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Effect of high-intensity hypoxic training on sea-level swimming performances

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¹*Institute for Exercise and Environmental Medicine, Presbyterian Hospital of Dallas and University of Texas Southwestern Medical Center at Dallas, Dallas, Texas 75231; and* ²*Department of Kinesiology, Faculty of Human Movement Sciences, Vrije Universiteit, 1081 BT Amsterdam, The Netherlands*

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Truijens, M. J., H. M. Toussaint, J. Dow, and B. D. Levine. Effect of high-intensity hypoxic training on sea-level swimming performances. *J Appl Physiol* 94: 733–743, 2003. First published October 11, 2002; 10.1152/jappphysiol.00079.2002.—The objective of this study was to test the hypothesis that high-intensity hypoxic training improves sea-level performances more than equivalent training in normoxia. Sixteen well-trained collegiate and Masters swimmers (10 women, 6 men) completed a 5-wk 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 they were assigned to either hypoxic [Hypo; inspired O₂ fraction (F_IO₂) = 15.3%, equivalent to a simulated altitude of 2,500 m] or normoxic (Norm; F_IO₂ = 20.9%) interval training in a randomized, double-blind, placebo-controlled design. All pool training occurred under Norm conditions. The primary performance measures were 100- and 400-m freestyle time trials. Laboratory outcomes included maximal O₂ uptake ($\dot{V}O_{2\max}$), anaerobic capacity (accumulated O₂ deficit), and swimming economy. Significant ($P = 0.02$ and <0.001 for 100- and 400-m trials, respectively) improvements were found in performance on both the 100- [Norm: -0.7 s (95% confidence limits: $+0.2$ to -1.7 s), -1.2% ; Hypo: -0.8 s (95% confidence limits: -0.1 to -1.5 s), -1.1%] and 400-m freestyle [Norm: -3.6 s (-1.8 to -5.5 s), -1.2% ; Hypo: -5.3 s (-2.3 to -8.3 s), -1.7%]. There was no significant difference between groups for either distance (ANOVA interaction, $P = 0.91$ and 0.36 for 100- and 400-m trials, respectively). $\dot{V}O_{2\max}$ was improved significantly (Norm: 0.16 ± 0.23 l/min, $6.4 \pm 8.1\%$; Hypo: 0.11 ± 0.18 l/min, $4.2 \pm 7.0\%$). There was no significant difference between groups ($P = 0.58$). We conclude that 5 wk of high-intensity training in a flume improves sea-level swimming performances and $\dot{V}O_{2\max}$ in well-trained swimmers, with no additive effect of hypoxic training.

hypoxia; 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 (IHT), 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 (35). More recently, hypoxic training has been shown to enhance the transcription of mRNA for hypoxia inducible factor-1 α , although 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, 36, 41). However, the results of such training showed no significant effects on aerobic performance markers, such as maximal oxygen uptake ($\dot{V}O_{2\max}$) (24, 36, 41).

In contrast, some previous reports (4, 24, 36) 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 (23). Moreover, several studies demonstrated that, for anaerobic capacity to increase, high-intensity training must be done (33, 34).

We hypothesized that for sports of relatively high intensity and 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 <2 min in duration. In this regard, Ogita and Tabata (27) found a 10% increase in

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Table 1. *Subject characteristics*

Subject No.	C/M	Gender	Specialization	Level	PB	RB* (date)	Age, yr	Height, cm	Body mass, kg	%Fat	$\dot{V}O_{2\max}$, l/min
<i>Normoxic group</i>											
1	C	F	Free, fly	OT, NCAA, Nat	59.4		20	173.5	70.5	17.1	64.6
2	C	F	IM	OT, NCAA, Nat	62.5	62.5(01)	19	160.3	54.7	12.9	58.4
3	C	F	Breast	Nat	60.0		21	177.7	66.2	13.9	44.1
4	M	F	Breast, free	USMS			27	166.8	68.2	17.1	54.7
5	M	M	Free	NCAA, OT, Nat, USMS	51.4	51.7(01)	30	187.5	81.9	8.5	48.4
6	M	M	Free, fly	NCAA, Nat, Int, USMS, WR Masters	53		38	185.8	87.2	13.5	47.8
7	M	M	Free	USMS, Nat	54	62.3(00)	45	176.5	80.6	12.3	40.2
8	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
<i>Hypoxic group</i>											
1	C	F	Free, fly	Nat	58.4	59.7(01)	19	165.8	63	12.3	53.7
2	C	F	Free	Nat	60.2	61.2(01)	19	176	68.7	18.2	41.5
3	C	F	Breast	NCAA, Nat	61.7	63.5(01)	21	175.3	65	13.8	45.9
4	M	F	Back, fly	NCAA, Nat, Int, AR, OT, USMS	59.2		24	181	85.5	15.7	46.3
5	M	M	Back, free	OT, Nat	52		27	185.9	77.3	8.5	56.3
6	M	M	IM, back	USMS	57		39	171.1	80.6	19.1	45.8
7	M	M	Free	Recr			54	167.8	75.2	23.8	51.7
8	M	F	Free	Recr		85(01)	27	173	62.7	14	41.4
Average							28.8	174.5	72.3	15.7	47.8

