"Living high-training low": effect of moderate-altitude acclimatization with low-altitude training on performance

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Levine, Benjamin D., and James Stray-Gundersen. "Living high-training low": effect of moderate-altitude acclimatization with low-altitude training on performance. *J. Appl. Physiol.* 83(1): 102–112, 1997.—The principal objective of this study was to test the hypothesis that acclimatization to moderate altitude (2,500 m) plus training at low altitude (1,250 m), "living high-training low," improves sea-level performance in well-trained runners more than an equivalent sea-level or altitude control. Thirty-nine competitive runners (27 men, 12 women) completed *1*) a 2-wk lead-in phase, followed by *2*) 4 wk of supervised training at sea level; and *3*) 4 wk of field training camp randomized to three groups: "high-low" ($n = 13$), living at moderate altitude (2,500 m) and training at low altitude (1,250 m); "high-high" ($n = 13$), living and training at moderate altitude (2,500 m); or "low-low" ($n = 13$), living and training in a mountain environment at sea level (150 m). A 5,000-m time trial was the primary measure of performance; laboratory outcomes included maximal O_2 uptake ($\dot{V}O_{2max}$), anaerobic capacity (accumulated O_2 deficit), maximal steady state (MSS; ventilatory threshold), running economy, velocity at $\dot{V}O_{2max}$, and blood compartment volumes. Both altitude groups significantly increased $\dot{V}O_{2max}$ (5%) in direct proportion to an increase in red cell mass volume (9%; $r = 0.37$, $P < 0.05$), neither of which changed in the control. Five-kilometer time was improved by the field training camp only in the high-low group (13.4 ± 10 s), in direct proportion to the increase in $\dot{V}O_{2max}$ ($r = 0.65$, $P < 0.01$). Velocity at $\dot{V}O_{2max}$ and MSS also improved only in the high-low group. Four weeks of living high-training low improves sea-level running performance in trained runners due to altitude acclimatization (increase in red cell mass volume and $\dot{V}O_{2max}$) and maintenance of sea-level training velocities, most likely accounting for the increase in velocity at $\dot{V}O_{2max}$ and MSS.

altitude; hypoxia; training; exercise; sports

ALTITUDE TRAINING is frequently used by competitive athletes to improve sea-level performance (14). However, the objective benefits of altitude training are controversial (21). On one hand, acclimatization to high altitude results in central and peripheral adaptations that improve oxygen delivery and utilization (4, 8, 26, 31, 34, 38, 44). Moreover, hypoxic exercise may increase the training stimulus, thus magnifying the effects of endurance training (7, 41). Conversely, hypoxia at altitude limits training intensity (23), which in elite athletes may result in relative deconditioning.

Numerous anecdotal reports since the 1940s have suggested that endurance athletes may achieve some benefit from altitude training for sea-level performance (3, 12, 15). However, incomplete characterization of athletic performance, lack of appropriate controls, and small subject numbers have complicated the interpretation of the majority of previous studies of altitude training (3, 11, 12, 15, 17, 18). When appropriate control groups have been included, living and training at altitude have not been proven to be advantageous compared with equivalent training at sea level (1).

We reasoned that if athletes could live at moderate altitude, above 2,500 m (22), but train at low altitude, below 1,500 m, they could acquire the physiological advantages of altitude acclimatization for maximizing oxygen transport, without the detraining associated with hypoxic exercise (24). The present study was designed to test this hypothesis by examining *1*) competitive athletes, already well trained under supervised conditions at sea level; *2*) adequate numbers of subjects to allow sufficient statistical power to detect meaningful differences among treatment and control groups; *3*) a balanced, randomized design providing both sequential and parallel controls; *4*) high-dose iron supplementation in iron-deficient athletes to ensure appropriate acclimatization to high altitude; and *5*) comprehensive characterization of performance including track and laboratory-based markers.

METHODS

Subjects

Forty-one distance runners were recruited from collegiate track and cross-country teams, local running clubs, and USA Track and Field development teams, and 39 (27 men, 12 women, aged 18–31 yr) completed all the testing and training sessions. Sample size was estimated from pilot data demonstrating an increase in maximal O_2 uptake ($\dot{V}O_{2max}$) of 5% (3 $ml \cdot kg^{-1} \cdot min^{-1}$) with an SD of 3.2 $ml \cdot kg^{-1} \cdot min^{-1}$, requiring 11 athletes/group ($\beta = 0.80$, $\alpha = 0.05$). Athletes were required to be competitive at a distance between 1,500 m and the marathon and to have a recent personal best 5,000-m time (or equivalent) of <16:30 for men and <18:30 for women. All were sea-level residents and could not have been to altitude above 1,500 m for a period exceeding 1 wk in the previous 10 mo. All subjects gave their voluntary written informed consent to a protocol approved by the Institutional Review Board of the University of Texas Southwestern Medical Center at Dallas.

Study Design

An outline of the study design is shown in Fig. 1; it consists of four major phases.

Sea-level lead-in phase. Athletes were brought to Dallas, Texas (150 m), 2–4 wk after the spring track season for a 2-wk period of supervised training at sea level and familiarization with laboratory testing procedures. We have previously shown that this phase is necessary and sufficient to bring all the athletes to an equivalent level of training readiness, and that

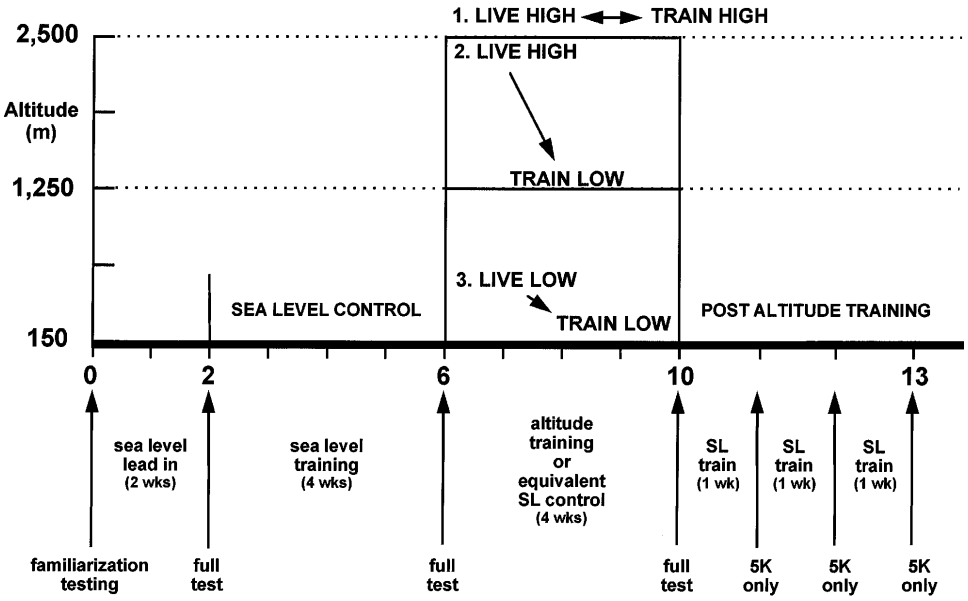

Fig. 1. The study consisted of the following phases: *1*) initial time trial and series of laboratory tests for familiarization; *2*) sea-level (SL) "lead-in" phase designed to overcome effect of supervised training and to begin aggressive iron supplementation to normalize serum ferritin; *3*) full set of laboratory testing to serve as baseline; *4*) 4 wk of sea-level training in Dallas, TX, designed to maximize fitness in all athletes and to serve as a longitudinal sea-level control; *5*) 2nd set of baseline measurements before field training camp; and *6*) 4 wk of training camp with athletes divided into 3 groups by using a balanced randomized design: living high (2,500 m), training low (1,250 m; 1); living high (2,500 m), training high (2,500–2,700 m; 2); and living low (150 m), training low (150 m; 3). After 4 wk of training camp, athletes returned to Dallas for *7*) repeat series of laboratory testing and *8*) repeat 5-km (5K) time trials each week for total of 3 wk to determine optimal timing of competition.

may account for a substantial portion of the supervised training camp effect observed in many training studies (23, 39). During this phase, serum ferritin was measured for the assessment of bone marrow iron stores, and iron maintenance or replacement therapy was initiated for all subjects (37).

