“Living high-training low” altitude training improves sea level performance in male and female elite runners

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Stray-Gundersen, James, Robert F. Chapman, and Benjamin D. Levine. “Living high-training low” altitude training improves sea level performance in male and female elite runners. J Appl Physiol 91: 1113–1120, 2001.—Acclimatization to moderate high altitude accompanied by training at low altitude (living high-training low) has been shown to improve sea level endurance performance in accomplished, but not elite, runners. Whether elite athletes, who may be closer to the maximal structural and functional adaptive capacity of the respiratory (i.e., oxygen transport from environment to mitochondria) system, may achieve similar performance gains is unclear. To answer this question, we studied 14 elite men and 8 elite women before and after 27 days of living at 2,500 m while performing high-intensity training at 1,250 m. The altitude sojourn began 1 wk after the USA Track and Field National Championships, when the athletes were close to their season’s fitness peak. Sea level 3,000-m time trial performance was significantly improved by 11.1% (95% confidence limits 0.3–19.9%). One-third of the athletes achieved personal best times for the distance after 27 days of acclimatization to moderate altitude, which produces an increase in maximal oxygen uptake (VO2 max), plus the maintenance of sea level oxygen flux during low-altitude training, which preserves skeletal muscle structure and function and facilitates an improvement in sea level running performance.

However, despite numerous anecdotal reports of the success of altitude training for world class athletes, some recent reports have suggested that HiLo, or any form of altitude training, may not be advantageous for elite compared with collegiate level athletes (1, 2, 14). The concept of symmorphosis, as elaborated by Hoppeler and Weibel (17), argues that, for any system, such as the respiratory chain for oxygen transport, the maximal capacity of each parameter is adjusted quantitatively to match the structural and functional limits of the demands placed on the system as a whole. Thus, for the “elite athletes” of the animal kingdom, each step of the pathway of oxygen from the atmosphere to the mitochondria has evolved toward optimal function and maximal aerobic power, allowing little room for further adaptive improvement. Therefore, for elite human athletes, small, short-term improvements in one step of the oxygen cascade may be met by functional limits in other steps, minimizing the potential performance benefit of altitude training. However, elite human athletes living and training at sea level are unable to develop similar levels of circulating hemoglobin/red cell mass as “high-endurance” animal species who have the ability to autotransfuse by splenic contraction (23, 25). Thus raising circulating hemoglobin levels conceivably has the greatest potential for improving elite endurance performance in humans. In addition, the interaction between convective and diffusive components of
oxygen transport, as described by Wagner (35), would predict an increase in \( V_{O_2} \text{max} \) with increasing circulating hemoglobin and red cell mass. In support of these concepts, transfusion studies (5, 10, 37) and those administering recombinant erythropoietin (3, 4) suggest that an increase in red cell mass by itself will increase the \( V_{O_2} \text{max} \) for all endurance athletes, regardless of performance level.

Thus the present study was designed to investigate the effect of the HiLo paradigm on elite runners who were likely to be much closer to their ultimate performance potential than the athletes previously studied with this approach. The study was timed such that the athletes would be in the best shape of the year [i.e., after they had just completed the spring track season culminating with the National Collegiate Athletic Association (NCAA) championships and the USA Track and Field National Championships]. We hypothesized that the combination of acclimatization to 2,500 m and high-intensity training at 1,250 m would improve sea level performance in elite middle- and long-distance runners.

**METHODS**

**Subjects**

Twenty-six distance runners (17 men and 9 women) were recruited. Athletes were required to be competitive at a national level in an event from the 1,500 m to the marathon. Twenty-four of the 26 athletes were ranked in the US top 50 for their event in 1997. The athletes included two 1996 Olympians, and 50% of the athletes had competed in the 1996 US Olympic Trials. All but four athletes competed in the 1997 NCAA Championships or the 1997 USA Track and Field Championships or both. Three of those four athletes were attempting to run qualifying times up to the day of the meet. Exclusion criteria included altitude residence (>1,000 m) or recent illness or injuries preventing normal training and racing. The subjects gave their written consent to the study, which had received approval from the Institutional Review Board of the University of Texas Southwestern Medical Center.