C/M, collegiate or Masters; F, female; M, male; PB, personal best; RB, recent best; *, i.e., best time on 100-m freestyle within a year from the start of the study (year in parentheses; note that some elite specialists do not have a recent best time in the 100-m freestyle); $\dot{V}O_{2\max}$, maximal oxygen uptake as measured during the pretest; AR, American Record; Int, International Championships level; Nat, National Championship level; NCAA, National Collegiate Athletic Association All-American; OT, Olympic trials; Recr, recreational level; USMS, US Masters Swimming All-American; WR Masters, World Record Masters.

anaerobic capacity, as measured by the accumulated oxygen deficit (AOD) after only 2 wk of high-intensity hypoxic training in nine 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 and short duration; however, none of these studies generated conclusive evidence in support of this method and all suffered from several limitations. Therefore, the present study was designed to investigate the effect of high-intensity intermittent hypoxic training on well-trained swimmers by 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 and/or US Masters Swimming 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 swimmers. Individual descriptive characteristics are provided in Table 1.

All subjects were sea-level residents and gave 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.

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; inspired oxygen fraction (F_{IO_2}) = 20.9 ± 0.1%; n = 8; 3 men, 5 women] or experimental group [living in normoxia and high-intensity training in hypoxia (F_{IO_2} = 15.3 ± 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

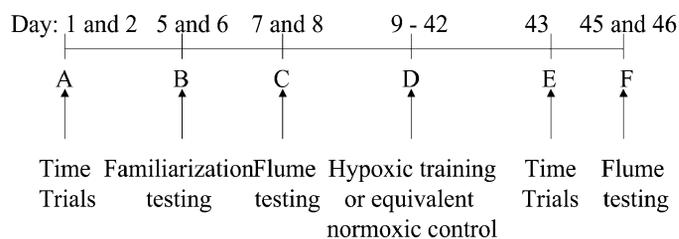


Fig. 1. The study consisted of the following phases: initial 100- and 400-m time trials (A); single initial session for familiarization with testing and training procedures (B); initial laboratory testing (C); 5-wk training period with swimmers divided into 2 groups by balanced randomization (D); repeat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

participated in a 5-wk 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, 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 to ensure a minimal "dead space" of 30 ml (40). The inspiratory tube (length: 1.65 m; diameter: 36 mm) was connected to a large reservoir that 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 data were analyzed. The oxygen 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 were 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 >10 bouts, 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 400-m freestyle. Pre- and posttest time trials were conducted in an Olympic-sized pool in Dallas between 4:30 and 7:00 PM. Time trials were conducted similar to normal swimming events by starting in groups of four to six swimmers. Subjects had ~45 min of rest between events and swam the 100-m event first. Starts were made from the starting blocks by using a whistle as the starting signal. Finish times were measured in duplicate by stopwatch, with one stopwatch functioning as backup only. Day-to-day variability in 100-m performance was determined in a separate series of time trials and was calculated as the mean \pm 95% confidence limits of the individual percent difference between the two 100-m times. These measurements were conducted 7 mo after the training intervention by 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 h between tests.

Flume testing. A supramaximal test was used to determine both $\dot{V}O_{2\max}$ 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, swimmers wore the same mask as described above, with the inspiratory tube in open connection with room air, whereas the expiratory tube was ultimately connected to a series of Douglas bags.

