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Improved running economy in elite runners after 20 days of simulated moderate-altitude exposure

P. U. Saunders, R. D. Telford, D. B. Pyne, R. B. Cunningham, C. J. Gore, A. G. Hahn, and J. A. Hawley. Improved running economy in elite runners after 20 days of simulated moderate-altitude exposure. J Appl Physiol 96: 931–937, 2004. First published November 7, 2003; 10.1152/japplphysiol.00725.2003.—To investigate the effect of altitude exposure on running economy (RE), 22 elite distance runners [maximal O$_2$ consumption (V$\dot{O}_2$)$_{max}$] 72.8 ± 4.4 ml·kg$^{-1}$·min$^{-1}$; training volume 128 ± 27 km·wk$^{-1}$], who were homogenous for maximal V$\dot{O}_2$ and training, were assigned to one of three groups: live high (simulated altitude of 2,000–3,100 m)-train low (LHTL; natural altitude of 600 m), live moderate-train moderate (LMTM; natural altitude of 1,500–2,000 m), or live low-train low (LLTL; natural altitude of 600 m) for a period of 20 days. RE was assessed during three submaximal treadmill runs at 14, 16, and 18 km/h before and at the completion of each intervention. V$\dot{O}_2$, minute ventilation (V$\dot{E}$), respiratory exchange ratio, heart rate, and blood lactate concentration were determined during the final 60 s of each run, whereas hemoglobin mass (Hb$\text{mass}$) was measured on a separate occasion. All testing was performed under normoxic conditions at ~600 m. V$\dot{O}_2$ (l/min) averaged across the three submaximal running speeds was 3.3% lower ($P = 0.005$) after LHTL compared with either LMTM or LLTL. V$\dot{E}$, respiratory exchange ratio, heart rate, and Hb$\text{mass}$ were not significantly different after the three interventions. There was no evidence of an increase in lactate concentration after the LHTL intervention, suggesting that the lower aerobic cost of running was not attributable to an increased anaerobic energy contribution. Furthermore, the improved RE could not be explained by a decrease in V$\dot{E}$ or by preferential use of carbohydrate as a metabolic substrate, nor was it related to any change in Hb$\text{mass}$. We conclude that 20 days of LHTL at simulated altitude improved the RE of elite distance runners.

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Subjects were assigned to three different inter-
within-subject repeated-measures design with a nested temporal de-

The Australian Institute of Sport (Belcon-
nen, Australia) used the Douglas bag principle (16) to collect all expirate into one of two 150-liter aluminized bags. While one bag is being filled, the other has the expired volume and gas fractions determined. Standard algorithms were employed to compute minute values of \( V_{\text{E}} \), expired \( O_2 \), \( CO_2 \), and RER from the sum of two consecutive 30-s samples. The \( O_2 \) and \( CO_2 \) gas analyzers (AEI Technologies, Pittsburgh, PA) were calibrated before each test with three precision gas mixtures, with an acceptable calibration being within \( \pm 0.03\% \) of all target values. Volume was measured with a precision-calibrated linear displacement piston coupled to real-time measurement of temperature and pressure inside the piston. The typical error of measurement (24) or standard deviation of the differences divided by square root of two for \( V_{\text{E}} \) in our laboratory is 2.4% for the pooled data for running at 14, 16, and 18 km/h. Typical error of measurement was established from duplicate trials conducted on 11 subjects before the start of the main study.

\( Hb_{\text{mass}} \). Before and after the three experimental periods, one-half of each group of runners underwent measurement of total \( Hb_{\text{mass}} \) by using a CO-rebreathing technique modified from Burge and Skinner (7). The alterations used two doses of 99.5% CO, which were rebreathed for 10 min each (20-ml initial dose and a second dose of 1.25 ml CO/kg body mass), and percent \( HbCO \) (\%HbCO) was measured on capillary instead of venous blood (1). An average of \%HbCO of four capillary blood samples determined on an ABL700 Series blood-gas analyzer (Radiometer Medical, Copenhagen, Denmark) for both CO doses was obtained, and the change in \%HbCO (difference between first and second measures) was used to calculate \( Hb_{\text{mass}} \) (7). Typical error of measurement for \( Hb_{\text{mass}} \) in our laboratory is 2.7% (1) when capillary blood samples are used.

