

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

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Saunders, P. U., 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/jappphysiol.00725.2003.—To investigate the effect of altitude exposure on running economy (RE), 22 elite distance runners [maximal O₂ consumption ($\dot{V}O_2$) 72.8 ± 4.4 ml·kg⁻¹·min⁻¹; training volume 128 ± 27 km/wk], who were homogenous for maximal $\dot{V}O_2$ 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. $\dot{V}O_2$, minute ventilation (\dot{V}_E), respiratory exchange ratio, heart rate, and blood lactate concentration were determined during the final 60 s of each run, whereas hemoglobin mass (Hb_{mass}) was measured on a separate occasion. All testing was performed under normoxic conditions at ~600 m. $\dot{V}O_2$ (l/min) averaged across the three submaximal running speeds was 3.3% lower ($P = 0.005$) after LHTL compared with either LMTM or LLTL. \dot{V}_E , respiratory exchange ratio, heart rate, and Hb_{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 \dot{V}_E or by preferential use of carbohydrate as a metabolic substrate, nor was it related to any change in Hb_{mass}. We conclude that 20 days of LHTL at simulated altitude improved the RE of elite distance runners.

oxygen consumption; intermittent hypoxia

THE EFFECT OF ALTITUDE TRAINING ON endurance performance has been researched extensively (2–4, 8, 15, 18, 19, 22, 25–29, 33–36, 39, 43). There is a widespread belief in the athletic community that altitude training can enhance sea-level athletic performance (15, 26, 43), with at least three independent studies demonstrating that altitude training increases both sea-level maximal O₂ consumption ($\dot{V}O_{2\max}$) and running performance (8, 26, 36). The mechanisms for these improvements are not clear but have been proposed to include hematological changes (i.e., increased red cell mass) (8, 26) and local muscular adaptations (such as improved skeletal muscle buffer capacity) (19). The traditional approach to altitude training was for athletes to live and train at moderate natural altitude. A

recent approach has been for athletes to live and sleep at altitude and train near sea level, i.e., the so-called live high-train low (LHTL) method (26). Because the geography of many countries does not readily permit LHTL, a further refinement has been for athletes to live at simulated altitude under normobaric conditions and train at, or close to, sea level (34).

Altitude acclimatization results in both central and peripheral adaptations, which improve O₂ delivery and utilization (6, 28, 38, 45); decreased O₂ utilization for a given speed would improve an athlete's running economy (RE). Potential mechanisms for improved RE are the amount of ATP per mole O₂ used increases, the amount of ATP necessary for running at a given speed decreases, or a combination of both of these mechanisms (22). To date, little research has been undertaken on the effects of altitude exposure on RE in highly trained distance runners. Two investigations (26, 39) have reported no change in submaximal O₂ consumption ($\dot{V}O_2$) after a period of altitude exposure, whereas another (25) found improved RE after intermittent hypoxic altitude exposure. Similarly, after a period of altitude acclimatization, sea-level $\dot{V}O_2$ during submaximal cycling is either reduced (19, 22, 23, 27) or unchanged (5, 33). Therefore, it appears that, in both running and cycling, altitude exposure certainly has no detrimental effects on economy and, in some athletes, may lead to improvements. To the best of our knowledge, only one study has demonstrated an improved RE in well-trained runners as a result of altitude training (25). Improving RE is advantageous to distance-running performance because it reduces the utilization of O₂ at any given steady-state running speed (9, 11–13, 40). Previous studies demonstrating improved economy as a result of altitude exposure have used cycling or two-legged kicking as the mode of exercise, and subjects were mountain climbers and triathletes (19, 22, 23, 27). Furthermore, these investigations used natural or simulated altitudes ranging from 3,000 m (19) to 6,194 m (22, 27), which are generally higher than altitudes typically used by athletes. Therefore, the present investigation was undertaken to determine the effects of altitude exposure on RE in elite distance runners. We hypothesized that RE would be improved after both natural moderate [live moderate-train moderate (LMTM)] and simulated altitude (LHTL).