$\dot{V}O_{2\max}$. Oxygen uptake ($\dot{V}O_2$) was measured simultaneously with the Douglas bag technique and an on-line system for breath-by-breath measurements. Douglas bags were considered standard for all $\dot{V}O_2$ measurements. Breath-by-breath data served as backup and was used for identification of steady states, plateaus, and kinetic patterns in the $\dot{V}O_2$ time curve. The on-line system for breath-by-breath measurements consisted of four one-way valves to direct flow, two sample lines to measure gas fractions, and a turbine flow meter (VMM, Interface Associates) to measure ventilation. Breath-by-breath data were 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 they were 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 before the study confirmed their equivalence (slope = 1.0, intercept = 0.0, $r^2 = 1.0$). $\dot{V}O_{2\max}$ was defined as the highest $\dot{V}O_2$ measured from at least a 30-s Douglas bag. A plateau in $\dot{V}O_2$ during supramaximal swimming was achieved in 100% of these tests documenting the identification of $\dot{V}O_{2\max}$. The observation that $\dot{V}O_2$ remained constant or decreased slightly during continued supramaximal exercise was defined as a plateau. In addition, heart rate was monitored continuously (Polar CIC, 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. (23) adapted for swimming (26). Details of the derivation of this relationship are provided in the APPENDIX. $\dot{V}O_2$ was measured at five 2-min submaximal swimming speeds at intensities ranging from 40 to 80% $\dot{V}O_{2\max}$, 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 min after the last submaximal swim, subjects performed a supramaximal swim at an individually determined speed to exhaust the subject between 2 and 4 min as described in *Flume testing*. 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 $\dot{V}O_2$ during swimming, gives the power generated by anaerobic metabolic processes. Power was transformed to its energy equivalent and expressed in milliliters per kilogram per minute and milliliters per kilogram lean body mass per minute. Fat-free mass was calculated from skinfold measurements (Lange skinfold calipers, Cambridge Scientific Industries, Cambridge, MA), taken from eight sites by using customized software employing the equations of Siri (30).

The same procedure, including individual determination of swimming economy, was performed both pre- and posttraining. To allow comparison with previous reports in the literature [Ogita and Tabata (27)], a secondary calculation of anaerobic capacity was also made by 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-min 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

Hb and hematocrit were measured and used to monitor gross hematologic 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 min. Hb was determined by using an Instrumentation Laboratories CO-oximeter. Hematocrit 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 of being of low, moderate, or high intensity.

Training characterization. To determine precisely the demands of a typical flume training session, swimming velocity, $\dot{V}O_2$, minute ventilation (\dot{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. posttraining) and treatment (hypoxic vs. normal)] was used for analysis by using SigmaStat 2.03 (SPSS). A separate two-way ANOVA with swimmer type (collegiate vs. Masters) and treatment (normal vs. hypoxic) 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's 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 $\dot{V}O_{2\text{max}}$ 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 $\dot{V}O_{2\text{max}}$, 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

Table 2. Hematologic parameters and performance indexes

	Group	Norm	Hyp
100-m time, s			
Pre	65.39 \pm 7.64	64.36 \pm 5.23	66.43 \pm 9.76
Post	64.62 \pm 7.33*	63.62 \pm 5.76	65.62 \pm 8.93
%Change (95% confidence limits)	-1.2 (-0.3 to -2.1)	-1.2 (+0.6 to -3.0)	-1.1 (+0.0 to -2.2)
400-m time, s			
Pre	305.91 \pm 33.19	301.08 \pm 27.12	310.74 \pm 39.65
Post	301.43 \pm 32.09*	297.46 \pm 26.70*	305.41 \pm 38.18*
%Change (95% confidence limits)	-1.5 (-0.9 to -2.0)	-1.2 (-0.5 to -1.9)	-1.7 (-0.5 to -2.9)
$\dot{V}O_{2\text{max}}$, l/min			
Pre	2.99 \pm 0.56	3.05 \pm 0.58	2.92 \pm 0.57
Post	3.12 \pm 0.50*	3.22 \pm 0.48*	3.03 \pm 0.53
%Change (95% confidence limits)	+5.3 (\pm 7.4)	+6.4 (\pm 8.1)	+4.2 (\pm 7.0)
AnaCap, ml/kg(lbm)			
Pre	31.06 \pm 11.87	32.26 \pm 8.16	29.86 \pm 15.23
Post	27.95 \pm 13.30	28.16 \pm 14.33	27.75 \pm 13.16
%Change (95% confidence limits)	-2.0 (\pm 51.0)	-12.1 (\pm 35.2)	+8.1 (\pm 64.1)
HR _{max} , beats/min			
Pre	184 \pm 7	183 \pm 6	186 \pm 8
Post	187 \pm 5	186 \pm 2	188 \pm 6
$\dot{V}_{E\text{max}}$ (BTPS), l/min			
Pre	97.74 \pm 20.63	98.91 \pm 21.06	96.58 \pm 21.58
Post	108.09 \pm 19.94*	110.80 \pm 20.42*	105.38 \pm 20.45*
Economy, W/V ³			
Pre	460.5 \pm 110.4	463.6 \pm 89.9	457.3 \pm 134.2
Post	435.9 \pm 92.6	433.7 \pm 84.2	438.1 \pm 106.2
Hemoglobin, g/dl			
Pre	14.5 \pm 1.3	14.4 \pm 1.3	14.5 \pm 1.4
Post	14.5 \pm 1.1	14.2 \pm 0.9	14.7 \pm 1.2
Hematocrit, %			
Pre	40 \pm 2.5	39.4 \pm 2.9	40.5 \pm 2.1
Post	39.5 \pm 2.8	39.5 \pm 3.2	39.5 \pm 2.7

