>
<td>171.1 ± 4.5</td>
<td>186.3 ± 4.9</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>65.2 ± 4.9</td>
<td>76.7 ± 9.5</td>
<td>61.4 ± 4.4</td>
<td>79.2 ± 6.8</td>
</tr>
<tr>
<td>$\dot{V}O_{2\text{max}}$ (ml·min$^{-1}$)</td>
<td>3,086 ± 312</td>
<td>4,033 ± 655</td>
<td>2,915 ± 535</td>
<td>4,216 ± 551</td>
</tr>
<tr>
<td>$\dot{V}O_{2\text{max}}$/BM (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>47.3 ± 3.5</td>
<td>52.4 ± 2.7</td>
<td>47.7 ± 9.1</td>
<td>53.3 ± 6.5</td>
</tr>
<tr>
<td>Total Hb mass (g)</td>
<td>665 ± 51</td>
<td>1,042 ± 146</td>
<td>692 ± 117</td>
<td>957 ± 197</td>
</tr>
<tr>
<td>FPS (points)</td>
<td>811 ± 45</td>
<td>764 ± 44</td>
<td>888 ± 55</td>
<td>823 ± 50</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Groups are: Lo-Lo, living and training at low altitude (4 wks); Hi-Hi3, living and training at altitude (3 weeks); Hi-Hi, living and training at altitude (4 wks); Hi-HiLo, living at altitude and training at altitude and lower level (4 wks). BM, body mass; Total Hb mass, total hemoglobin mass; $\dot{V}O_{2\text{max}}$, maximal oxygen uptake during 4x200-m swimming test; FPS, FINA Points Score 2012 of personal best time (long course).
<table>
<thead>
<tr>
<th></th>
<th>TT50 (s)</th>
<th>TT400 (s)</th>
<th>TT100/200 (s)*</th>
<th>TT100 (s)</th>
<th>TT200 (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lo-Lo (n=11, 8F/3M)</td>
<td>Hi-Hi3 (n=15, 8F/7M)</td>
<td>Hi-Hi (n=16, 10F/6M)</td>
<td>Hi-HiLo (n=12, 4F/8M)</td>
<td></td>
</tr>
<tr>
<td>PRE</td>
<td>28.7 ± 1.5</td>
<td>27.7 ± 1.7</td>
<td>28.1 ± 2.3</td>
<td>27.3 ± 2.0</td>
<td></td>
</tr>
<tr>
<td>POST</td>
<td>28.9 ± 1.4</td>
<td>27.9 ± 1.6</td>
<td>30.2 ± 2.2</td>
<td>27.4 ± 1.7</td>
<td></td>
</tr>
<tr>
<td>PostW1</td>
<td>28.3 ± 0.4 †</td>
<td>†</td>
<td>28.4 ± 1.7 †#</td>
<td>26.5 ± 2.0 †#</td>
<td></td>
</tr>
<tr>
<td>PostW2</td>
<td>27.9 ± 1.7 †#</td>
<td>27.4 ± 1.6 †</td>
<td>27.0 ± 2.0 †#</td>
<td>25.9 ± 1.7 †#</td>
<td></td>
</tr>
<tr>
<td>PostW3</td>
<td>28.2 ± 0.5</td>
<td>28.2 ± 1.3 (7)</td>
<td>25.7 ± 1.88 †#</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>PostW4</td>
<td>27.8 ± 1.7 †#</td>
<td>–</td>
<td>26.8 ± 2.5 †#</td>
<td>25.8 ± 2.1 †#</td>
<td></td>
</tr>
<tr>
<td>POST</td>
<td>277.5 ± 9.7</td>
<td>275.6 ± 11.4</td>
<td>271.7 ± 15.6</td>
<td>273.7 ± 16.0</td>
<td></td>
</tr>
<tr>
<td>PostW1</td>
<td>274.5 ± 11.6 †#</td>
<td>267.5 ± 0.0 #</td>
<td>270.2 ± 7.1 †#</td>
<td>278.5 ± 12.6 †</td>
<td></td>
</tr>
<tr>
<td>PostW2</td>
<td>275.2 ± 13.9 †#</td>
<td>273.6 ± 14.3 #</td>
<td>267.8 ± 14.3 †#</td>
<td>262.1 ± 12.9 †#</td>
<td></td>
</tr>
<tr>
<td>PostW3</td>
<td>275.8 ± 13.0 †#</td>
<td>274.4 ± 15.1 #</td>
<td>262.0 ± 18.4 †#</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>PostW4</td>
