

## IMPROVED RUNNING ECONOMY IN ELITE RUNNERS AFTER 20 DAYS OF MODERATE SIMULATED ALTITUDE EXPOSURE

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**ABSTRACT**

To investigate the effect of altitude exposure on running economy (RE), 22 elite distance runners ( $\dot{V}O_{2\max}$   $72.8 \pm 4.4$  ml.min<sup>-1</sup>.kg<sup>-1</sup>; training volume  $128 \pm 27$  km.wk<sup>-1</sup>) homogenous for  $\dot{V}O_{2\max}$  and training were assigned to one of three groups; live-high (simulated altitude 2000-3100 m) train-low (natural altitude 600 m; LHTL), live-moderate train-moderate (natural altitude 1500-2000 m; LMTM) or live-low train-low (natural altitude 600 m; LLTL) for a period of 20 d. RE was assessed during three sub-maximal treadmill runs at 14, 16 and 18 km.h<sup>-1</sup> prior to and at the completion of each intervention. O<sub>2</sub> consumption ( $\dot{V}O_2$ ), ventilation ( $\dot{V}_E$ ), respiratory exchange ratio (RER), heart rate (HR) and blood lactate concentration [La] were determined during the final 60 s of each run, while haemoglobin mass (Hb<sub>mass</sub>) was measured on a separate occasion. All testing was performed under normoxic conditions ~600 m.  $\dot{V}O_2$  (L.min<sup>-1</sup>) averaged across the three sub-maximal running speeds was 3.3% lower ( $p=0.005$ ), after LHTL compared with either LMTM or LLTL.  $\dot{V}_E$ , RER, HR and Hb<sub>mass</sub> were not significantly different after the three interventions. There was no evidence of an increase in [La] 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  $\dot{V}_E$ , by preferential use of carbohydrate as a metabolic substrate, nor was it related to any change in Hb<sub>mass</sub>. We conclude that 20 d LHTL at simulated altitude improved the RE of elite distance runners.

Key words: Running, oxygen consumption, intermittent hypoxia

## INTRODUCTION

The effect of altitude training on endurance performance has been researched extensively (2-6, 8, 15, 18, 19, 21-23, 25-29, 33-36, 39, 41, 43-45). 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  $\dot{V}O_{2\max}$  and running performance (8, 26, 36). The mechanisms for these improvements are not clear, but have been proposed to include haematological 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/sleep at altitude and train near sea-level, 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 that improve  $O_2$  delivery and utilization (6, 28, 38, 45), decreased  $O_2$  utilization for a given speed would improve an athlete's running economy (RE). Two potential mechanisms for improved RE are either the amount of ATP per mole  $O_2$  used increases, or 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 have reported no change in sub-maximal  $\dot{V}O_2$  after a period of altitude exposure (26, 39) while another found improved RE after

intermittent hypoxic altitude exposure (25). Similarly, after a period of altitude acclimatization, sea level  $\dot{V}O_2$  during sub-maximal 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 as it reduces the utilization of  $O_2$  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 3000 m (19) to 6194 m (22, 27) which are generally higher than that 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 (LMTM) and simulated altitude (LHTL).

## **METHODS**

*Subjects.* Twenty-two elite male middle/long distance runners volunteered to participate in this study. Subjects all competed at a 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 2000-3100 m (Table 2). During this period subjects spent 5 nights per wk (9-12 h.night<sup>-1</sup>) in a normobaric hypoxic chamber with N<sub>2</sub> enrichment and 2 nights per 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 (1570 m) for a period of 20 d, with training altitudes ranging from 1500-2000 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 group (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 d. Subjects in the control group were tested within the 12 week period that the other interventions occurred with subjects in this group also 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 wash-out period of 6 wk was used for subjects in multiple groups. Prior to 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 sub-maximal speeds (see below), with  $\dot{V}O_{2\max}$  measured during the baseline test. Haemoglobin mass (Hb<sub>mass</sub>) was determined by carbon monoxide (CO) re-breathing on a separate occasion to the sub-maximal RE test.

*Treadmill testing.* RE was determined by measuring sub-maximal  $\dot{V}O_2$  for 4 min at constant running speeds of 14, 16 and 18 km.h<sup>-1</sup> on a custom-built motorized treadmill (Australian Institute of Sport, Belconnen, Australia).  $\dot{V}O_2$ , ventilation ( $\dot{V}_E$ ), respiratory exchange ratio (RER), heart rate (HR) and blood lactate concentration [La] 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  $\dot{V}O_2$  versus running speed was also determined as a measure of RE.  $\dot{V}O_{2max}$  was measured during an incremental test to volitional exhaustion performed 2 min after the third sub-maximal effort. Subjects completed an incremental protocol commencing at a treadmill speed of 18 km.h<sup>-1</sup> that was increased 1 km.h<sup>-1</sup> each min up until a speed of 20 km.h<sup>-1</sup>. After 1 min at 20 km.h<sup>-1</sup> (0% gradient), the treadmill gradient increased by 1% each min 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 [La].