METHODS

Subjects. Twenty-two elite male middle- and long-distance runners volunteered to participate in this study. Subjects all competed at a

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Table 1. *Subject characteristics*

Parameter	LHTL	LMTM	LLTL	Combined
Age, yr	25.3 ± 2.6	24.3 ± 3.4	25.1 ± 2.7	24.9 ± 2.9
Body mass, kg	67.4 ± 8.4	66.4 ± 5.9	65.7 ± 7.6	66.5 ± 7.3
$\dot{V}O_{2\max}$ l/min	4.90 ± 0.52	4.73 ± 0.42	4.75 ± 0.40	4.79 ± 0.45
$\dot{V}O_{2\max}$, ml·kg ⁻¹ ·min ⁻¹	73.0 ± 2.8	71.7 ± 3.8	73.5 ± 5.7	72.8 ± 4.4
Training volume, km/wk	126 ± 26	132 ± 30	126 ± 26	128 ± 27
Training duration h/wk	9.7 ± 1.7	10.2 ± 2.0	10.1 ± 1.4	10.0 ± 1.7
Training intensity (1–5 scale)	2.5 ± 0.2	2.5 ± 0.2	2.5 ± 0.4	2.5 ± 0.3

Values are means ± SD; $n = 10$ [live high-train low (LHTL)], $n = 10$ [live and train at moderate altitude (LMTM)], and $n = 13$ [live and train at natural low (600 m) altitude (LLTL)]. $\dot{V}O_{2\max}$, maximal O_2 consumption.

national ($n = 12$) or international ($n = 10$) level (Table 1). They were informed of the experimental procedures and possible risks involved with participation before written consent was obtained. The Australian Institute of Sport Ethics Committee approved all testing procedures.

Overview of experimental protocol. The study was a between- and within-subject repeated-measures design with a nested temporal design within subjects. Subjects were assigned to three different intervention groups, with each group homogenous in regard to $\dot{V}O_{2\max}$ and training (Table 1). Ten subjects were assigned to the LHTL group, consisting of 4 wk at simulated altitude that ranged from 2,000 to 3,100 m (Table 2). During this period, subjects spent 5 nights/wk (9–12 h/night) in a normobaric hypoxic chamber with N_2 enrichment and 2 nights/wk at the ambient altitude (~600 m) during the 4-wk period. Ten subjects were assigned to the LMTM group. The LMTM intervention was performed at Falls Creek, Victoria, Australia (1,570 m) for a period of 20 days, with training altitudes that ranged from 1,500 to 2,000 m. The LMTM intervention took place 6 wk after the completion of the LHTL intervention, and subjects in this group were also part of the LHTL ($n = 6$) or control groups ($n = 1$). The control group [live low-train low (LLTL)] consisted of 13 runners who lived and trained near sea level (600 m) for a period of 20 days. Subjects in the control group were tested within the 12-wk period that the other interventions occurred, with subjects in this group also being part of the LHTL ($n = 1$) and LMTM ($n = 1$). The fact that some subjects were included in more than one intervention group was factored into the subsequent statistical analysis. A washout period of 6 wk was used for subjects in multiple groups. Before and on completion of the three interventions, all subjects performed testing under normoxic conditions in Canberra, ACT, Australia (600 m). RE was measured at three submaximal speeds (see *Treadmill testing*), with $\dot{V}O_{2\max}$ measured during the baseline test. Hemoglobin mass (Hb_{mass}) was determined by carbon monoxide (CO) rebreathing on a separate occasion to the submaximal RE test.