Sea-level training. After the lead-in phase, athletes underwent a period of supervised training at sea level that was designed to provide a longitudinal sea-level control. Training was conducted according to an individualized template based on a 4-wk mesocycle intended to provide increasing volume and intensity over the first 3 wk, with a slight taper during the last week. The first week involved exclusively base running on area trails with a volume equivalent to ~80% of their usual base volume. Laboratory testing also occurred during this week and included one 5,000-m time trial. This testing session served as a baseline for the response to sea-level training. In the second week, volume was increased by 20–25% by increasing both the duration and number of base training workouts. Training intensity was also increased by adding one interval session consisting of five to six 1,000-m intervals (110% of race pace) and one 5,000-m road race. During the third week, base volume was increased by an additional 20%, and intensity was increased by adding a hill running/pliometric training session in addition to the 1,000-m intervals. Finally, during the fourth week, base training volume was reduced by 25–30%, and no interval training sessions were performed. Repeat laboratory testing also occurred during this last week at sea level and included a 5,000-m time trial. This testing session served as the comparison for the response to sea-level training and provided the primary baseline for the altitude and sea-level control training camps. All training sessions were directly supervised by either the investigators or staff and carefully monitored as described below.

Altitude training camp. After the last time trial at sea level, athletes were then matched for gender, 5,000-m time trial performance, and training history into groups of three and then randomized (balanced randomization) to *1*) "high-low" [living at moderate altitude (2,500 m) and training at low altitude (1,200–1,400 m); n = 13; 9 men, 4 women; primary experimental group]; *2*) "high-high" [living at moderate altitude (2,500 m) and training at moderate altitude (2,500–2,700 m); n = 13; 9 men, 4 women; typical altitude-training control group]; or *3*) "low-low" [living at sea level (150 m) and training at sea level (150 m); n = 13; 9 men, 4 women; sea-level control group]. Moderate-altitude living occurred in Deer Valley, Utah, with training on trails and roads in the Wasatch and Uinta mountain ranges. Low-altitude training occurred nearby, an ~30-min drive, in Salt Lake City, Utah. The sea-level training camp took place at the US Olympic Training Center in Chula Vista (San Diego), California, in the foothills of the San Ysidro Mountains, which closely matched the terrain and weather conditions of the altitude camps but were at sea level. The training program during the field camp matched the training program at sea level in Dallas, on the basis of the same 4-wk mesocycle. The first week was an easy acclimatization week. The subsequent 2 wk involved increasing volume and intensity, as described above, with a slight taper during the last week before the return to Dallas.

Sea-level testing period. The first week after return from the field training camp was a testing week and included two 5,000-m time trials on the third and seventh days after return from altitude. Plasma and blood volume and submaximal exercise performance were measured on the second day, the incremental test of $\dot{V}O_{2max}$ was on the fourth day, and the anaerobic capacity test was on the fifth day after return from altitude. This testing session was compared with the last testing session before the training camps, with all testing

performed in the same order, and served as the primary experimental comparison. Over the subsequent 2 wk, the athletes performed primarily easy base running, supplemented by short, fast runs, with a 5,000-m time trial at the end of each week. The purpose of this phase was to determine the optimal time for competition after return from the altitude training camp or control.

Evaluation of Performance

The primary outcome measure of this study was running performance, as measured both on a track and in the laboratory on a treadmill. An outline of the testing schedule is included in Fig. 1.

Track evaluation. 5,000-M TIME TRIAL. Multiple time trials over 5,000 m were conducted on a 400-m track. Time trials were performed at sea level in Dallas at 7:00–8:00 AM (temperature 22–26°C, humidity 80–100%, wind 0–10 km/h). To avoid racing strategies, all starts were staggered by at least 2 min.

Treadmill evaluation. $\dot{V}O_{2MAX}$. $\dot{V}O_{2max}$ was measured with a modified Astrand-Saltin protocol (3) involving incremental exercise on a treadmill. After a brief warm-up, subjects ran at 9.0 miles/h (mph) for men and 8.0 mph for women at 0% grade for 2 min. The grade was then increased 2% every 2 min until exhaustion, which usually occurred after 6–8 min. Oxygen uptake ($\dot{V}O_2$) was measured by using the Douglas bag method, gas fractions were analyzed by mass spectrometer (Marquette MGA 1100), and ventilatory volume was measured with either a Tissot spirometer or dry-gas meter (Collins). $\dot{V}O_{2max}$ was defined as the $\dot{V}O_2$ measured from at least a 40-s Douglas bag. In nearly all cases, a plateau in $\dot{V}O_2$ was observed with increasing work rate, confirming the identification of $\dot{V}O_{2max}$. However, to verify that $\dot{V}O_{2max}$ was achieved, on a separate day a supramaximal treadmill run was performed, with the measurement of $\dot{V}O_2$ and anaerobic capacity as described below. The highest value of $\dot{V}O_2$ achieved on either test was accepted as $\dot{V}O_{2max}$. In addition, heart rate was monitored continuously (Polar CIC, Port Washington, NY), and fingertip capillary blood was obtained during the second minute of each stage for the measurement of lactate concentration [Yellow Springs Instruments (YSI) 23L, Yellow Springs, OH].

MAXIMAL STEADY STATE (MSS). MSS was estimated from the ventilatory threshold according to standard criteria (2) as follows. During the incremental test of $\dot{V}O_{2max}$, breath-by-breath $\dot{V}O_2$ was calculated and displayed online by using gas fractions measured at the mouth by mass spectrometer (Marquette), and ventilation ($\dot{V}E$) by turbine flowmeter (VMM, Interface Associates). The $\dot{V}O_2$ at ventilatory threshold for all tests was determined by a single, blinded, experienced observer during simultaneous examination of multiple plots of $\dot{V}O_2$ vs. $\dot{V}E$, $\dot{V}O_2$ vs. $\dot{V}E/\dot{V}O_2$, $\dot{V}O_2$ vs. CO_2 production ($\dot{V}CO_2$), and $\dot{V}O_2$ vs. $\dot{V}E/\dot{V}CO_2$ by using either commercial (First Breath, Marquette) or proprietary software.