Values are means \pm SD. Pre, before training; Post, after training; %change, Mean of the individual percent differences; $\dot{V}O_{2\text{max}}$, maximal oxygen uptake; Anacap, anaerobic capacity; HR_{max}, maximal heart rate; $\dot{V}_{E\text{max}}$, maximal ventilation; Group, all subjects combined; Norm, normoxic subjects; Hyp, hypoxic subjects. *Significant *F* statistic ($P < 0.05$; Tukey's test). †Due to technical problems, heart rate data represents 13 (Norm: 7; Hypo: 6) subjects for pretesting and only 5 (Norm: 3; Hypo: 2) for posttesting.

Table 3. *Flume training characteristics*

	%HR _{max} Training	%V̇E _{max} Training	%V̇O _{2max}		%P _{max}		%Aer	
			30 s	60 s	30 s	60 s	30 s	60 s
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. %HR_{max}, percentage of maximal heart rate; %V̇E_{max}, percentage of maximal ventilation; %V̇O_{2max}, percentage of V̇O_{2max}; and %P_{max}, exercise intensity related to maximal power output as obtained during the pretest; %Aer, percentage of %V̇O_{2max}. %P_{max}, and %Aer were divided into 30-s (30 s) and 60-s (60 s) intervals. *Significant *t* statistic (*P* < 0.05; unpaired *t*-test).

no significant differences between the groups in terms of time trial performance, V̇O_{2max}, anaerobic capacity, or hematologic parameters.

Training

All subjects completed a minimum of 12 flume training sessions (out of a maximum of 14 sessions). Three subjects missed two sessions, and six subjects missed one session.

During both the exercise bouts of 30 s and 1 min, the hypoxic group trained with significantly (*P* < 0.02) reduced V̇O₂ compared with the normoxic group (hypoxic group: 69.0 ± 13.9% of pretraining V̇O_{2max} for 30 s, and 75.7 ± 9.2% for 1-min sessions; normoxic group: 90.9 ± 10.3% for 30 s, and 93.8 ± 11.5% for 1-min sessions). Furthermore, the power output during training, expressed as percentage of the maximal power output as determined on the pretest, was significantly (*P* < 0.01) lower, by 7.2 ± 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 with the normoxic group (65.2 ± 12.7 vs. 78.1 ± 11.3% and 74.1 ± 6.5 vs. 81.5 ± 8.7% for the 30- and 60-s bouts, respectively).

Both groups trained at very high ventilations (normoxic group: 105 ± 13.5%; hypoxic group: 111 ± 13.4% of pretest maximal V̇E) and heart rates (normoxic group: 96.4 ± 1.6%; hypoxic group: 96.5 ± 3.0% of pretest maximal heart rate), consistent with a maximal effort. No significant differences between groups were found. In summary, high-intensity training under hypoxic conditions was accomplished with a substan-

tially reduced oxygen flux, a slightly reduced power output, and consequently required a greater anaerobic contribution compared with 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 per 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.

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 χ^2 testing with Fisher's exact test), indicating the successfulness of the design.

Response to Training

Hematologic changes. No significant changes in Hb or hematocrit were observed.

Time trial performance. Both groups improved significantly (*P* = 0.02 for 100-m event, *P* < 0.001 for 400-m event) in performance on both the 100- [normoxic group: -0.7 s (95% confidence limits +0.2 to -1.7 s) and -1.2% (95% confidence limits +0.6 to -3.0%); hypoxic group: -0.8 s (95% confidence limits -0.1 to -1.5 s) and -1.1% (95% confidence limits +0.0 to -2.2%)] and 400-m freestyle [normoxic group: -3.6 s (95% confidence limits -1.8 to -5.5 s) and -1.2%