*Gas analysis.* Respiratory gases were analyzed on a custom designed and built open-circuit indirect calorimetric 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 L aluminized bags. While one bag is being filled, the other has the expired volume and gas fractions determined. Standard algorithms are employed to compute minute values of  $\dot{V}_E$ , expired CO<sub>2</sub>,  $\dot{V}O_2$  and RER from the sum of two consecutive 30 s samples. The O<sub>2</sub> and CO<sub>2</sub> gas analysers (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 (TEM) (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<sup>-1</sup>. The TEM was established from duplicate trials conducted on 11 subjects prior to the start of the main study.

*Haemoglobin mass.* Before and after the three experimental periods, half the runners in each group underwent measurement of total Hb<sub>mass</sub> using a CO re-breathing technique modified from Burge and Skinner (7). The alterations used two doses of 99.5% CO re-breathed for 10 min each (20 ml initial dose and a second dose of 1.25 ml CO.kg<sup>-1</sup> body mass) and measuring %HbCO on capillary instead of venous blood (1). An average of %HbCO of four capillary blood samples determined on an ABL700 Series Blood Gas Analyser (Radiometer Medical A/S, Copenhagen, Denmark) for both CO doses was obtained and the  $\Delta\%$ HbCO (difference between first and second measures) used to calculate the Hb<sub>mass</sub> (7). The TEM for Hb<sub>mass</sub> in our laboratory is 2.7% (1) when using capillary blood samples.

*Statistical Analysis.* As some individuals participated in multiple groups the design was not fully balanced. Consequently, a general linear mixed model analysis (Chi-squared) was undertaken. Mean profiles along with the standard errors are shown graphically and the amount of variability between the groups is given by the least significant difference (LSD). Any pair of means differing by more than the LSD are considered significant ( $p < 0.05$ ) changes between pre- and post-tests amongst 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) 6th Edition (VSN International Ltd, Oxford, UK) was employed for statistical computation. Slopes and intercepts of  $\dot{V}O_2$  versus running speed were compared on group mean data using Prism™ software (2002) version 3.03 (GraphPad Software Inc., 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<sup>-1</sup>, as well as an assumed value of 0.304 L.min<sup>-1</sup> (LHTL only) for standing (0 km.h<sup>-1</sup>)  $\dot{V}O_2$  based on existing data (20, 32, 37).

## RESULTS

*Running economy.*  $\dot{V}O_2$  was similar across the three groups during the pre-test with means  $\pm$  SD of 3.53  $\pm$  0.51 (LHTL), 3.51  $\pm$  0.34 (LMTM) and 3.47  $\pm$  0.36 (LLTL) L.min<sup>-1</sup>, respectively, for the pooled data of the 3 running speeds. However, 20 days of LHTL simulated altitude exposure decreased (3.3%,  $p=0.005$ )  $\dot{V}O_2$  to 3.41  $\pm$  0.53 L.min<sup>-1</sup>, while the LMTM and LLTL groups remained unchanged with post-test means  $\pm$  SD of 3.50  $\pm$  0.32 and 3.49  $\pm$  0.37 L.min<sup>-1</sup>, 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  $\dot{V}O_2$  versus running speed pre- and post-intervention for the three groups. However, the LHTL slope was offset after the 20 d 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<sup>-1</sup>) and percentage (3.4%) change observed during the three running speeds, there was still no significant difference in the slopes between pre- and



post-tests (Table 3). We calculate that a 17% reduction in running and standing  $\dot{V}O_2$  was required to make the pre- and post-intervention slopes significantly different.

*Cardiorespiratory and physiological measures.* When the three groups were compared between pre- and post-tests 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 [La] ( $[La]_{log}$ ) was taken because initial analysis showed this measure was not normally distributed. The difference between pre- and post-tests for  $[La]_{log}$  for the three groups (pooled data of the three running speeds) was not significant ( $p=0.12$ ). However, the LHTL group showed a trend towards decreased (24%,  $p=0.12$ ) [La] after the altitude exposure with the pre-test mean  $\pm$  SD of  $2.5 \pm 1.1$  mM decreasing to  $2.1 \pm 1.2$  mM. The [La] in the other groups was unchanged, with pooled means  $\pm$  SD of  $2.1 \pm 1.2 - 2.0 \pm 1.0$  mM (LMTM) and  $3.0 \pm 1.3 - 2.8 \pm 1.3$  mM (LLTL) between pre- and post-tests, respectively (Fig. 4).