ANAEROBIC CAPACITY. Anaerobic capacity was estimated from the accumulated oxygen deficit, according to the method of Medbo et al. (28). Briefly, subjects had their uphill running economy measured during two, 5-min submaximal runs up an 8% grade (5 and 6 mph for women, 6 and 7 mph for men), during which $\dot{V}O_2$ was measured with the Douglas bag method. After at least 4 h of rest, subjects performed a supramaximal run at 8% grade with the speed chosen individually to exhaust the subject between 2 and 4 min. The accumulated oxygen deficit was defined as the difference between the predicted $\dot{V}O_2$ (calculated from submaximal economy and time) and the measured $\dot{V}O_2$ (28). In addition, the total treadmill time was used as a high-intensity short-duration (3-min) performance marker. Immediately after the supramaximal run, fingertip capillary blood samples were collected every 2 min for 10 min during recovery to identify peak lactate concentration.

SUBMAXIMAL RUNNING ECONOMY AND PERFORMANCE. Submaximal economy during flat treadmill running was estimated from the relationship between $\dot{V}O_2$ and treadmill speed during three, 5-min submaximal runs at 0% grade: 8, 10, and 12 mph for men; 8, 9, and 10 mph for women. $\dot{V}O_2$ at each level was measured from a 1-min Douglas bag obtained from the third to fourth minutes. Running economy was defined as the slope of the regression relating $\dot{V}O_2$ to treadmill speed. Velocity at $\dot{V}O_{2max}$ was also calculated by identifying the treadmill speed that would elicit $\dot{V}O_{2max}$, on the basis of the flat running economy regression equation and $\dot{V}O_{2max}$. $\dot{V}E$, heart rate, and capillary lactate were measured during each stage. Cardiac output was also measured during these runs by using a foreign gas-rebreathing method, with acetylene as the soluble and helium as the insoluble gas (42). Recent modifications in our laboratories designed to facilitate measurement during high-velocity treadmill running have been validated against standard invasive methods, with an r^2 of 0.91 and an SE of the estimate of 1.1 l/m over a range of cardiac output from 2.75 to 27.0 l/min compared with both direct Fick and thermodilution (32). Stroke volume was calculated from cardiac output and heart rate, and arteriovenous oxygen difference [(a-v)D_{O_2}] was calculated from $\dot{V}O_2$ and cardiac output. For purposes of statistical comparison, physiological variables at 10 mph and 0% grade were used as index markers of submaximal running performance.

Other Laboratory Measures

Blood compartments. Plasma volume, blood volume, and red cell mass volume were measured at each testing time point at sea level. Plasma volume was measured by using the Evans blue dye indicator dilution technique (30). Briefly, after at least 30 min of quiet, supine rest, a known quantity of Evans blue dye was injected through a peripheral intravenous catheter, and venous blood was drawn at 10, 20, and 30 min after injection for the measurement of absorbance at 620 and 740 nm via spectrophotometry (DU 600, Beckman). Hematocrit was measured via microcapillary centrifuge, and blood volume was estimated by dividing plasma volume by 1 − hematocrit, using appropriate corrections for trapped plasma and peripheral sampling (30). Red cell mass volume was defined as blood volume − plasma volume.

Evaluation of Training

Training logs. Each runner kept a detailed training logbook that included duration and intensity of each workout, along with resting and training heart rate (Polar). Logs also included descriptions of well-being, fluid intake, body weight, and quantity and quality of sleep and were reviewed weekly by investigators and staff. Diet was also monitored to ensure adequate nutrition.

Training stimulus. To derive an index that would allow us to quantify the training stimulus (that stimulus induced by a training session that results in an adaptative response) and compare training among the three groups, we used the method of Bannister and Wenger (5) for the calculation of the training impulse (TRIMPS). This method multiplies the duration of a training session by the average heart rate achieved during that session, weighted for exercise intensity (5). Total training time and an estimate of training distance were calculated from the information in the training logs.

Training characterization. To precisely quantify the metabolic requirements of a typical training session, we measured running velocity, $\dot{V}O_2$, $\dot{V}E$, heart rate, and lactate during

typical base and interval training under all the conditions at which training occurred during the study: sea level (Dallas and San Diego, both 150 m), low altitude (Salt Lake City, 1,250 m), and moderate altitude (Deer Valley, 2,700 m) after 2 wk of acclimatization. $\dot{V}O_2$ in the field was measured with a small telemetry device (K2, Cosmed) that combines a turbine flowmeter built into a face mask to measure $\dot{V}E$, with a polarographic electrode to measure expired oxygen fraction, assuming a respiratory quotient of 1.0 (which may underestimate $\dot{V}O_2$ at very high work rates) (25). Testing sessions were conducted over measured trails, allowing the calculation of running velocity. Heart rate was measured simultaneously (Polar), and samples of fingertip capillary blood were obtained immediately after each characterization session for the measurement of lactate concentration (YSI).

Statistics

Analytic approach. The primary statistical comparison was between the testing sessions before and after the altitude training camp or sea-level control and was analyzed with a two-way, repeated-measures analysis of variance by using commercially available software (Winstar, Anderson Bell). An interaction statistic F-value < 0.05 was considered statistically significant and was then followed by Student-Newman-Keul's post hoc test for multiple comparisons to determine the source of the difference. The relationship between the change in $\dot{V}O_{2max}$ and the change in red cell mass volume, as well as the change in $\dot{V}O_{2max}$ and the change in 5,000-m time before and after the training camp, were compared by using linear regression and Pearson's coefficient. All data are expressed as means ± SD.

RESULTS

Subjects

Subject characteristics for all three groups are shown in Table 1, which includes the 39 subjects who completed all testing and training phases of the study. Only two subjects dropped out during the course of the study. One subject left because of homesickness. One subject suffered from chronic Epstein-Barr virus infection and was unable to complete the training at altitude. No data from these two subjects are included in the analysis. At baseline and after the sea-level control training period, there were no statistically or physiologically significant differences among the three groups in terms of 5,000-m time, $\dot{V}O_{2max}$, or blood compartment volumes.

Training

There was no significant difference in sea-level training by any criteria among the groups (Fig. 2, *A*-*C*). During the field training camps, all three groups had small but similar increases in total training duration and total training distance, from sea level in Dallas to the field training camp, with no significant difference among the groups. This increase was predominantly because the first and last weeks of training in Dallas included the laboratory testing. Similar to training at sea level in Dallas, there was no significant difference