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

	Week 1		Week 2				Week 3				Week 4				Week 5					
	Normoxia		Hypoxia		Normoxia		Hypoxia		Normoxia		Hypoxia		Normoxia		Hypoxia		Normoxia		Hypoxia	
	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C
Frequency	1.3 ± 1.3	6.7 ± 0.6	1.5 ± 1.7	7.3 ± 0.6	1.8 ± 1.5	7.3 ± 1.2	2.3 ± 1.2	8 ± 2	2 ± 1.2	7.7 ± 0.6	2 ± 1.4	8.3 ± 1.5	1.8 ± 1.5	5.7 ± 2	1.5 ± 1.7	6.3 ± 2.3	1.3 ± 1	5.7 ± 2.3	1.3 ± 1.2	5.3 ± 3.1
Distance, km	3.7 ± 4.1	35.8 ± 6.9	4.2 ± 4.9	39.8 ± 5.9	4.7 ± 4.5	39.6 ± 8.9	6.3 ± 3.4	44.7 ± 11.8	5.1 ± 3.4	44.3 ± 4.1	5.4 ± 3.9	46.7 ± 7.9	4.4 ± 4.2	34.6 ± 6.1	4.1 ± 4.8	38.1 ± 17.7	2.5 ± 1.9	33.2 ± 14.4	3.7 ± 3.2	31.4 ± 19.6
Intensity	Low-moderate		Low-moderate		Low-moderate		Low-moderate		Low-moderate		Low-moderate		Low-moderate		Low-moderate		Low-moderate		Low-moderate	

Values are means ± 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).

(95% confidence limits -0.5 to -1.9%); hypoxic group: -5.3 s (95% confidence limits -2.3 to -8.3 s) and -1.7% (95% confidence limits -0.5 to -2.9%). There was no significant difference between groups for either distance (ANOVA interaction $P = 0.91$ and 0.36 for 100- and 400-m events, respectively). This difference was nearly fivefold greater than the mean of individual differences between two 100-m time trials of -0.3% (95% confidence limits $+0.5$ to -1.0%) (Fig. 2, A and B). No significant difference between Masters and collegiate swimmers was observed (ANOVA interaction $P = 0.43$ and 0.49 for 100- and 400-m events, respectively).

$\dot{V}O_{2\max}$ and maximal $\dot{V}E$ and heart rate. Both groups improved $\dot{V}O_{2\max}$ significantly ($P = 0.02$) (normoxic group: $+0.16 \pm 0.23$ l/min, $+6.4 \pm 8.1\%$; hypoxic group: $+0.11 \pm 0.18$ l/min, $+4.2 \pm 7.0\%$) (Fig. 3, A and B). No significant difference between groups could be detected ($P = 0.58$).

Maximal $\dot{V}E$ significantly ($P < 0.001$) increased in both groups (normoxic group: 11.90 l/min, $13.6 \pm 12.7\%$; hypoxic group: 8.81 l/min, $10.0 \pm 7.6\%$). There was no significant difference between groups ($P = 0.71$). No change in maximal heart rate was observed.

Anaerobic power and capacity. All individual relationships between submaximal $\dot{V}O_2$ and V^3 were highly linear ($r = 0.98 \pm 0.03$). There were no sig-

nificant differences in anaerobic capacity between ($P = 0.79$) or within ($P = 0.80$) groups after training (Fig. 3 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 in either hypoxia or normoxia.

Predicting swimming performance: multiple regression. With the data of $\dot{V}O_{2\max}$, anaerobic capacity, and swimming economy, we were able to predict 100- and 400-m times with an accuracy of 72 (SE of estimate: 4.1 s) and 67% (SE of estimate: 19.6 s), respectively. However, the change in performance that resulted from the training period could not be predicted with this simple linear, three-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 400 m. However, this improvement was not enhanced by performing such training under hypoxic conditions. This conclusion is strengthened by the carefully matched groups containing