**Protocol**

The study protocol was a modification of one previously developed by the authors for collegiate runners (24). Briefly, the athletes were assessed at sea level in the week before and the week after 27 days of living at 2,500 m (see Fig. 1). The NCAA Championships were held at sea level 3 wk before the altitude sojourn. The time trials were run in men’s and women’s heats in the early evening (1900 to 2000). Athletes were instructed to achieve the best time possible on each time trial. Experienced pace setters (athletes not otherwise involved in the project) were utilized to set a fast, competitive pace for the first 1,600 m of the 3,000 m race to ensure physiological rather than tactical performance. The pace setter or “rabbit” ran the same preselected race pace in both the prealtitude and postaltitude time trials. Temperatures ranged from 25 to 27°C, relative humidity ranged between 50 and 75%, and there was no wind. Time was recorded for each subject to the nearest 0.1 s.

**Treadmill assessment.** After a 15-min warm-up, the athlete ran to volitional exhaustion performing a protocol with constant velocity and increase in grade of 2% every 2 min. Inspired ventilation was measured by a dual-thermistor flow probe (Trentor 1200, Hector Engineering), and expired gas concentrations were measured in a 5-liter mixing chamber by mass spectrometer (Marquette RMS M-100, Milwaukee, WI). Heart rate was recorded at the end of each minute by telemeter (Polar). Percent arterial oxyhemoglobin saturation was measured by ear oximetry (Hewlett-Packard 47201A). Data were collected and displayed with the use of a data acquisition control system (Workbench for Windows 2.0, Strawberry Tree) sampling at 40 Hz. Values for arterial oxygen saturation, oxygen uptake, and minute ventilation were averaged over each minute of exercise.

**Hematology assessment.** Venous blood was drawn into tubes containing EDTA, with the subject in the supine position, between 0600 and 0700 on four occasions: 3 days before the altitude sojourn, after the first night at altitude (20 h), after 19 days at altitude, and 20 h after return to sea level. Whole blood was assayed in duplicate for hemoglobin concentration (Radiometer OSM-3) and hematocrit (spun capillary tubes). Plasma was then obtained by centrifugation and stored frozen (−80°C) until assayed. Plasma was assayed by RIA with commercial kits and a gamma counter (ISODATA 20–20) for ferritin (DSL, Webster, TX), erythropoietin (DSL), and soluble transferrin receptor concentrations (Orion).
Statistics

Data are presented in the tables as means ± SD. SPSS 6.1 was utilized for statistical calculations. Performance and VO_{2\text{max}} were compared by paired t-test. The hematologic data were compared by one-way ANOVA. Gender differences were tested by using a two-way ANOVA (altitude × gender). Significance was set at P ≤ 0.05. When a significant effect was obtained, post hoc analysis was performed with the Student-Newman-Keuls test to identify differences.

RESULTS

Subjects

Fourteen men (25 ± 3 yr, 179 ± 5 cm, 63.6 ± 5.2 kg) and eight women (24 ± 3 yr, 168 ± 5 cm, 53.3 ± 4.9 kg) successfully completed the protocol for a total of 22 complete subjects. Four subjects (three men and one woman) suffered injury (n = 2) or illness (n = 2) during the sojourn that prevented normal training or racing and were not included in the analysis. There were no gender differences with respect to the response to the altitude sojourn; therefore, data for men and women are considered together.

Performance

Group 3,000-m performance at sea level was significantly improved after the HiLo treatment (Table 1 and Fig. 2). Men and women improved to similar extents, reducing time trial time by 5.8 s (95% confidence limits 1.8–9.8 s) or 1.1% (95% confidence limits 0.3–1.9%). Three athletes improved their sea level 3,000-m time by as much as 23 s, whereas one athlete ran 18 s slower.