Table 1. *Subject characteristics and performance indexes*

	Plasma Volume,‡ ml/kg			Blood Volume,† ml/kg			Red Cell Mass,† ml/kg			Hemoglobin,† mg/dl		
	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High
Baseline	53.1 ± 1.8	54.0 ± 2.7	52.0 ± 1.6	82.7 ± 2.2	80.3 ± 3.5	79.9 ± 2.2	29.6 ± .83	26.2 ± .98	27.9 ± .91	13.6 ± .27	13.3 ± .22	13.8 ± .23
Sea-level training	51.5 ± 1.8	56.2 ± 2.1	54.4 ± 2.0	79.4* ± 2.5	84.3 ± 3.0	83.2 ± 3.0	28.0 ± .96	28.1 ± 1.0	28.7 ± 1.1	14.0 ± .24	13.5 ± .24	13.8 ± .22
Altitude training	51.0 ± 1.8	51.1§ ± 2.0	53.3 ± 1.6	78.7 ± 2.5	80.7 ± 3.3	85.0 ± 2.3	27.8 ± 1.1	29.6* ± 1.4	31.7* ± .96	14.1 ± .30	14.8* ± .23	15.0* ± .20
	5,000-m Time,† min			$\dot{V}O_{2max}$,† ml·kg^{-1}·min^{-1}			HR_{max}, beats/min			$\dot{V}E_{max}$, l/min		
	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High
Baseline	17.53 ± .48	17.64 ± .39	17.44 ± .46	64.4 ± 1.8	62.4 ± 1.4	64.2 ± 1.5	193.9 ± 2.0	191 ± 1.9	189.8 ± 1.9	153.2 ± 7.6	143.1 ± 4.8	148.3 ± 5.5
Sea-level training	17.23 ± .46	17.23 ± .43	17.04* ± .42	64.7 ± 1.8	63.8 ± 1.4	64.8 ± 1.0	193.6 ± 1.9	189 ± 1.7	188.5 ± 2.0	146.3 ± 7.9	143.3 ± 6.1	150.0 ± 5.2
Altitude training	17.67 ± .62	17.00* ± .53	17.10 ± .43	63.7 ± 1.8	66.3* ± 1.8	67.0* ± 1.5	193.6 ± 1.7	191.6 ± 1.9	189.1 ± 2.3	149.5 ± 10	146.0 ± 6.0	150.6 ± 7.5
	Max Lactate, mM			$\dot{V}O_2$ at Maximal Steady State,† ml·kg^{-1}·min^{-1}			Velocity at $\dot{V}O_{2max}$,† mph			Anaerobic Capacity, ml/kg		
	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High	Low-Low	High-Low	High-High
Baseline	13.2 ± .98	12.4 ± 1.2	12.8 ± .91	51.5 ± 1.5	51.4 ± 1.5	50.3 ± 1.5	13.16 ± 1.20	13.02 ± 1.27	13.30 ± 1.10	60.5 ± 3.8	54.7 ± 4.0	53.5 ± 4.4
Sea-level training	12.5 ± 1.1	11.9 ± .92	11.9 ± .68	52.7 ± 1.6	53.0 ± 1.4	53.2 ± 1.0	13.24 ± 1.39	13.61 ± 1.30	13.72 ± 1.05	56.1 ± 3.0	52.4 ± 4.2	58.2 ± 4.4
Altitude training	12.7 ± .90	11.9 ± .73	11.7 ± .50	51.9 ± 1.5	56.3* ± 2.7	53.7 ± 1.5	13.07 ± 1.05	13.98* ± 1.78	13.94 ± 1.67	53.4 ± 4.2	48.7 ± 4.7	53.0 ± 5.5

Values are means ± SD; n, no. of subjects. Low-low, group living and training in a mountain environment at sea level (150 m; $n = 13$); high-low, group living at moderate altitude (2,500 m) and training at low altitude (1,250 m; $n = 13$); high-high, group living and training at moderate altitude (2,500 m; $n = 13$); $\dot{V}O_{2max}$, maximal O_2 uptake; HR_{max}, maximal heart rate; $\dot{V}E_{max}$, maximal ventilation; Max, maximal. *$P <$ 0.05 compared with previous baseline (post hoc test). Significantly different compared across groups (interaction statistic) by 2-way analysis of variance (ANOVA): †$P < 0.05$; ‡$P < 0.15$; §$P < 0.10$.

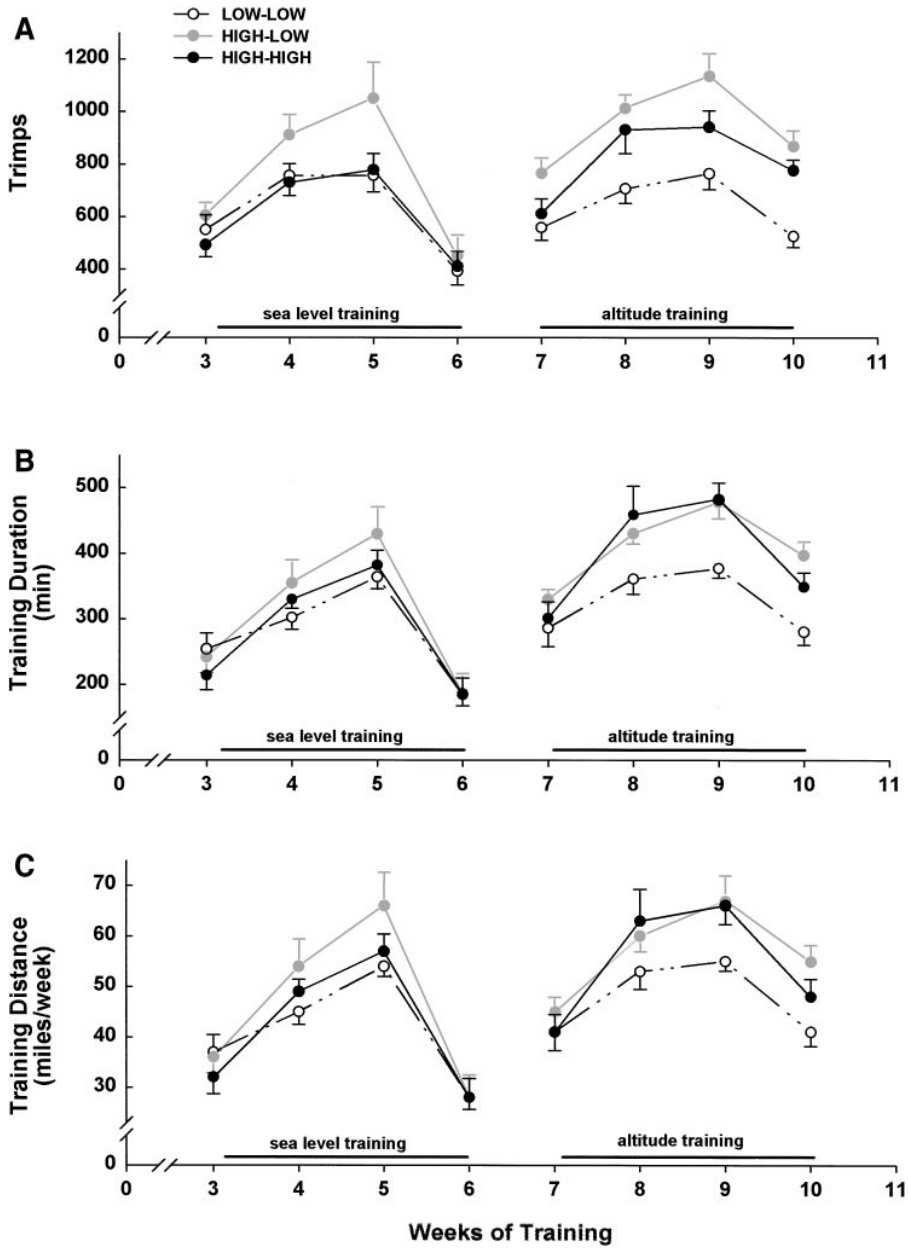

Fig. 2. Quantification of training during 4-wk mesocycles of sea-level training in Dallas and altitude training camp or sea-level control (altitude training) for 3 groups: low-low = sea-level control group in San Diego, CA; high-low = living at 2,500 m and training at 1,250–1,400 m in Salt Lake City, UT; and high-high = living at 2,500 m and training at 2,500–2,700 m in Deer Valley, ID. *A*: "training impulse" (TRIMPS) calculated as described in text on basis of a weighted score incorporating training duration and training heart rate. *B*: training duration (in min) obtained from training logs. *C*: estimated training distance, obtained by multiplying training duration by appropriate speed calculated for typical base and interval training (Table 2).

among groups for training during the training camp for either TRIMPS, training duration, or estimated total mileage, supporting the conclusion that training was closely matched among the groups during both 4-wk mesocycles.