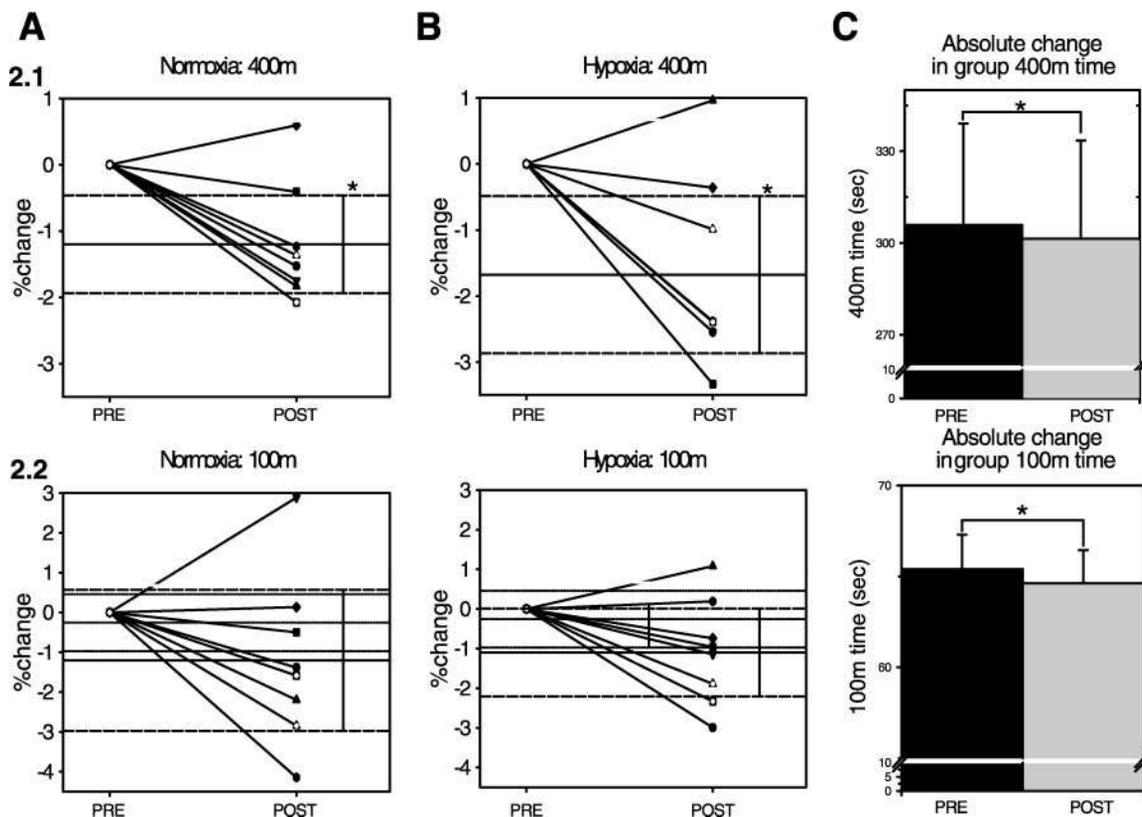


Fig. 2. Percent change in 400- (top) and 100-m (bottom) time trial performances for each individual subject training in normoxia (A), hypoxia (B), and absolute changes for all subjects together (C), before (pre) and after (post) the training period. Similar symbols are used for each pair of matched subjects.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