Maximal Exercise

VO_{2\text{max}} was significantly increased by 3% after the altitude camp (see Fig. 3). Maximal ventilation was also significantly increased after the altitude camp (Table 2). There was a significant relationship between the change in VO_{2\text{max}} and the change in maximal minute ventilation (r = 0.67, P = 0.0006). Moreover, there was a less robust but still statistically significant relationship between the change in VO_{2\text{max}} and the change in 3,000-m running time (r = −0.48, P = 0.02). Maximal heart rate was unchanged. Arterial oxygen saturation was reduced to 89 ± 4% during maximal exercise but was unaffected by the altitude camp. Time to exhaustion on the treadmill was not significantly different.

Hematology Assessments

Hemoglobin concentration increased on acute ascent to altitude, remained elevated during the camp, and was significantly elevated on return to sea level (see Table 3). Hematocrit was significantly elevated when measured on the 19th day at altitude and remained significantly elevated on return to sea level. Plasma ferritin concentrations were not significantly altered from the initial value. However, despite oral iron supplementation, there was a trend (P = 0.07) for subsequent values to be lower than the initial value. Plasma erythropoietin concentration doubled after one night at altitude.

Table 1. Sea level performance

<table>
<thead>
<tr>
<th></th>
<th>Pre-HiLo</th>
<th>Post-HiLo</th>
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<tr>
<td></td>
<td>3,000 m time, min:s</td>
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<tr>
<td>Group (n = 22)</td>
<td>8:45.4 ± 0.39</td>
<td>8:39.6 ± 0.39*</td>
</tr>
<tr>
<td>Women (n = 8)</td>
<td>9:32.4 ± 0.111</td>
<td>9:26.9 ± 0.113*</td>
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<tr>
<td>Men (n = 14)</td>
<td>8:18.4 ± 0.140</td>
<td>8:12.6 ± 0.108†</td>
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Values are means ± SD. HiLo, living high-training low. *P ≤ 0.05; †P ≤ 0.10 pre vs. post.

Table 2. VO_{2\text{max}}

<table>
<thead>
<tr>
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<th>Pre-HiLo</th>
<th>Post-HiLo</th>
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<tr>
<td>Time to exhaustion, min</td>
<td>8.8 ± 1.1</td>
<td>9.0 ± 1.0</td>
</tr>
<tr>
<td>VE, l/min</td>
<td>152 ± 31</td>
<td>163 ± 34*</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>192 ± 7</td>
<td>191 ± 8</td>
</tr>
<tr>
<td>%Arterial saturation</td>
<td>89 ± 4</td>
<td>89 ± 4</td>
</tr>
<tr>
<td>VO_{2\text{max}}, ml·kg^{-1}·min^{-1}</td>
<td>72.1 ± 6.9</td>
<td>74.4 ± 6.8*</td>
</tr>
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</table>

Values are means ± SD; n = 22 (18 men, 8 women). VO_{2\text{max}}, maximal oxygen uptake; VE, minute ventilation; HR, heart rate. *P ≤ 0.05 pre- vs. post-HiLo.
2,500 m and was not different from baseline after 19 days at the camp. Then plasma erythropoietin levels decreased significantly on return to sea level. Soluble transferrin receptor concentrations were significantly elevated (25%) after 19 days at altitude, consistent with active erythropoiesis (3, 5), and returned to baseline on return to sea level.

**DISCUSSION**

The major finding of this study is that, in this group of elite runners, sea level 3,000-m running performance improved significantly in response to a 27-day camp utilizing the HiLo paradigm. In fact, nine athletes recorded personal records at the distance after the HiLo camp, despite having prepared for and competed in national championship events just before the sojourn. The mechanism of the improvement appears to be similar to that previously described in carefully controlled studies of collegiate-level athletes (24) with a stimulation of erythropoiesis leading to an apparent increase in oxygen delivery to peripheral tissues as evidenced by 1) a near doubling of plasma erythropoietin concentration and a 43% reduction in serum ferritin concentration despite oral iron supplementation on acute exposure to altitude, 2) a rise in soluble transferrin receptor concentration with chronic exposure to altitude, and 3) an increase in hemoglobin concentration and hematocrit on return to sea level with a decrease in plasma erythropoietin concentration below original sea level baseline.