Table 2. \textit{HHTL simulated altitude protocol}

<table>
<thead>
<tr>
<th>Week</th>
<th>Mon</th>
<th>Tue</th>
<th>Wed</th>
<th>Thu</th>
<th>Fri</th>
<th>Sat</th>
<th>Sun</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>600</td>
<td>2000</td>
<td>2000</td>
<td>2200</td>
<td>2500</td>
<td>600</td>
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<td>2500</td>
<td>2500</td>
<td>2500</td>
<td>2700</td>
<td>600</td>
<td>600</td>
</tr>
<tr>
<td>3</td>
<td>2500</td>
<td>2700</td>
<td>2800</td>
<td>2800</td>
<td>3000</td>
<td>600</td>
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<tr>
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<td>2700</td>
<td>2900</td>
<td>3000</td>
<td>3000</td>
<td>3100</td>
<td>600</td>
<td>600</td>
</tr>
</tbody>
</table>

Values are in meters.
RUNNING ECONOMY AND ALTITUDE EXPOSURE

Fig. 1. Absolute oxygen consumption ($\dot{V}O_2$; in l/min) before (pre) and after (post) 20 days of altitude exposure, averaged across 3 running speeds (14, 16, and 18 km/h). Values are individual responses as well as mean responses for each intervention; mean values are represented with a thicker line and different shade as individuals; $n = 10$ (LHTL; A), $n = 10$ (LMTM; B), and $n = 13$ (LLTL; C).

DISCUSSION

The major finding from the present study was that 20 nights of sleeping at 2,000- to 3,100-m simulated altitude while training at 600-m altitude (LHTL) reduced whole body $\dot{V}O_2$ (i.e., improved RE) in elite distance runners compared with a control group who lived and trained near sea level (LLTL). To the best of our knowledge, this is the first investigation to find improvements in RE (i.e., reductions in submaximal O$_2$ cost of running) in elite athletes after short-term exposure to simulated moderate altitude. Of interest was the observation that RE was improved over a range of running speeds (14, 16, and 18 km/h) and was independent of changes in Hbmass, V$_E$, RER, and HR.

A second important finding of the present study was that living at 1,500 m and training at an altitude of ~2,000 m was insufficient stimulus to alter variables associated with RE. Our results are not in accordance with the conventional view that submaximal $\dot{V}O_2$ remains unchanged at sea level after returning from a period of altitude exposure, with multiple...
studies observing no change in submaximal $\dot{V}O_2$ at sea level (21, 26, 29, 39, 44), nor even changes in submaximal $\dot{V}O_2$ under chronic hypoxic conditions up to 7,440-m terrestrial altitude (31). However, Katayama et al. (25) have previously reported that simulated-altitude exposure (3-wk exposure comprising 3 sessions/wk for 90 min/session of intermittent hypobaria of 4,500 m) improved RE in highly trained runners. Indeed, our findings are consistent with a growing number of studies that have shown that various forms of altitude exposure can reduce submaximal $\dot{V}O_2$ (19, 22, 23, 25, 27). These exposures include mountain climbing over 3 wk to 6,194 m (22, 27), 23-night exposure to 3,000-m simulated altitude (19), 3-wk exposure to intermittent hypobaria of 4,500 m (25), and long-term residence at 4,200 m (23). A rigorous experimental design as well as good precision in our indirect calorimetry system give us confidence in our data.

Mechanisms that have been suggested to improve economy after altitude exposure include a decreased cost of $\dot{V}E$ (22). In the present study, $\dot{V}E$ and HR did not markedly change after 20 days of LHTL simulated-altitude exposure, suggesting that the increase in RE was not strongly associated with these parameters. A potential mechanism that could conceivably improve economy is greater carbohydrate utilization for oxidative phosphorylation after a period of altitude acclimatization. It has previously been observed that 4,300-m altitude acclimatization for 21 days decreased the reliance on fat as a fuel at both rest and during low-intensity (50% $\dot{V}O_2$ max) cycling (33). It has been suggested that a shift toward increased dependence on glucose metabolism and away from reliance on fatty acid consumption under conditions of acute and chronic hypoxia is advantageous because glucose is a more efficient fuel in terms of generating ATP per mole of $O_2$ (5, 19, 22). In the present study, there was no evidence to support a shift toward greater carbohydrate utilization during submaximal exercise, because RER was not different between the three groups. Another potential mechanism underlying the improved economy is a reduced energy requirement of one or more processes involved in excitation and contraction of the working muscles as a result of metabolic adaptations associated with altitude acclimatization (22). However, such a hypothesis was not tested in the present investigation.