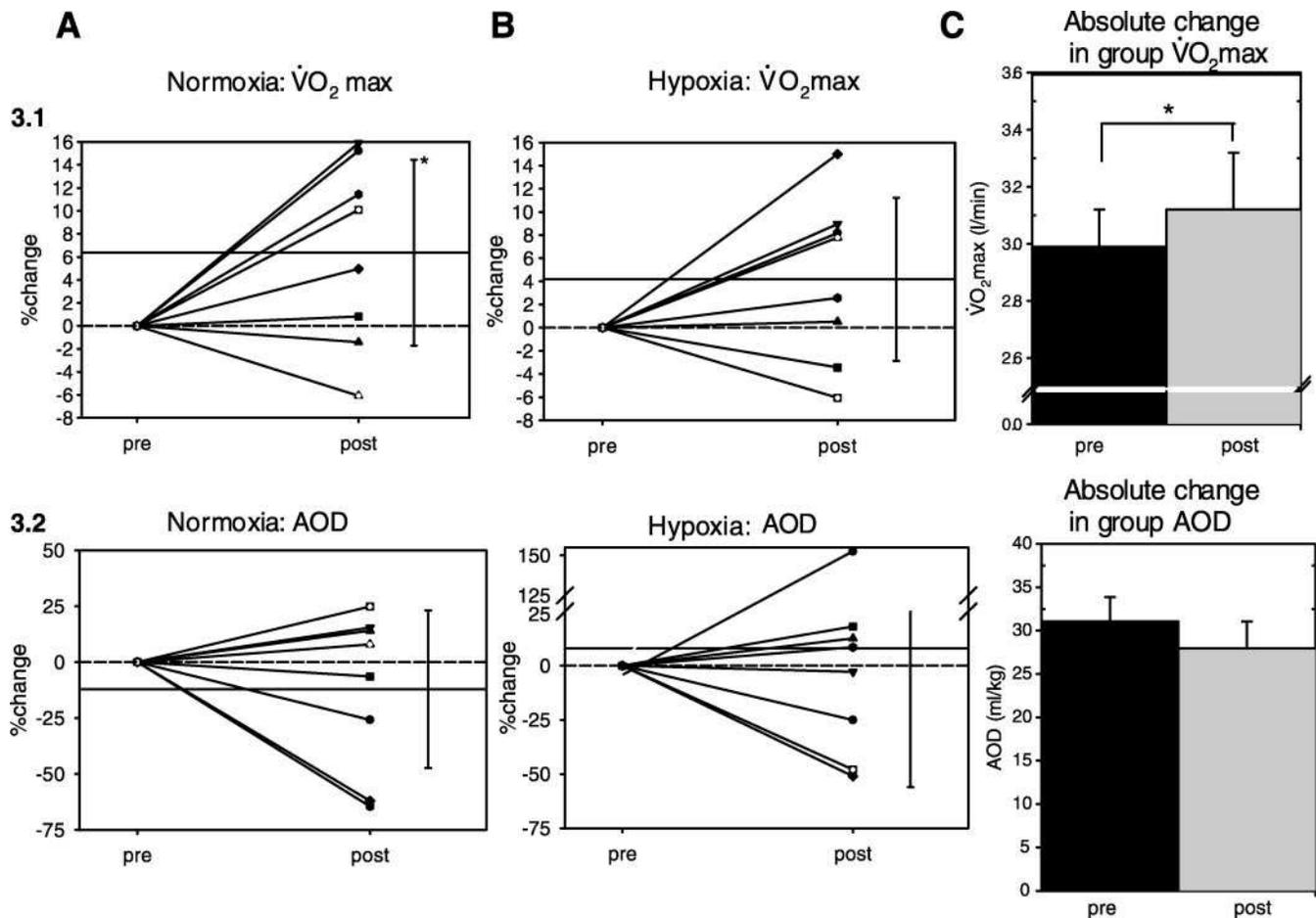


Fig. 3. Percent change in maximal oxygen consumption ($\dot{V}O_{2\max}$; top) and anaerobic capacity (bottom) for each individual subject training in normoxia (A), hypoxia (B), and absolute changes for all subjects together (C), before and after the training period. Horizontal solid line with vertical bar, mean \pm SD. Horizontal dashed line, zero level. *Significant *F* statistic ($P < 0.05$; repeated-measures ANOVA).

well-trained swimmers and by 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 that have examined the effects of hypoxic exercise. For example, Terrados et al. (36) investigated the effect of intermittent hypoxic training in eight elite cyclists randomly assigned to either hypobaric hypoxia (2,300-m elevation) 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 $\dot{V}O_{2\max}$ or maximal power output in five elite triathletes after intermittent hypoxic training (4,000 m), although 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 athletes trained in a hypobaric chamber at a simulated altitude of 2,500 m, whereas eight fitness-matched controls trained at sea level. Again, no significant differences between groups were found after the first posttest 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 $\dot{V}O_{2\max}$ between groups were found. Unfortunately, training was not controlled during this intermediate period, which limits the strength of the conclusion.

In summary, previous work in both untrained subjects and well-trained athletes has not convincingly demonstrated an additive effect of hypoxia superim-

posed on endurance training for improvements in aerobic power, at least during whole body cycling exercise. On the basis of these limited data, several other investigators (4, 28, 35, 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 that training was predominantly aerobic in nature (ranging from 60 to 85% of $\dot{V}O_{2\max}$). 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 wk of high-intensity, intermittent hypobaric hypoxic (3,000 m) training on anaerobic capacity and $\dot{V}O_{2\max}$ in nine collegiate swimmers. No significant change in $\dot{V}O_{2\max}$ 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 whether the improvement in anaerobic capacity resulted in faster times in a swimming pool. Third, only 14 min of the 2-h training load were specified; the other 106 min 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 posttest, AOD was measured at higher water flow rates than during the pretest. 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 $\dot{V}O_{2\max}$ 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

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, 22, 34). 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 $\dot{V}O_2$ and oxygen transport capacity (14, 34), findings that support the results of the present study. It should be noted that the increase in $\dot{V}O_{2\max}$ was not related to a difference in Hb because no change in Hb was observed. This lack of increase in Hb is likely to be due to the short hypoxic exposure time, and it 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 $\dot{V}O_{2\max}$ 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), which increase the tolerance for products of anaerobic metabolism and delay the onset of fatigue. Indeed, hypoxia is often thought to increase this effect by putting an additional stress on the body (28).

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. (29) 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-wk period of "living high" (at a simulated altitude of 3,000 m) 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. (35). By training a small muscle mass (one leg), they enabled the subjects to train at the same absolute work rate in hypoxia compared with normoxia. However, in the present study, the hypoxic group trained at a significantly ($P < 0.01$) lower percentage of maximal power output as was obtained during the pretest. 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 1 min, work is reduced proportionately to the reduction in $\dot{V}O_2$ since maximal anaerobic capacity is apparently not affected by hypoxia (23).