Base training at sea level (Dallas and San Diego) was performed at 82–84% of sea-level 5,000-m race pace, which required 71% of $\dot{V}O_{2max}$, 85% of maximal heart rate, and lactate values of 3.5 mmol/l (Table 2). With increasing altitude, there was a trend for base training to be performed at progressively slower speed and at a lower percentage of sea-level $\dot{V}O_{2max}$, which reached statistical significance at 2,700 m. However, base training heart rate was similar under all three conditions, suggesting that base training was performed at similar relative work rates, even though the absolute work rates were less (slower speeds). For 1,000-m interval sessions, training at sea level (Dallas and San Diego) was accomplished at 110% of sea-level 5,000-m race pace, 87% of sea-level $\dot{V}O_{2max}$, 96% of sea-level maximal heart rate, and lactate values of 10 mmol/l. For unclear reasons, lactate measured after the interval sessions in San Diego was significantly lower than in Dallas. With increasing altitude, running speed, $\dot{V}O_2$, and heart rate were all lower than at sea level. Despite the relative oxygen lack at moderate altitude, peak lactate was significantly lower at 2,700 m than at either sea level or 1,250 m (33).

Response to Training

Blood compartment volumes (Table 1). Plasma volume tended to increase in the high-low and high-high groups by training at sea level in the heat in Dallas (5%, $P = 0.08$) and decreased back to baseline after training in the cooler mountain environments. Plasma

Table 2. *Training characterization*

	Base					Interval, 1,000 m				
	Running speed,† %5-km time	$\dot{V}O_2$,† %SL_{max}	HR,§ beats/min (%SL_{max})	Lactate,† mM	$\dot{V}E$,† l/min BTPS	Running speed,† %5-km time	$\dot{V}O_2$,† %SL_{max}	HR,‡ beats/min (%SL_{max})	Lactate,† mM	$\dot{V}E$,§ l/min BTPS
Sea-level training (150 m; n = 39)										
	83.9 ± 6.2	70.5 ± 8.6	164 ± 10 (85.6 ± 4.6)	3.5 ± 1.3	83.6 ± 17.5	109.3 ± 3.4	87.2 ± 7.9	183 ± 8 (95.7 ± 4.5)	10.1 ± 2.8	125.7 ± 25.1
Field training camp										
Sea level (150 m; $n = 13$)	81.5 ± 5.7	71.9 ± 7.5	163 ± 7 (84.4 ± 4.5)	3.4 ± 1.1	78.2 ± 13.3	110.6 ± 4.5	92.0 ± 6.8	188 ± 5 (97.2 ± 3.0)	8.0 ± 1.5*	128.6 ± 27.0
Low altitude (1,250 m; $n = 13$)	77.3 ± 9.0*	67.2 ± 5.4	164 ± 6 (86.8 ± 3.3)	2.7 ± 0.9*	90.8 ± 15.3	104.0 ± 5.1*	86.1 ± 7.8	181 ± 6 (96.0 ± 2.8)	9.9 ± 1.0	138.5 ± 24.0*
Moderate altitude (2,700 m; $n = 13$)	75.9 ± 4.4*	63.5 ± 4.2*	160 ± 9 (85.0 ± 3.9)	3.8 ± 0.9	104.5 ± 22.3*	95.9 ± 4.6*	73.5 ± 4.7*	174 ± 7* (92.3 ± 3.2)	8.0 ± 2.4	145.0 ± 25.6*

Values are means ± SD; n = no. of subjects. $\dot{V}O_2$, O_2 uptake; SL_{max}, sea level maximum; HR, heart rate; $\dot{V}E$, ventilation. *$P < 0.05$ compared with sea-level training (post hoc test). Significantly different sea-level values compared with field training values across groups (interaction statistic) by 2-way ANOVA: †$P < 0.05$; ‡$P < 0.10$; §$P < 0.15$.

volume was unchanged in the sea-level control group throughout the study. Living at moderate altitude, regardless of training altitude, resulted in a significant increase in red cell mass volume of 9% ($P < 0.01$), which was not observed in the sea-level control. Blood volume changes paralleled the changes in plasma volume during sea-level training in Dallas, when red cell mass volume did not change. In contrast, after subjects lived at moderate altitude, the reduction in plasma volume was offset by an increase in red cell mass volume, leaving total blood volume unchanged but with an increase in oxygen-carrying capacity (increased hemoglobin concentration).

Laboratory treadmill performance. $\dot{V}O_{2MAX}$. After the 2-wk lead-in phase, an additional 4 wk of training at sea level in Dallas did not increase $\dot{V}O_{2max}$ in any group, confirming the fact that the athletes had reached a plateau in aerobic power induced by this training program at sea level (Fig. 3). However, after an additional 4 wk of living at moderate altitude, both high-low and high-high groups increased $\dot{V}O_{2max}$ significantly by an additional 5% ($P < 0.05$ for each). Approximately one-half of the subjects increased $\dot{V}O_{2max}$ by achieving a higher work rate (higher grade) on the incremental treadmill test. The other half was able to increase the proportion of work performed aerobically at the highest work rate and therefore had a higher $\dot{V}O_2$ at the same peak treadmill grade. In contrast, there was no change in $\dot{V}O_{2max}$ in the sea-level control despite an equivalent supervised training program. The change in $\dot{V}O_{2max}$ was loosely but significantly correlated with both the change in red cell mass volume during the training camp ($r = 0.37$, $P = 0.02$) and the change in hemoglobin concentration ($r = 0.40$, $P = 0.01$).

MSS. Similar to $\dot{V}O_{2max}$, $\dot{V}O_2$ at MSS did not change in any group during 4 wk of supervised training at sea level in Dallas (Fig. 4). However, in contrast to $\dot{V}O_{2max}$, MSS increased significantly only in the high-low group after the altitude training camp ($P < 0.05$).

ANAEROBIC CAPACITY. There were no significant changes in accumulated oxygen deficit in any group after training at either sea level or altitude (Table 1). Uphill treadmill run time did not change in any group after training at sea level in Dallas. After the field training camp, uphill treadmill run time increased only in the high-high group (159 ± 10 to 182 ± 13 s, $P < 0.05$).