There was no significant difference in [Lac] after LHTL altitude exposure, which suggests that the improved RE demonstrated in the present study was not a result of an increased anaerobic energy contribution. One of the potential mechanisms for lower plasma [Lac] accumulation is an increase in skeletal muscle oxidative enzyme capacity (17), with a resultant shift in metabolism away from anaerobic toward aerobic. Weston et al. (42) reported that Kenyan runners who live and train at altitude have higher oxidative enzyme activities than their $\dot{V}O_2$ max-matched Caucasian runners and that this is associated with a better RE. On the other hand, Van Hall and associates (41) demonstrated that reduced peak [Lac] may be a

Table 3. Comparison of $\dot{V}O_2$ vs. speed slopes pre- and post-LHTL intervention with estimated adjustments to standing $\dot{V}O_2$

<table>
<thead>
<tr>
<th>LHTL (a)</th>
<th>LHTL (b)</th>
<th>LHTL (c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
</tr>
<tr>
<td>Slope</td>
<td>0.205</td>
<td>0.204</td>
</tr>
<tr>
<td>y Intercept</td>
<td>0.273</td>
<td>0.158</td>
</tr>
<tr>
<td>$r$</td>
<td>0.998</td>
<td>0.999</td>
</tr>
<tr>
<td>$r^2$</td>
<td>0.996</td>
<td>0.997</td>
</tr>
</tbody>
</table>

Comparison of slopes

| t Value | 0.01 | 0.54 | 2.95 |
| P value | 0.99 | 0.62 | 0.04 |

LHTL (a), postintervention standing $O_2$ consumption ($\dot{V}O_2$) adjusted to prevalence minus 0.12 l/min (absolute change that occurred during running at 14, 16, and 18 km/h); LHTL (b), postintervention standing $\dot{V}O_2$ adjusted to prevalence minus 3.4% (percent change that occurred during running at 14, 16, and 18 km/h); LHTL (c), all postvalues reduced by 17% to obtain significant difference between pre- and posttest slopes.
transient phenomenon, with lower levels merely reflecting a disturbance to muscle acid-base balance.

The slope between $V\dot{O}_2$ and running speed and power output has been used as a means of detecting changes in economy (22). The failure to observe any significant differences in the slopes for either of the hypoxia groups implies that the enhancement in RE is not directly attributable to improved locomotor muscle metabolism or that more invasive procedures are required to detect this change. At muscle level, a reduced ATP cost of contraction should coincide with a change in slope of the $V\dot{O}_2$ vs. speed relationship. The same is true for a better ATP yield per mole of $O_2$ used. If the improved RE is not occurring at a muscular level, it may simply reflect a decreased resting metabolism by an unknown mechanism.

RE did not change after the LMTM intervention at natural moderate altitude (1,500–2,000 m). One plausible explanation for this finding is that the altitude employed in the LMTM intervention was insufficient to stimulate the mechanism(s) responsible for inducing whole body improvements in economy. Previous research demonstrating improved economy as a result of altitude exposure (19, 22, 23, 25, 27) has utilized markedly higher elevations (3,000–6,200 m). In the present study, the LHTL intervention, which resulted in improved RE, was conducted at an elevation between 2,000 and 3,100 m.

Although further work is clearly required to elucidate the dose-response relationship in terms of altitude and duration of exposure, the results of the present study suggest that a “threshold altitude” to alter economy might exist and may be between 2,000 and 3,100 m.

In agreement with previous investigations from our laboratory (2–4) that demonstrated no change in Hb mass or erythrocyte production after LHTL, we did not detect any significant changes in Hb mass after any intervention. This is in contrast to the results of studies conducted by others (8, 26). It is possible that 20 days of 8–12 h/day altitude exposure to 2,000–3,200 m is insufficient time to elicit marked increases in red cell mass. However, we have also been unable to detect an increase after a 31-day training camp at 2,690 m (18). It may well be that longer periods at these altitudes are required to elicit changes in Hb mass. Indeed, evidence suggests that cyclists living permanently at 2,600 m have a higher Hb mass than their sea-level counterparts (35). In the present study, which investigates the effects of altitude exposure on RE, it is apparent that Hb mass has no relationship with the improved RE demonstrated.