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 100- and 400-m pool swimming performance and $\dot{V}O_{2\max}$ 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 flume training; changes in swimming performance and $\dot{V}O_{2\max}$ 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 there-

fore speculate, but cannot 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 before 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, careful matching of subjects created pairs who 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 cannot 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 h/wk for a weekly distance of 38.1 ± 11.7 km outside of the flume sessions. Therefore, we considered whether this additional training may have led to some overtraining, which limited 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 ~ 20 min. This may seem rather low compared with previous studies on intermittent hypoxic training, where the hypoxic stimulus was at least an hour each session. However, the studies of Terrados et al. (35) and Vogt et al. (42) demonstrated that a hypoxic stimulus of half an hour, three to five 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 AOD 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 $\dot{V}O_2$ may be nonlinear (46). 2) When a linear relationship between exercise intensity and $\dot{V}O_2$ is assumed, 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, and these efficiencies may in fact not be equal (6, 12). 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 aerobic power output ($P_{V_{O_2}}$)- V^3 relationships during both pre- ($r^2 = 0.97$) and posttests ($r^2 = 0.99$) justifies the use of this relationship to estimate the AOD according to the method of Medbo et al. (23). Nevertheless, large variations in the change in anaerobic capacity were observed. The mean percent change was $-2.0 \pm 51.0\%$, whereas other training studies that measured AOD according to the method of Medbo et al. reported percentages change of $+28$ (34) and $+10\%$ (27). 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 pretest regression equations to determine AOD during both pre- and posttest. This way, a possible source of variability [i.e., the day-to-day variability in submaximal \dot{V}_{O_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 percent 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 pretest regression equations for analysis of both pre- and posttest data.

In conclusion, 5 wk of high-intensity training in a flume significantly improved sea-level swimming performances and $\dot{V}_{O_{2\max}}$ in well-trained swimmers. There was no significant, additive effect of hypoxic training under the conditions of this controlled experiment.

APPENDIX

The equation for the power balance for swimming (39) is taken as starting point for determining the relationship between power output (P_o) and swimming velocity

$$P_o - (P_d + P_k) = dE/dt \quad (A1)$$

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 (P_{met}) according to (16)

$$P_o = P_{\text{met}} \cdot e_m \quad (A2)$$

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

$$P_{\text{met}} = P_{\text{met,aer}} + P_{\text{met,an}} \quad (A3)$$

where subscripts of aer and an identify aerobic and anaerobic measurements, respectively. Combining Eqs. 1–3 gives

$$P_{\text{mech}} = P_{\text{met,aer}} \cdot e_{m,\text{aer}} + P_{\text{met,an}} \cdot e_{m,\text{an}} = P_d + P_k + dE/dt \quad (A4)$$

where P_{mech} is mechanical power.

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

$$dE/dt = 0 \quad \text{and} \quad P_o = P_d + P_k \quad (A5)$$

Several studies demonstrated that active drag is related to the square of the swimming velocity (38, 37). Consequently

the power to overcome drag is related to the V^3 ($V \cdot V^2$) and a drag factor (K)

$$P_d = KV^3 \quad (A6)$$

How P_k is related to velocity is not perfectly clear yet. Introducing the concept of propelling efficiency (e_p) (16, 37) helps to solve this problem. Swimming at constant velocity

$$e_p = P_d/P_o = P_d/(P_d + P_k) \quad (A7)$$

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

$$P_o = P_d/e_p = K \cdot V^3/e_p = K/e_p \cdot V^3 \quad (A8)$$

K/e_p is constant, and thus P_o is related to V^3 . Evidence for this relationship is experimentally generated by Toussaint et al. (37). Combining Eqs. 2 and 8 gives the expression for metabolic energy expenditure

$$P_{\text{met}} = KV^3/e_m \cdot e_p \quad (A9)$$

When e_m is assumed to be constant, the linear relationship between P_{met} and V^3 is established. This relationship is experimentally supported by Toussaint et al. (37) and Ogita et al. (26).

When it is considered that at low exercise intensities the total mechanical power output is primarily generated by means of aerobic metabolic processes and that the measurement of \dot{V}_{O_2} is a reliable measure of aerobic energy production, the relationship can be transformed into

$$\dot{V}_{O_2} = KV^3 \quad (A10)$$

which can be transformed to its power equivalent according to

$$P\dot{V}_{O_2} = 1/60 \cdot 10^3 \cdot [4.1868 \cdot (4.047 + \text{RER})] \cdot \dot{V}_{O_2} \quad (A11)$$

where RER is the respiratory exchange ratio.

This linear relationship between $P_{V_{O_2}}$ and V^3 only holds for steady-state exercises within the range of aerobic performance (37), i.e., below 80% of $\dot{V}_{O_{2\max}}$ (1). Now, by measuring \dot{V}_{O_2} at several submaximal intensities, the linear relationship can be experimentally established and used for extrapolation to higher exercise intensities to determine anaerobic capacity.

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