SUBMAXIMAL ECONOMY AND PERFORMANCE. Treadmill running economy was stable throughout the study and did not change in any group from any training stimulus (Table 3). Similarly stable was the relationship be-

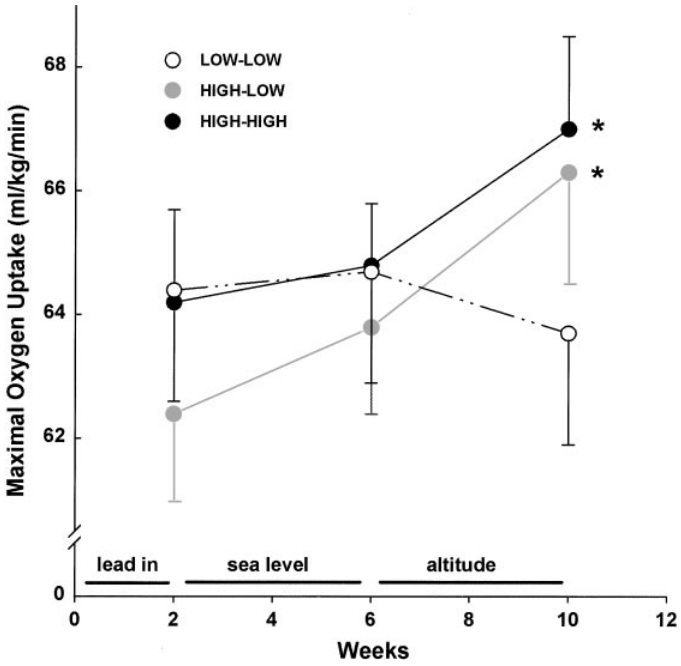

Fig. 3. Maximal oxygen uptake at baseline after sea-level training in Dallas (sea level) and after altitude training camp or sea-level control (altitude). Group characteristics and figure symbols are defined as in Fig. 2. *$P < 0.05$ compared with previous time point.

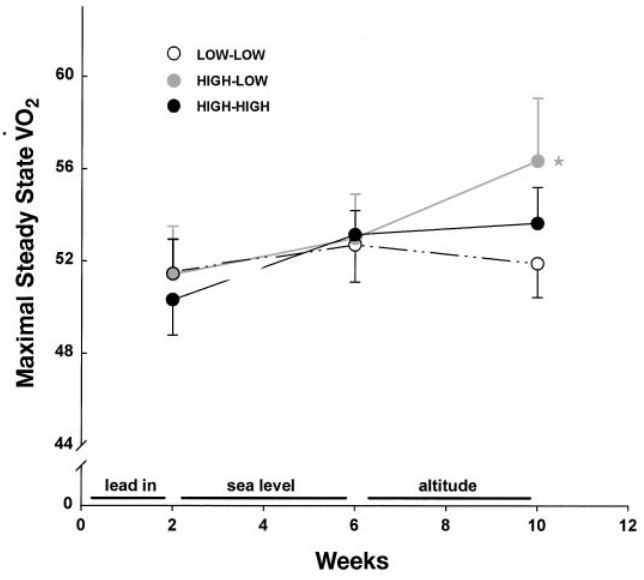

Fig. 4. Oxygen uptake ($\dot{V}O_2$) at maximal steady state, determined from ventilatory threshold, at baseline, after sea-level training in Dallas (sea level), and after altitude training camp or sea-level control (altitude). Group characteristics and figure symbols are defined as in Fig. 2. *$P < 0.05$ compared with previous time point.

tween cardiac output (Table 3) and $\dot{V}O_2$ (slope of cardiac output/$\dot{V}O_2$), which was constant in all groups at all measured time points. However, at velocities near 5,000-m time-trial speeds (12 mph for men), cardiac output tended to be lower in both altitude groups ($P = 0.09$), and (a-$\dot{v}$)DO_2 was significantly higher ($P = 0.01$). Velocity at $\dot{V}O_{2max}$ increased significantly after the altitude training camp only in the high-low group ($P < 0.05$, Table 1). At our index submaximal level of 10 mph and 0% grade, there were initial improvements in heart rate and lactate in all three groups after 4 wk of training at sea level in Dallas but no further changes in these variables after altitude or sea-level field training camps.

5,000-m time trial. In all three groups, 5,000-m time improved to a similar degree (mean 22.3 ± 5 s) by training at sea level in Dallas ($P < 0.05$ only for high-high group). After the training camp, however, 5,000-m time was improved additionally by an average of 13.4 ± 10 s for the high-low group ($P < 0.05$) (Fig. 5*A*). On average, 5,000-m time was 3.3 ± 9 s longer (slower) for the high-high group (P = not significant) and 26.7 ± 13 s longer for the sea-level control (P = not significant) on return to Dallas (2-way analysis of variance interaction statistic, $P < 0.05$, confirming a statistically significant difference in response among the groups). This improvement was even more prominent when men were examined separately (Fig. 5*B*) and persisted for at least 3 wk after return from altitude. Although there was a trend for the high-high group to improve 5,000-m time 2–3 wk after return to sea level, this trend was not statistically significant. Thus, for all groups, there were no statistically significant differences between the 5,000-m times achieved immediately after return from the field training camp and those performed 1, 2, and 3 wk after return. Moreover, the correlations among these times were highly linear and significant, with a Pearson's $r > 0.94$ ($P < 0.00001$) for each comparison. For all subjects, the change in 5,000-m time after the altitude or sea-level training camp was significantly correlated with the change in $\dot{V}O_{2max}$ (Fig. 6) ($r = 0.65$, $P < 0.00001$).

DISCUSSION

The principal new observation from this study is that acclimatization to moderate altitude, when combined with training at low altitude, results in an improvement in sea-level running performance over 5,000 m in already well-trained, competitive runners. Such an improvement was not observed when acclimatization was combined with training at moderate altitude, or with an equivalent supervised training camp at sea level. The mechanism of this improvement appears to be twofold: an altitude-acclimatization effect, increase in blood oxygen-carrying capacity and $\dot{V}O_{2max}$, which was translated into improved performance by low-altitude training, with maintenance of training velocities and oxygen flux, presumably allowing an increase in velocity at $\dot{V}O_{2max}$ and MSS.

High-Altitude Acclimatization Effect

The rationale for this study was based on the assumption that, if altitude training works to improve sea-level endurance performance, then the physiological benefits of altitude training must derive from either the development of acclimatization, an enhancement of the training effect by hypoxic exercise, or both (23). Acclimatization to high altitude includes a number of physiological adaptations that might theoretically improve oxygen transport during exercise. Ventilatory adaptations could improve alveolar oxygenation in some athletes (13) but are likely to be short lived. Structural and biochemical adaptations in skeletal muscle may be more robust and could improve oxygen extraction and substrate utilization (4, 8, 26, 31, 34, 41, 44). All have been reported in animal models and frequently in humans. However, probably the most important adaptation that would improve sea-level performance is an increase in red blood cell mass (43), which increases the oxygen-carrying capacity of the blood and improves aerobic power (9, 16, 20). In the present study, we have demonstrated that 4 wk of living at an altitude of 2,500 m was sufficient to stimulate erythropoietin secretion (37) and increase red blood cell mass volume by ~10%. This increase in oxygen-carrying capacity of the blood is on the order of magnitude observed in previous studies of acute erythrocyte infusion that demonstrated a similar improvement in $\dot{V}O_{2max}$ (9). In this study, the significant, albeit loose, correlation between both the increases in red blood cell mass volume and hemoglobin concentration and the increase in $\dot{V}O_{2max}$ observed in both groups living at moderate high altitude suggests that this endogenous "erythrocyte infusion" is at least partially responsible for the improvement in maximal aerobic power. Moreover, at running speeds on the treadmill that approximated 5,000-m race velocity, the increase in oxygen-carrying capacity allowed a lower