## DISCUSSION

The major finding from the present study was that 20 nights of sleeping at 2000-3100 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 when 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 sub-maximal  $O_2$  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  $km \cdot h^{-1}$ ) 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 1500 m and training

at an altitude of ~2000 m was insufficient stimulus to alter variables associated with RE.

Our results are not in accordance with the conventional view that sub-maximal  $\dot{V}O_2$  remains unchanged, at sea-level, after returning from a period of altitude exposure with multiple studies observing no change in sub-maximal  $\dot{V}O_2$ , at sea level (21, 26, 29, 39, 44), nor even changes in submaximal  $\dot{V}O_2$  under chronic hypoxic conditions up to 7440 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 4500 m) improved RE in highly-trained runners. Indeed, our findings are consistent with a growing number of studies that have showed that various forms of altitude exposure can reduce sub-maximal  $\dot{V}O_2$  (19, 22, 23, 25, 27). These exposures include mountain climbing over 3 wk to 6194 m (22, 27), 23 nights exposure to 3000 m simulated altitude (19), 3 wk exposure to intermittent hypobaria of 4500 m (25) and long term residence at 4200 m (23). A rigorous experimental design as well as good precision in our indirect calorimetry system gives 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 current study  $\dot{V}_E$  and HR did not markedly change after 20 days 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 (CHO) utilization for oxidative phosphorylation after a period of altitude acclimatization. It has previously been observed that 4,300 m altitude acclimatization for 21 d 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 towards 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 current study, there was no evidence to support a shift towards greater CHO utilization during sub-maximal exercise, as RER was not different between the 3 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 current investigation.

There was no significant difference in [La] after LHTL altitude exposure suggesting 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 [La] accumulation is an increase in skeletal muscle oxidative enzyme capacity (17), with a resultant shift in metabolism away from anaerobic towards 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 [La] may be a transient phenomenon with lower levels merely reflecting a disturbance to muscle acid-base balance.

The slope between  $\dot{V}O_2$  and running speed/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 more invasive procedures are required to detect this change. At the muscle level a reduced ATP cost of contraction should coincide with a change in slope of the  $\dot{V}O_2$  versus 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 (1500-2000 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 (3000–6200 m). In the present study the LHTL intervention, which resulted in improved RE, was conducted at an elevation between 2000-3100 m. While further work is clearly required to elucidate the dose-response relationship in terms of altitude and duration of exposure, the results of the current study suggests that a “threshold altitude” to alter economy might exist and may be between 2000-3100 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 with the results of studies conducted by others (8, 26). It is possible that 20 d of 8-12 h.day<sup>-1</sup> altitude exposure to 2000-3200 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 d training camp at 2690 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 2700 m have a higher  $Hb_{mass}$  than their sea-level counterparts (35). In the current study, investigating the effects of altitude exposure on RE, it is apparent that  $Hb_{mass}$  has no relationship with the improved RE demonstrated.

Despite our finding on 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/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 the subjects (apart from two who didn't race during the duration of the study) ran personal or season best times over distances ranging from 1500-10000 m within a month of the LHTL intervention. In comparison only 3 of the 13 LLTL subjects ran personal or season best times within one month of the control intervention.

In conclusion, the results of the present study demonstrate that sleeping at a simulated altitude of 2000-3100 m using the LHTL model for 20 d resulted in a 3.3% improvement in RE of elite distance runners, while living and training at moderate altitude (1500-2000 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 sub-maximal  $O_2$  cost after LHTL are difficult to elucidate, but were

not related to  $\dot{V}_E$ , HR, RER or  $Hb_{mass}$ . The lack of change in  $Hb_{mass}$  strongly suggests that the mechanism(s) underlying the enhanced RE is independent of accelerated erythropoiesis. Finally, our results suggest that 20 days of LHTL is sufficient time to acquire benefits from altitude acclimatization, although the elevation must  $>2000$  m to provide sufficient stimulus to improve RE.

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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
$\dot{V}O_{2max}$ (L.min <sup>-1</sup> )	4.90 ± 0.52	4.73 ± 0.42	4.75 ± 0.40	4.79 ± 0.45
$\dot{V}O_{2max}$ (ml.min <sup>-1</sup> .kg <sup>-1</sup> )	73.0 ± 2.8	71.7 ± 3.8	73.5 ± 5.7	72.8 ± 4.4
Training Volume (km.wk <sup>-1</sup> )	126 ± 26	132 ± 30	126 ± 26	128 ± 27
Training Duration (hrs.wk <sup>-1</sup> )	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 (LHTL), n=10 (LMTM) and n=13 (LLTL). LHTL, live-high train-low at simulated high altitude; LMTM, live and train at natural moderate altitude; LLTL, live and train at natural low altitude (600 m).