Treadmill testing. RE was determined by measuring submaximal $\dot{V}O_2$ for 4 min at constant running speeds of 14, 16, and 18 km/h on a custom-built motorized treadmill (Australian Institute of Sport, Belconnen, Australia). $\dot{V}O_2$, minute ventilation ($\dot{V}E$), respiratory exchange ratio (RER), heart rate (HR), and blood lactate concentration ([Lac]) were measured during the RE tests. RE was defined as the total $\dot{V}O_2$ collected during the last 60 s of each 4-min running stage. The slope of $\dot{V}O_2$ vs. running speed was also determined as a measure of RE. $\dot{V}O_{2\max}$ was measured during an incremental test to volitional exhaustion, which was performed 2 min after the third submaximal effort. Subjects completed an incremental protocol that commenced at a treadmill speed of 18 km/h and was increased 1 km/h each minute up to a speed of 20 km/h. After 1 min at 20 km/h (0% gradient), the treadmill gradient increased by 1% each minute until volitional exhaustion was reached. HR was measured by short-range telemetry

(Polar Vantage NV, Kempele, Finland), and on immediate completion of the test a capillary blood sample was drawn for measurement of [Lac].

Gas analysis. Respiratory gases were analyzed on a custom-designed and -built open-circuit indirect calorimetry system with associated in-house software (Australian Institute of Sport, Belconnen, Australia). This automated system uses 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 $\dot{V}E$, expired CO_2 , $\dot{V}O_2$, 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 ±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 $\dot{V}O_2$ 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_{mass} . Before and after the three experimental periods, one-half of each group of runners underwent measurement of total Hb_{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_{mass} (7). Typical error of measurement for Hb_{mass} in our laboratory is 2.7% (1) when capillary blood samples are used.

Statistical analysis. Because some individuals participated in multiple groups, the design was not fully balanced. Consequently, a general linear mixed-model analysis (χ^2 test) was undertaken. Mean profiles along with the standard errors are shown graphically, and the amount of variability between groups is given by the least significant difference. Any pair of means differing by more than the least significant difference are considered significant ($P < 0.05$) changes between pre- and posttests among the three treatment groups. Means are pooled values of the three running speeds, because differences were independent of speed, indicated by no group × test × speed interaction. The statistical package Genstat (2003) sixth edition (VSN International, Oxford, UK) was employed for statistical computation. Slopes and intercepts of $\dot{V}O_2$ vs. running speed were compared on group mean data by using Prism software (2002) version 3.03 (GraphPad Software, San Diego, CA). The regression data for the three groups were fitted through the measured $\dot{V}O_2$ at 14, 16, and 18 km/h, as well as an assumed value of 0.304 l/min (LHTL only) for standing (0 km/h) $\dot{V}O_2$ on the basis of existing data (20, 32, 37).

RESULTS

RE. $\dot{V}O_2$ was similar across the three groups during the pretest with means ± SD of 3.53 ± 0.51 (LHTL), 3.51 ± 0.34

Table 2. *LHTL simulated altitude protocol*

Week	Mon	Tue	Wed	Thu	Fri	Sat	Sun
1	600	2,000	2,000	2,200	2,500	600	600
2	2,500	2,500	2,500	2,500	2,700	600	600
3	2,500	2,700	2,800	2,800	3,000	600	600
4	2,700	2,900	3,000	3,000	3,100	600	600

Values are in meters.

(LMTM), and 3.47 ± 0.36 (LLTL) l/min, respectively, for the pooled data of the three running speeds. However, 20 days of LHTL simulated-altitude exposure decreased (3.3%, $P = 0.005$) $\dot{V}O_2$ to 3.41 ± 0.53 l/min, whereas the LMTM and LLTL groups remained unchanged with posttest means \pm SD of 3.50 ± 0.32 and 3.49 ± 0.37 l/min, respectively (Fig. 1). The reduction in $\dot{V}O_2$ during the LHTL intervention was present at all three running speeds (Fig. 2). There were no significant differences between the slopes of $\dot{V}O_2$ vs. running speed pre- and postintervention for the three groups. However, the LHTL slope was offset after the 20-day altitude exposure (Fig. 3). When the LHTL slopes were compared after adjusting for the estimated standing $\dot{V}O_2$ by the same absolute (0.12 l/min) and percent (3.4%) change observed during the three running speeds, there was still no significant difference in the slopes between pre- and posttests (Table 3). We calculated that a 17% reduction in running and standing $\dot{V}O_2$ was required to make the pre- and postintervention slopes significantly different.