Table 3. *Submaximal exercise responses*

	Cardiac Output, l/min			$\dot{V}O_2$, ml·kg^{-1}·min^{-1}			HR, beats/min			Lactate, mM		
	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)
					8 mph ($n=39$)							
Baseline	21.8	22.2	22.4	40.4	39.5	40.2	153	148	150	3.0	2.4	2.3
	±4.4	±2.4	±4.6	±2.8	±3.0	±3.2	±15	±9	±10	±1.3	±1.6	±1.6
SL training	21.1	21.4	21.8	39.4	38.1	38.4*	149	148	146	2.1	1.6	2.2
	±3.9	±2.1	±2.1	±2.2	±2.2	±2.2	±16	±9	±14	±1.2	±1.6	±2.0
Altitude training	21.1	21.5	20.3	38.4*	37.7	39.1	146	154*	151	2.9	1.4	2.3
	±3.3	±3.0	±4.3	±4.2	±3.6	±2.9	±14	±11	±13	±1.0	±0.9	±1.4
					10 mph ($n=39$)							
Baseline	25.7	23.6	25.6	50.9	49.2	51.1	175	169	170	4.0	3.1	3.5
	±5.6	±2.5	±4.0	±4.8	±3.4	±3.4	±12	±10	±13	±2.4	±1.5	±2.0
SL training	24.5	23.5	24.7	50.8	48.3	48.9	172	165	163*	2.6*	2.0*	2.2*
	±3.7	±1.6	±4.8	±5.7	±2.5	±3.4	±11	±11	±12	±1.7	±1.2	±2.0
Altitude training	24.5	24.5	23.6	50.3	48.3	52.5	174	166	167	3.3	2.3	2.7
	±5.0	±3.2	±3.3	±3.2	±3.5	±8.5	±12	±13	±11	±1.5	±1.4	±1.2
					12 mph [$n=27$ (men only)]							
Baseline	29.1	29.2	31.4	60.3	57.0	59.3	187	184	182	6.6	5.5	6.1
	±7.4	±5.7	±4.0	±2.0	±3.4	±3.0	±7	±10	±7	±1.7	±2.3	±2.0
SL training	29.6	27.7	29.1	61.5	58.4	60.0	185	184	178	5.2*	4.7	4.8*
	±4.1	±4.5	±4.2	±2.7	±3.0	±4.1	±6	±5	±5	±2.1	±1.8	±1.4
Altitude training	29.6	26.0	27.6*	60.7	59.4	60.2	186	182	182	5.7	4.6	5.4
	±6.7	±2.1	±4.3	±3.5	±3.3	±4.4	±6	±11	±4	±2.3	±2.0	±1.9

	$\dot{V}_E$, l/min			Stroke Volume, ml			(a-v)D_{O_2}, ml/dl			Economy, ml·min^{-1}·kg^{-1}· miles·h^{-1}		
	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)	Low-Low ($n=13$)	High-Low ($n=13$)	High-High ($n=13$)
					8 mph ($n=39$)							
Baseline	69	67	69	145	150	150	11.3	11.4	11.3			
	±9	±6	±8	±36	±17	±35	±1.6	±1.5	±1.8			
SL training	66	67	68	143	134	151	11.4	11.4	11.1			
	±8	±6	±9	±37	±43	±16	±1.4	±1.6	±1.2			
Altitude training	71	72*	73*	146	140	136	11.2	11.4	12.7			
	±18	±9	±11	±34	±23	±33	±1.9	±1.9	±3.1			
					10 mph† ($n=39$)							
Baseline	92	88	95	148	140	152	12.9	13.3	12.5			
	±14	±13	±13	±36	±18	±27	±1.7	±1.9	±1.8			
SL training	91	87	91	143	143	153	12.6	13.1	12.5			
	±13	±12	±11	±28	±15	±31	±2.1	±1.3	±1.6			
Altitude training	96	89*	96*	143	149	143	12.9	12.6	14.5*			
	±15	±10	±12	±33	±23	±23	±1.7	±2.0	±3.6			
					12 mph‡ [$n=27$ (men only)]							
Baseline	121	115	125	156	159	173	13.6	12.8	12.4	4.6	4.4	4.5
	±16	±12	±16	±42	±29	±22	±2.4	±2.8	±1.5	±0.2	±0.2	±0.2
SL training	126	113	123	160	151	164	13.2	13.7	13.7	4.6	4.3	4.4
	±18	±12	±18	±24	±24	±22	±1.0	±2.1	±1.9	±0.2	±0.2	±0.3
Altitude training	128*	119	132*	158	143	152	13.5	14.8*	14.7*	4.5	4.4	4.5
	±18	±16	±24	±34	±15	±24	±2.3	±1.4	±2.1	±0.3	±0.3	±0.3

Values are means ± SD; n = no. of subjects. SL, sea level; (a-v)D_{O_2}, arteriovenous O_2 difference. *$P < 0.05$ compared with previous baseline (post hoc test). Significantly different compared across groups (interaction statistic) by 2-way ANOVA: †$P < 0.05$; ‡$P < 0.15$.

cardiac output and therefore more peripheral diffusion time and oxygen extraction [i.e., increased (a-v)D_{O_2}], as well as providing for additional cardiac flow reserve. Finally, the close correlation between the increase in $\dot{V}O_{2max}$ and the improvement in 5,000-m time after the field training camp argues strongly that this is a key adaptation during altitude training and a necessary mechanism for improving sea-level performance.

However, this adaptation, by itself, may be necessary but not sufficient to improve sea-level performance. Thus the high-high group was exposed to exactly the same living conditions at 2,500 m and had similar increases in red cell mass volume and $\dot{V}O_{2max}$ as the high-low group, yet they did not increase running performance over 5,000 m at sea level. The only difference between these two groups was the training site, which in the high-high group was also at moderate altitude.

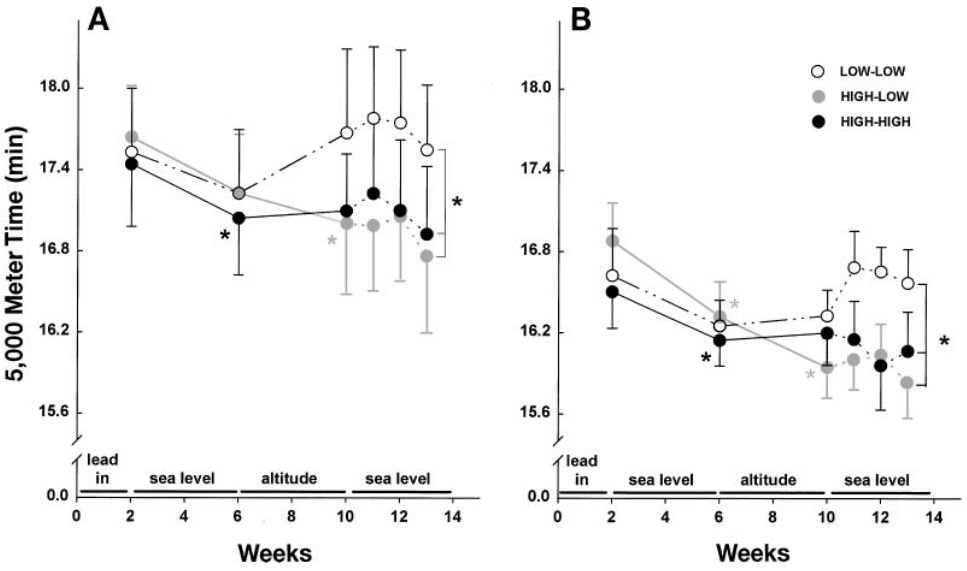

Fig. 5. Time trial (5,000 m) results for all subjects (n = 13/group; 9 men, 4 women, *A*) and also for men only (n = 9/group; *B*) at baseline, after sea-level training in Dallas (sea level), and after altitude training camp or sea-level control (altitude). Time trials (5,000 m) were performed 3, 7, 14, and 21 days after leaving training camp. Group characteristics and figure symbols are defined as in Fig. 2. *$P < 0.05$ compared with previous time point. Asterisks next to brackets, interaction statistics for analysis of variance, $P < 0.05$.