<td>275.1 ± 12.3 †#</td>
<td>–</td>
<td>261.7 ± 14.4 †#§ &amp;</td>
<td>261.0 ± 14.2 †#§</td>
<td></td>
</tr>
<tr>
<td>POST</td>
<td>121.1 ± 35.8</td>
<td>108.6 ± 40.9</td>
<td>128.6 ± 21.6</td>
<td>102.4 ± 38.1</td>
<td></td>
</tr>
<tr>
<td>PostW1</td>
<td>120.4 ± 35.8</td>
<td>99.4 ± 44.7</td>
<td>129.9 ± 23.3 †</td>
<td>104.0 ± 37.8</td>
<td></td>
</tr>
<tr>
<td>PostW2</td>
<td>89.7 ± 33.5 #</td>
<td>98.7 ± 38.0</td>
<td>133.9 ± 5.0 †#</td>
<td>93.0 ± 42.1 †</td>
<td></td>
</tr>
<tr>
<td>PostW3</td>
<td>97.5 ± 35.4</td>
<td>96.9 ± 41.5</td>
<td>126.8 ± 22.4 †#</td>
<td>97.1 ± 36.7 †#</td>
<td></td>
</tr>
<tr>
<td>PostW4</td>
<td>85.1 ± 34.0</td>
<td>99.2 ± 39.8</td>
<td>119.4 ± 29.7 †#</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>POST</td>
<td>126.5 ± 0.6 (3)</td>
<td>68.8 ± 8.1 (4)</td>
<td>–</td>
<td>68.7 ± 0.8 (2)</td>
<td></td>
</tr>
<tr>
<td>PostW1</td>
<td>65.3 ± 0.8 (3)</td>
<td>65.5 ± 7.3 (6)</td>
<td>58.4 ± 0.0 (1)</td>
<td>62.8 ± 6.5 (6)</td>
<td></td>
</tr>
<tr>
<td>PostW2</td>
<td>65.3 ± 0.9 (3)</td>
<td>67.7 ± 8.1 (4)</td>
<td>57.6 ± 0.0 (1)</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>PostW3</td>
<td>65.4 ± 0.7 (2)</td>
<td>67.5 ± 8.1 (4)</td>
<td>57.5 ± 0.0 (1)</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>PostW4</td>
<td>65.4 ± 0.1 (3)</td>
<td>67.5 ± 8.1 (4)</td>
<td>57.5 ± 0.0 (1)</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>POST</td>
<td>141.3 ± 11.7 (8)</td>
<td>144.8 ± 9.6 (8)</td>
<td>133.2 ± 11.7 (15)</td>
<td>137.7 ± 11.7 (6)</td>
<td></td>
</tr>
<tr>
<td>PostW1</td>
<td>140.2 ± 13.5 (8)</td>
<td>154.6 ± 12.1 (4)</td>
<td>135.0 ± 12.3 (14)</td>
<td>135.8 ± 11.8 (6)</td>
<td></td>
</tr>
<tr>
<td>PostW2</td>
<td>126.5 ± 0.6 (2)</td>
<td>138.5 ± 8.3 (3)</td>
<td>133.9 ± 5.0 (7)</td>
<td>141.6 ± 0.0 (1)</td>
<td></td>
</tr>
<tr>
<td>PostW3</td>
<td>129.7 ± 5.0 (3)</td>
<td>143.9 ± 12.2 (4)</td>
<td>131.6 ± 12.4 (14)</td>
<td>131.4 ± 10.2 (6)</td>
<td></td>
</tr>
<tr>
<td>PostW4</td>
<td>124.4 ± 0.0 (1)</td>
<td>141.2 ± 5.7 (3)</td>
<td>128.3 ± 17.3 (7)</td>
<td>–</td>
<td></td>
</tr>
</tbody>
</table>
Values are mean ± SD. Group size (n) and number of female (F) and male (M) subjects is noted. Values in parenthesis denote number of tests if different from group size. Groups are: Lo-Lo, living and training at low altitude; Hi-Hi3, living and training at altitude; Hi-Hi, living and training at altitude; Hi-HiLo, living at altitude and training at altitude and lower level. Swimming time trials are: TT50, 50 m freestyle; TT400, 400 m freestyle; TT100, 100 m at best stroke; TT200, 200 m at best stroke. * TT100 and TT200 values combined. Time points are: PRE, before the training camp; POST, immediately after the training camp; PostW1 to Post W4, 1 to 4 weeks after the training camp.