Cardiorespiratory and physiological measures. When the three groups were compared between pre- and posttests across the three running speeds, $\dot{V}E$ ($P = 0.89$), HR ($P = 0.31$), RER ($P = 0.48$), and Hb_{mass} ($P = 0.22$) remained unchanged (Table 4). The log of [Lac] was taken because initial analysis showed this measure was not normally distributed. The difference between pre- and posttests for log of [Lac] for the three groups (pooled data of the 3 running speeds) was not significant ($P = 0.12$). However, the LHTL group showed a trend toward decreased (24%, $P = 0.12$) [Lac] after the altitude exposure with the pretest value of 2.5 ± 1.1 mM decreasing to 2.1 ± 1.2 mM. [Lac] in the other groups was unchanged, with pooled values of 2.1 ± 1.2 to 2.0 ± 1.0 mM (LMTM) and 3.0 ± 1.3 to 2.8 ± 1.3 mM (LLTL) between pre- and posttests, respectively (Fig. 4).

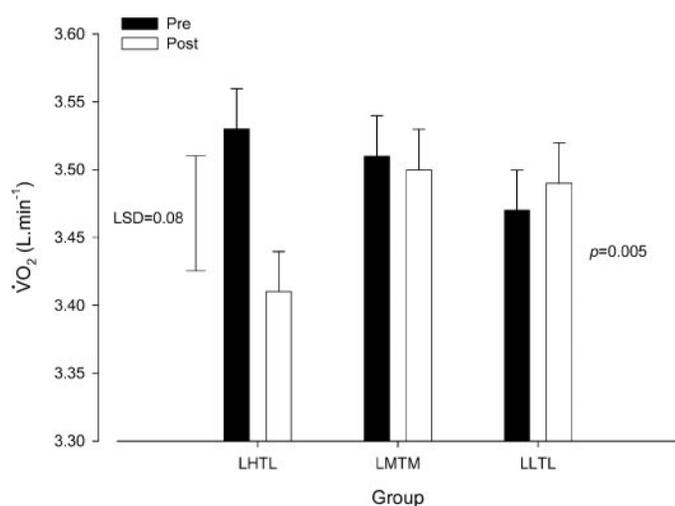


Fig. 1. Absolute oxygen consumption ($\dot{V}O_2$; in l/min) before (pre) and after (post) 20 days of altitude exposure, averaged across 3 running speeds (14, 16, and 18 km/h). Values are means \pm SE; $n = 10$ [live high-train low at simulated high altitude (LHTL)], $n = 10$ [live and train at natural moderate altitude (LMTM)], and $n = 13$ [live and train at natural low altitude (LLTL; 600 m)]. Changes between pre- and posttests are significantly different ($P < 0.05$) among the 3 treatment groups, signified by the least significant difference (LSD), which is 2 times the standard error of the differences.