Low-Altitude Training

Training (as opposed to living) at moderate altitude is associated with relatively severe hypoxemia, with oxyhemoglobin saturations reported to be <80% during typical base training (19). This hypoxia results in a decrease in maximal aerobic power of ~1% for every 100 m above 1,500 m (11). Particularly for well-trained athletes, there are more marked reductions in aerobic power even at lower altitudes (41). Thus elite athletes are not able to sustain the high work rates at altitude necessary to maintain competitive fitness (35). In the present study, this limitation was manifested most clearly during interval training that was performed nearly 15% slower, and at 20% lower $\dot{V}O_2$, than comparable training at sea level. Despite an equivalent effort (V_E was 16% greater than at sea level), heart rate and lactate were also significantly lower at 2,700 m, consistent with previous reports of decreased maximal heart rate and maximal lactate after acclimatization to high altitude (33). Such a reduction in interval-training intensity in trained runners has recently been shown to decrease running performance over 5,000 m despite a preservation of $\dot{V}O_{2max}$ (27). It thus appears likely that, in the high-high group, the increase in red blood cell mass and $\dot{V}O_{2max}$ was offset by a reduction in training velocity and oxygen flux, leading to no change in running performance.

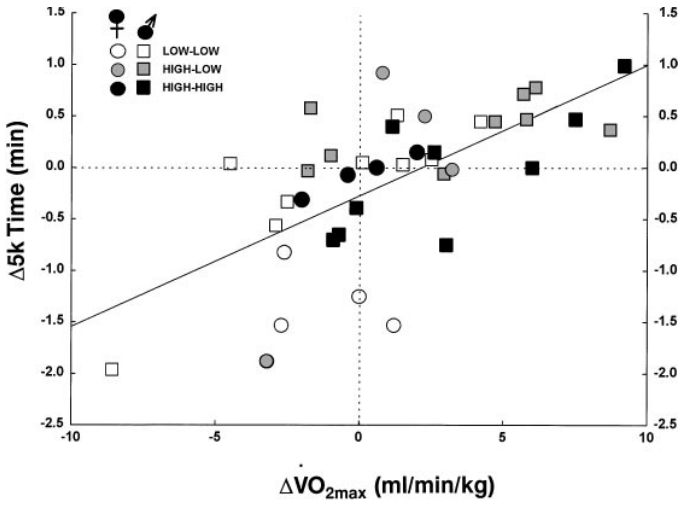

Fig. 6. Relationship between change in maximal oxygen uptake ($\Delta\dot{V}O_{2max}$) and change in 5,000-m time trial performance (Δ5K) after training camp for all subjects. Group characteristics and symbols are defined as in Fig. 2, except for men (squares) and women (circles).

In contrast to the training in the high-high group, who performed all interval training at 2,700 m, similar training at 1,250 m in the high-low group was only slightly (6%) slower than at sea level and was accomplished at virtually the same $\dot{V}O_2$, heart rate, and lactate concentration. Although the mechanism is not entirely clear, such a maintenance of training velocity and oxygen flux is likely to be critical toward sustaining competitive performance, as has been recently shown in runners who decrease training volume but maintain intensity (6). This difference in training between high-high and high-low groups appeared to be the most important factor that, combined with the increase in $\dot{V}O_{2max}$ induced by altitude acclimatization, allowed for both an increase in the $\dot{V}O_2$ at MSS and the velocity at $\dot{V}O_{2max}$ only in the high-low group.

Whether these characteristics associated with "low-altitude training" are specifically responsible for the improvement in 5,000-m time or simply markers for some other skeletal muscle adaptation is not clear. Adaptations such as increases in MSS would be expected to have a greater impact on longer distance events, during which competition occurs at some fraction below $\dot{V}O_{2max}$, than on 5,000-m performance, which is run essentially at $\dot{V}O_{2max}$. One previous study has demonstrated an increase in muscle buffer capacity after a period of "high-high" altitude training (29) associated with an increase in oxygen deficit and an increase in treadmill run time. In the present study, we did observe an increase in uphill treadmill run time in our high-high group, raising the possibility of an in-

crease in buffer capacity. However, we did not observe any changes in anaerobic capacity, as measured by the accumulated oxygen deficit. The difference between the study of Mizuno et al. (29) and ours may derive from the fact that their athletes changed training modalities, from running to skiing, during their sojourn at altitude. They also did not have any controls doing similar ski training at sea level.

We were concerned with the observation that in the present experiment, the concurrent sea-level control, if anything, tended to have a worse 5,000-m performance after the training camp compared with before, although this did not reach statistical significance. We suspect that, because all time trials were conducted in the heat of the Texas summer, a loss of heat acclimatization in the cooler mountains outside of San Diego, without the benefit of altitude acclimatization, may have been responsible for some of this apparent deterioration. We also considered the possibility that the athletes in the San Diego control might not have been as motivated as the altitude groups on the basis of previously held expectations of a benefit from altitude training. However, the new Olympic training center represents the state of the art in training facilities available to American Olympic athletes and would not be available to the athletes in this study under other circumstances. Virtually all the athletes were therefore very excited about the opportunity to go to San Diego, thus eliminating any sense of disappointment that might occur on the basis of randomization to the sea-level control. Moreover, these athletes are by nature very competitive, and the athletes in San Diego were, if anything, more motivated to perform better on return from the camp to prove that their training experience was every bit as good as their altitude counterparts. Over the course of the training cycle, it was clear that these athletes were receiving an outstanding training experience. They bonded as a group, performed extremely well in the interim races to which they were assigned during the month, and uniformly felt that they had improved significantly. The observed and reported differences are therefore even more remarkable in this regard.

On closer inspection, the majority of this seeming decline was due to unusually poor performances in two of our women athletes. Because we had only four female athletes in each group, which might increase the variability, as an additional check we also examined the 5,000-m performance for all men in the study separately. As can be seen in Fig. 5*B*, the results for men only were less variable, with no clear change in performance in the low-low or high-high group, and an even greater improvement in the high-low group. We believe that further studies involving larger numbers of female athletes will be necessary to confirm the applicability of this study to all women. However, we speculate that as long as adequate iron is made available through supplementation, the results will be consistent for all athletes, regardless of gender.

In conclusion, well-trained competitive runners living at moderate altitude increased red cell mass and oxygen-carrying capacity of the blood and increased $\dot{V}O_{2max}$ after return to sea level. This increase in $\dot{V}O_{2max}$ was translated into improved performance by the maintenance of near sea-level training velocities and oxygen flux when interval training was performed at low altitude, resulting in an increase in $\dot{V}O_2$ at MSS and velocity at $\dot{V}O_{2max}$. Running performance over 5,000 m at sea level therefore improved only in the runners who lived at moderate altitude and trained near sea level (high-low group) but not in those who lived and trained at moderate altitude or lived and trained at sea level, after equivalent training programs.

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