**Limitations**

We must acknowledge that our study suffers from a major limitation shared by most research conducted in elite athletes: namely, the absence of a concurrent control group performing a similar training camp at sea level. Such a control group would be optimal to ensure that the athletes did not improve merely from the result of a training camp per se, rather than living high-training low. A number of lines of evidence, however, suggest that the study design employed in this experiment was sufficient to account for most of this effect. First, in our previous studies (24), which included many years of piloting work, we determined that for collegiate athletes at least 2 wk of controlled training were necessary to overcome the “training camp effect.” For example, in one preliminary study of a sea level training camp, six male runners increased their VO$_2$$_{max}$ from 68 ± 1.5 to 70 ± 1.4 ml·kg$^{-1}$·min$^{-1}$ after 2 wk of supervised training, but did not increase further after an additional 2 wk of training (70 ± 1.8 ml·kg$^{-1}$·min$^{-1}$) (Levine and Stray-Gundersen, unpublished observations). Thus, for all the 52 male and female athletes studied in our previously published reports (24, 34), after a 2-wk “lead-in” phase of supervised training there was no significant increase in VO$_2$$_{max}$ with an additional 4 wk of structured training at sea level (64 ± 0.8 to 64 ± 0.8 ml·kg$^{-1}$·min$^{-1}$). Moreover, after these 6 wk of sea level training by collegiate athletes in these studies, there was no further improvement obtained even by an outstanding training camp environment at sea level for an additional 4 wk (24). In the present study, we considered that the months of preparation by these elite athletes for their national championships were at least equivalent to the 2 wk of training applied to collegiate athletes a number of weeks after their competitive season for the purpose of minimizing the training camp effect. Additionally, on review of each athlete’s training program leading up to the championships, all had peaked on this research by 10.220.32.246 on November 10, 2017 http://jap.physiology.org/ Downloaded from

<table>
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<th>Table 3. Hematologic assessments</th>
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<tr>
<td>--------------------------------</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
</tr>
<tr>
<td>Hematocrit, %</td>
</tr>
<tr>
<td>Ferritin, μg/ml</td>
</tr>
<tr>
<td>Erythropoietin, ng/ml</td>
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<td>Soluble transferrin receptor, μg/ml</td>
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Values are means ± SD. *P < 0.05 different from initial sea level value (Student-Newman-Keuls post hoc tests); † significant F statistic (P = 0.05) (1-way ANOVA).
anemia. All had normal hemoglobin concentration and hematocrit, and all had normal red cell size and distribution. 2) Despite oral iron supplementation, the iron requirements of altitude exposure were such that bone marrow iron stores as measured by serum ferritin did not increase over the course of the training camp. In fact, ferritin decreased with each longitudinal measurement (Table 3), suggesting that bone marrow iron stores were more rather than less depleted at the end of the altitude camp. 3) The baseline erythropoietin concentrations were normal and low, arguing against a physiologically significant anemia; anemia is the most potent stimulus to synthesis of erythropoietin (20). Thus the available evidence would argue against simple treatment of iron deficiency as the mechanism of the increase in hemoglobin and hematocrit in this study.

Finally, because of time constraints associated with performing the study in elite athletes during peak competition periods, the subjects were not as completely characterized, nor were the details of the training program as rigorously controlled to the same extent as in previous work (24). However, the same basic training template was used for the protocol, and key parameters were measured in both populations, including erythropoietin and hemoglobin concentrations, \( V_{O2\ max} \), and time trial performance, that allowed comparison to our previous studies.

When the results from carefully controlled, comprehensively assessed studies on collegiate runners are compared with the results of the present study (see Table 4), the results are remarkably similar in both direction and magnitude of the effect. In addition, when similar parameters were measured, the results suggest that the same mechanism produced the effect, i.e., an increase in erythropoietin leading to an increase in hemoglobin concentration, increased \( V_{O2\ max} \), and increased performance. Therefore, we believe that the compromises made in this study to evaluate elite athletes during a time of peak fitness did not compromise the validity of the results.