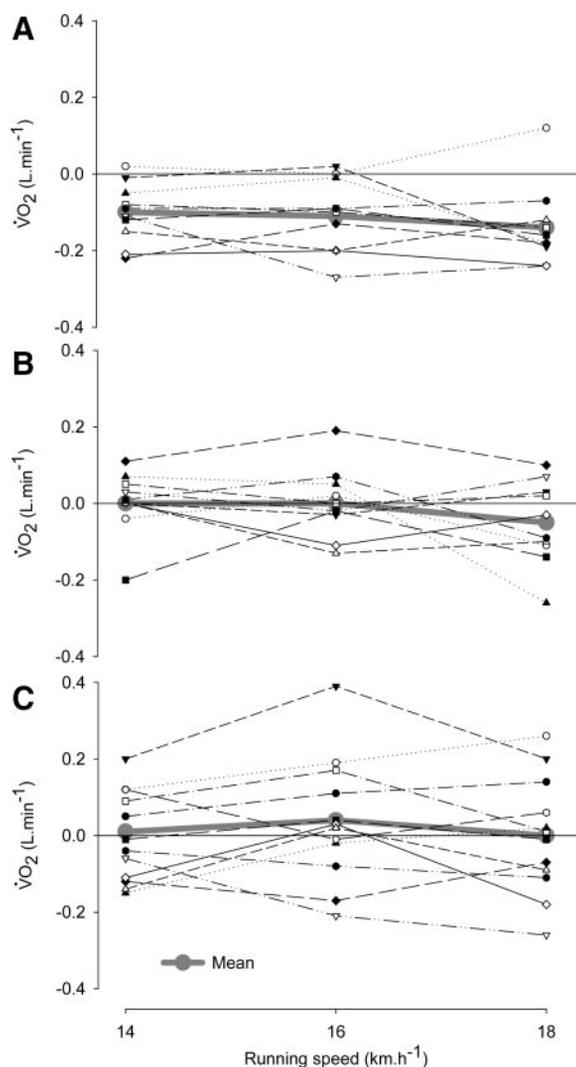


Fig. 2. Change in absolute $\dot{V}O_2$ (l/min) at 3 running speeds (14, 16, and 18 km/h) after 20 days of altitude exposure. Values are individual responses as well as mean responses for each intervention; mean values are represented with a thicker line and different shade as individuals; $n = 10$ (LHTL; 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 Hb_{mass} , $\dot{V}E$, RER, and HR. A second important finding of the present study was that living at 1,500 m and training at an altitude of \sim 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

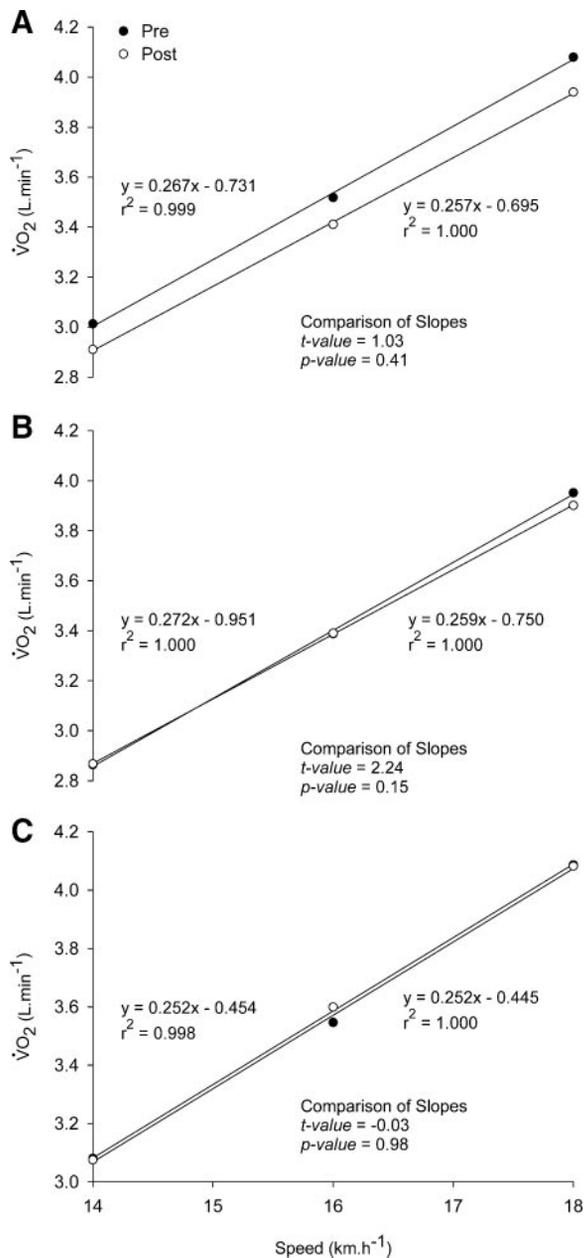


Fig. 3. Comparison of absolute $\dot{V}O_2$ (l/min) with speed (km/h) between pre- and posttest for the LHTL (A), LMTM (B), and LLTL (C) groups. Slope equations and r^2 values are given for each slope as well as the t and P values for the comparison of pre- and posttest slopes for the 3 groups.