Despite our finding of no changes in Hb mass, there are other potential benefits arising from short-term altitude exposure. Improved RE is a critical part of improving running performance, the ultimate goal of athletes using altitude and hypoxia. Although we did not determine performance per se in the present study, the relationship between RE and performance is well documented, with many independent reports demonstrating a strong relationship between RE and distance-running performance (9–12, 14, 30). Of note, but by no means conclusive evidence, is that all of the subjects (apart from 2 who did not race during the duration of the study) ran personal or season best times over distances ranging from 1,500 to 10,000 m within 1 mo of the LHTL intervention. In comparison, only 3 of the 13 LLTL subjects ran personal or season best times within 1 mo of the control intervention.

In conclusion, the results of the present study demonstrate that sleeping at a simulated altitude of 2,000–3,100 m using the LHTL model for 20 days resulted in a 3.3% improvement in RE of elite distance runners, whereas living and training at moderate altitude (1,500–2,000 m) and living and training near sea level (600 m) for the same duration had no effect on RE in elite distance runners. The underlying mechanisms for the reduction in submaximal $O_2$ cost after LHTL are difficult to elucidate but were not related to $V\dot{E}$, HR, RER, or Hb mass. The lack of change in Hb mass strongly suggests that the mecha-

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Table 4. Cardiorespiratory and physiological measures

<table>
<thead>
<tr>
<th>LHTL</th>
<th>LMTM</th>
<th>LLTL</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>67.8±8.6</td>
<td>67.0±8.3</td>
<td>66.1±5.9</td>
</tr>
<tr>
<td>RER</td>
<td>0.92±0.04</td>
<td>0.94±0.03</td>
<td>0.92±0.04</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>157±8</td>
<td>156±13</td>
<td>156±10</td>
</tr>
<tr>
<td>Hb mass, g</td>
<td>997±125</td>
<td>1,013±137</td>
<td>992±101</td>
</tr>
<tr>
<td>$V_{\dot{E}}$, l/min</td>
<td>95.7±15.6</td>
<td>97.7±17.0</td>
<td>95±10.5</td>
</tr>
</tbody>
</table>

Values are means ± SE, pre- and posttest after 20 days of altitude exposure, for the pooled data of 3 running speeds (14, 16, and 18 km/h); n = 10 (LHTL), n = 10 (LMTM), n = 13 (LLTL) except hemoglobin mass (Hb mass) in which n = 6 (LHTL), n = 6 (LMTM), n = 5 (LLTL). LSD, least significant difference, which is 2 times the standard error of the differences; any pair of means differing by more than the LSD are considered significant ($P < 0.05$) changes between pre- and posttests among the 3 treatment groups. RER, respiratory exchange ratio; HR, heart rate; $V_{\dot{E}}$, minute ventilation.

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Fig. 4. Log of blood lactate concentration before (pre) and after (post) 20 days of altitude exposure, averaged across 3 running speeds (14, 16, and 18 km/h). Values are means ± SE; n = 10 (LHTL), n = 10 (LMTM), and n = 13 (LLTL). LSD is 2 times the standard error of the differences; any pair of means differing by more than the LSD are considered significant ($P < 0.05$) changes between pre- and posttests among the 3 treatment groups.
nism(s) underlying the enhanced RE is independent of accelerated erythropoiesis. Finally, our results suggest that 20 days of LHTL are sufficient time to acquire benefits from altitude acclimatization, although the elevation must be >2,000 m to provide sufficient stimulus to improve RE.

ACKNOWLEDGMENTS

The authors acknowledge J. Woodside, N. Townsend, G. Allbon, S. Wright, and A. Cox for technical assistance during data collection; E. Lawton, R. Shugg, A. Martin, and M. Steinbronnn of the Department of Physiology, Australian Institute of Sport, for support in operating the altitude house; and BOC Gases Australia for technical support.

GRANTS

This study was funded by the Australian Sports Commission.

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