The Unique Model of Elite Athletes

Elite athletes of the animal kingdom provide a unique model of the concept of symmorphism, whereby the structural design of all components comprising a system is matched quantitatively to functional demand (17). For example, foxes, dogs, and horses have systems matched quantitatively to functional demand in the structural design of all components comprising a system.

Table 4. Comparison of elite and collegiate athletes for changes in selected hematologic and performance variables

<table>
<thead>
<tr>
<th></th>
<th>( \Delta \text{Epo}, % )</th>
<th>( \Delta \text{Hb}, \text{g/dl} )</th>
<th>( \Delta V_{O2\ max}, \text{ml/kg} \cdot \text{min}^{-1} )</th>
<th>( \Delta \text{Performance}, % )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elite runners</td>
<td>103 ± 742</td>
<td>1.0 ± 1.1</td>
<td>2.3 ± 2.6</td>
<td>1.1</td>
</tr>
<tr>
<td>College runners</td>
<td>59 ± 402</td>
<td>1.1 ± 0.7</td>
<td>2.5 ± 3.6</td>
<td>1.4</td>
</tr>
</tbody>
</table>

Values are means ± SD. \( \Delta \), change in; Epo, erythropoietin; M, men; F, women. Performance refers to 3,000-m racing time (elite runners) or 5,000-m racing time (college runners).
climatization to sea level. Further study will be necessary to confirm or exclude this hypothesis.

A more detailed analysis of human vs. nonhuman athletes suggests, in fact, that the most likely avenue for elite human athletes to improve oxygen transport would be to raise their red cell mass and circulating hemoglobin concentration. In human athletes, red cell mass is the one component of the oxygen cascade that does not increase to the level observed in "elite athletes" of the animal kingdom. Humans do not clearly autotransfuse by splenic contraction at the onset of exercise like horses (25) and dogs (23). This effect raises exercise hematocrit well into the 50s in those species. Thus, when the oxygen-carrying capacity of the blood is increased in elite athletes, either by acute red blood cell infusion (5, 10, 37) or by chronic administration of recombinant human erythropoietin (3, 4), \( \text{VO}_{2\text{max}} \) increases. The results shown in the present study are in the same direction as and half the magnitude of the results obtained by either an acute (transfusion) or chronic increase in red cell mass (exogenous rhEPO administration). At least one uncontrolled study has suggested altitude-induced improvements in \( \text{VO}_{2\text{max}} \) in undeniably elite athletes (7). One of these subjects (JR) went on to set a world record after living at altitude and training intermittently at sea level.

Some investigators, failing to observe an increase in hemoglobin/myoglobin mass after brief periods of time in normobaric hypoxic environments (8–10 h/night for 10 days to 3 wk), have questioned the erythropoietic effect of moderate altitude exposure (1, 2). However, the evidence in favor of such an altitude-mediated erythropoiesis is quite compelling. Cross-sectional studies in the Peruvian Andes (19, 28, 32) as well as in the Colorado Rockies (36) have demonstrated clearly that there is an elevated red cell mass in natives of high altitude that is proportional to the oxyhemoglobin saturation (19, 36).

Moreover, when sea level natives ascend acutely to altitude, there is an increase in iron turnover by more than twofold that begins within the first few hours of exposure and peaks by \( \sim 2–3 \) wk (12, 18, 28). Direct examination of the bone marrow during acute high-altitude exposure has documented a dramatic increase in nucleated red blood cells, virtually doubling by 7 days, indicative of accelerated erythropoiesis (18, 28). Although most of these data are from altitudes higher than 2,500 m studied in the present experiment, our elite endurance athletes spent significant time exercising at low-moderate altitudes, which causes further arterial desaturation (8), suggesting that athletes may have a greater stimulation of erythropoiesis for the same altitude than a more sedentary population.