Within-group differences in TT50, TT400 and TT100/200 compared to previous tests are: † PRE; # POST; ¶ PostW1; § PostW2; & PostW3; * PostW4. Within-group differences for TT100 and TT200 are not shown because of small subsample sizes in some comparisons.
TABLE 3. Oxygen uptake kinetics parameters and $\dot{V}_o_{2\text{max}}$ during the 4x200-m incremental test (T4x200) before and after the intervention.

<table>
<thead>
<tr>
<th></th>
<th>Lo-Lo (n=11)</th>
<th>Hi-Hi3 (n=15)</th>
<th>Hi-Hi (n=16)</th>
<th>Hi-HiLo (n = 12)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time (s)</strong></td>
<td>PRE</td>
<td>144.8 ± 5.4</td>
<td>144.1 ± 9.6</td>
<td>138.3 ± 5.1</td>
<td>142.5 ± 15.7</td>
</tr>
<tr>
<td></td>
<td>POST</td>
<td>144.8 ± 5.6</td>
<td>148.8 ± 8.8</td>
<td>141.0 ± 4.7</td>
<td>142.8 ± 6.5</td>
</tr>
<tr>
<td><strong>Time delay (s)</strong></td>
<td>PRE</td>
<td>21.2 ± 4.0</td>
<td>20.2 ± 3.6</td>
<td>19.0 ± 1.4</td>
<td>21.2 ± 4.0</td>
</tr>
<tr>
<td></td>
<td>POST</td>
<td>22.0 ± 1.8</td>
<td>19.7 ± 3.6</td>
<td>19.8 ± 5.7</td>
<td>20.1 ± 6.6</td>
</tr>
<tr>
<td><strong>Time constant $\tau$ (s)</strong></td>
<td>PRE</td>
<td>10.0 ± 0.8</td>
<td>11.3 ± 4.3</td>
<td>9.5 ± 0.8</td>
<td>13.9 ± 6.2</td>
</tr>
<tr>
<td></td>
<td>POST</td>
<td>10.5 ± 1.9</td>
<td>15.3 ± 6.3</td>
<td>10.3 ± 4.0</td>
<td>13.5 ± 3.1</td>
</tr>
<tr>
<td><strong>$\dot{V}<em>o</em>{2\text{max}}$ (ml·min$^{-1}$)</strong></td>
<td>PRE</td>
<td>3,370 ± 607</td>
<td>3,522 ± 851</td>
<td>3,757 ± 774</td>
<td>3,865 ± 718</td>
</tr>
<tr>
<td></td>
<td>POST</td>
<td>3,435 ± 595</td>
<td>3,573 ± 891</td>
<td>3,774 ± 727</td>
<td>3,916 ± 777</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Groups are: Lo-Lo, living and training at low altitude (4 weeks); Hi-Hi3, living and training at altitude (3 weeks); Hi-Hi, living and training at altitude (4 weeks); Hi-HiLo, living at altitude and training at altitude and lower level (4 weeks). Time, duration of the maximal 200-m test; Time delay, delay of the principal (fast) component (= phase I duration); $\tau$, time constant of the principal component; $\dot{V}_o_{2\text{max}}$, maximal oxygen uptake. See text for details. $P$, type I error probability in ANOVA interaction group x test (PRE, POST).