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 hypobarica 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 hypobarica 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 $\dot{V}O_2$

	LHTL (a)		LHTL (b)		LHTL (c)	
	Pre	Post	Pre	Post	Pre	Post
Slope	0.205	0.204	0.205	0.198	0.205	0.170
y Intercept	0.273	0.158	0.273	0.264	0.273	0.226
r	0.998	0.999	0.998	0.998	0.998	0.998
r ²	0.996	0.997	0.996	0.996	0.996	0.996
<i>Comparison of slopes</i>						
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 prevalue 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 prevalue 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.

Table 4. *Cardiorespiratory and physiological measures*

	LHTL		LMTM		LLTL		LSD
	Pre	Post	Pre	Post	Pre	Post	
Body mass, kg	67.8±8.6	67.0±8.3	66.1±5.9	66.7±5.9	65.8±7.5	65.6±7.7	1.08
RER	0.92±0.04	0.94±0.03	0.92±0.04	0.94±0.04	0.94±0.04	0.95±0.04	0.02
HR, beats/min	157±8	156±13	156±10	154±14	153±11	153±12	3.6
Hb _{mass} , g	997±125	1,013±137	992±101	987±109	985±51	976±56	33
VE, l/min	95.7±15.6	97.7±17.0	95.1±10.5	96.7±8.7	93.7±15.4	96.2±17.2	3.6

Values are means ± SD, 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; VE, minute ventilation.

transient phenomenon, with lower levels merely reflecting a disturbance to muscle acid-base balance.

The slope between $\dot{V}O_2$ 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 $\dot{V}O_2$ vs. speed relationship. The same is true for a better ATP yield per mole of O₂ 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 O₂ cost after LHTL are difficult to elucidate but were not related to VE, HR, RER, or Hb_{mass}. The lack of change in Hb_{mass} strongly suggests that the mecha-

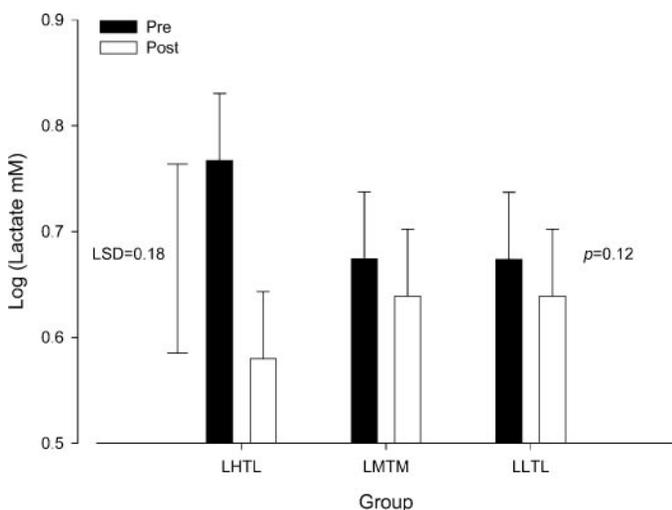


Fig. 4. Log of blood lactate concentration before (pre) and after (post) 20 days of altitude exposure, averaged across 3 running speeds (14, 16, and 18 km/h). Values are means ± SE; *n* = 10 (LHTL), *n* = 10 (LMTM), and *n* = 13 (LLTL). LSD is 2 times the standard error of the differences; any pair of 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 LH TL are sufficient time to acquire benefits from altitude acclimatization, although the elevation must be >2,000 m to provide sufficient stimulus to improve RE.

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