As in the present study with elite athletes, previous studies have also shown that both iron turnover (18, 28) as well as erythropoietin concentrations (6, 15, 20, 24, 30) return to sea level values relatively rapidly during chronic altitude exposure. Nevertheless, the red cell mass continues to increase for up to 8 mo of chronic altitude exposure, at least at altitudes above 4,000 m (28), suggesting that this level of stimulated erythro-

![Fig. 4. Comparison of athletes of various performance levels and the change in performance from a 4-wk living high-training low altitude sojourn. Race time represents the initial time trial presented as a percent of the American record (AR) in the event at the time. The change in performance is the percent change from the precamp time trial to the postcamp time trial.](http://www.jap.org)
of a prominent and sustained increase in erythropoietin at altitude leading to an increase in red cell mass (6). Although the genetic mechanisms responsible for determining the erythropoietic response to hypoxia in humans have not been entirely worked out, animal models suggest that this response may be transcriptionally regulated (26). Moreover, at least some individuals have genetic polymorphisms in the erythropoietin gene (33) or the erythropoietin receptor (27) that may profoundly influence the erythropoietic response to hypoxia (22). It is possible, therefore, particularly in studies with relatively small sample sizes, that the presence of significant numbers of nonresponders could bias the study outcome in favor of no detectable response.

**Practical Implications for Performance of Elite Athletes**

In previous work examining collegiate athletes (6, 24), we identified a 1.4% improvement in 5,000-m performance, a 2.5 ± 2.4 ml·kg⁻¹·min⁻¹ improvement in V˙O₂max, and a 1.1 ± 0.7 g/dl increase in hemoglobin concentration after 4 wk at 2,500 m. In this study, we obtained a 1.1% improvement in 3,000 m performance, a 2.3 ± 2.6 ml·kg⁻¹·min⁻¹ improvement in V˙O₂max, and a 1.0 ± 1.1 g/dl in hemoglobin concentration in elite runners. When all the athletes who have completed a HiLo or HiHiLo camp in our studies are examined together, there is no influence of performance ability on the response to such altitude training (Fig. 4).

Figure 4 also demonstrates that, although substantial individual variability remains within all athletes, the variation is smaller for the elite subjects. Thus the coefficient of variation for the collegiate athletes was 3.3%, whereas that for the elite athletes was just over half as great, or 1.9%. Because of this reduced variability, the percent improvement, expressed as a fraction of the variation within the elite population, is 0.58, well within the criteria of 0.5–0.7 recently recommended for individual variation in response to altitude training. Although a 1.1% improvement in performance may not seem like a large effect, at an elite level in sports, races are won or lost by small fractions of a percent. Thus the benefit of a HiLo or HiHiLo altitude training camp has the potential to substantially improve race outcome for individual elite athletes.

In conclusion, despite having prepared for and competed in national championship events, elite runners improved sea level running performance by 1.1% (95% confidence limits 0.3–1.9%) after 27 days of living at moderate altitude (2,500 m) and performing high-intensity training at low altitude (1,250 m). Data collected indicate that the magnitude and mechanism of the effect are similar to those obtained in collegiate runners undergoing the same experimental paradigm. The mechanism involves expansion of the red cell mass and an increase in circulating hemoglobin levels, accompanied by maintenance of oxygen flux to working muscle. Thus the HiLo training approach is effective in improving sea level running performance ranging from 50 to 90% of the world record in events lasting from ~7 to 20 min. We believe that this paradigm can be used to enhance sea level performances that are dependent on high levels of oxygen transport.

This project involved the coordinated support and effort of many people and several organizations. We thank the athletes who participated in the project. We also thank all the people who helped to bring about this experiment, including the speakers, the Department of Health Science, Indiana University, the track coaching staff at Indiana University, and the staff and volunteers of USA Track and Field and the US Olympic Committee. Particularly, thanks go to Drs. Harmon Brown, David Martin, Jay T. Kearney, and Martha Ludwig for tireless support and perseverance. A special note of gratitude and appreciation goes to Greg Harger for all of his work on this project.

**REFERENCES**