Table 2. *Live-high train-low (LHTL) simulated altitude protocol.*

Week	Mon	Tue	Wed	Thu	Fri	Sat	Sun
1	600 m	2000 m	2000 m	2200 m	2500 m	600 m	600 m
2	2500 m	2500 m	2500 m	2500 m	2700 m	600 m	600 m
3	2500 m	2700 m	2800 m	2800 m	3000 m	600 m	600 m
4	2700 m	2900 m	3000 m	3000 m	3100 m	600 m	600 m

Table 3. Comparison of  $\dot{V}O_2$  versus 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 <sup>2</sup>	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), post-intervention standing  $\dot{V}O_2$  adjusted to pre-value minus 0.12 L.min<sup>-1</sup> (absolute change that occurred during running at 14, 16 and 18 km.h<sup>-1</sup>); LHTL (b), post-intervention standing  $\dot{V}O_2$  adjusted to pre-value minus 3.4% (percentage change that occurred during running at 14, 16 and 18 km.h<sup>-1</sup>); LHTL (c), all post-values reduced by 17% in order to obtain significant difference between pre- and post-test slopes.

Table 4. *Cardiorespiratory and physiological measures*

Group	LHTL		LMTM		LLTL		LSD
Test	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 (b.min <sup>-1</sup> )	157 ± 8	156 ± 13	156 ± 10	154 ± 14	153 ± 11	153 ± 12	3.6
Hb <sub>mass</sub> (g)	997 ± 125	1013 ± 137	992 ± 101	987 ± 109	985 ± 51	976 ± 56	33
$\dot{V}_E$ (L.min <sup>-1</sup> )	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 post after 20 days altitude exposure, for the pooled data of three running speeds (14, 16 and 18 km.h<sup>-1</sup>); n=10 (LHTL), n=10 (LMTM), n=13 (LLTL) except Hb<sub>mass</sub>, in which, n=6 (LHTL), n=6 (LMTM), n=5 (LLTL). LSD, least significant difference which is two 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 post-tests amongst the 3 treatment groups. RER, respiratory exchange ratio; HR, heart rate (b.min<sup>-1</sup>); Hb<sub>mass</sub>, haemoglobin mass (g);  $\dot{V}_E$ , minute ventilation (L.min<sup>-1</sup>); LHTL, live-high train-low at simulated high altitude; LMTM, live and train at natural moderate altitude; LLTL, live and train at natural low altitude (600 m).

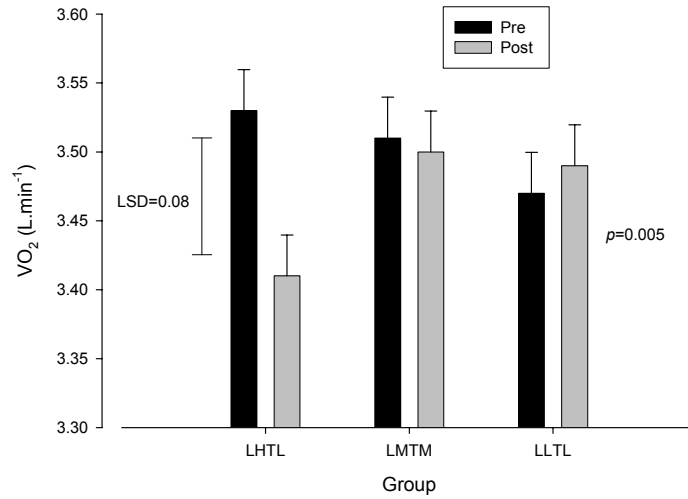


Fig. 1. Absolute oxygen consumption in L.min<sup>-1</sup> pre and post 20 days altitude exposure, averaged across 3 running speeds (14, 16 and 18 km.h<sup>-1</sup>). Values are means  $\pm$  SE; n=10 (LHTL), n=10 (LMTM), n=13 (LLTL). Changes between pre- and post-tests are significantly different ( $p < 0.05$ ) amongst the 3 treatment groups signified by the least significant difference (LSD), which is two times the standard error of the differences. LHTL, live-high train-low at simulated high altitude; LMTM, live and train at natural moderate altitude; LLTL, live and train at natural low altitude (600 m).

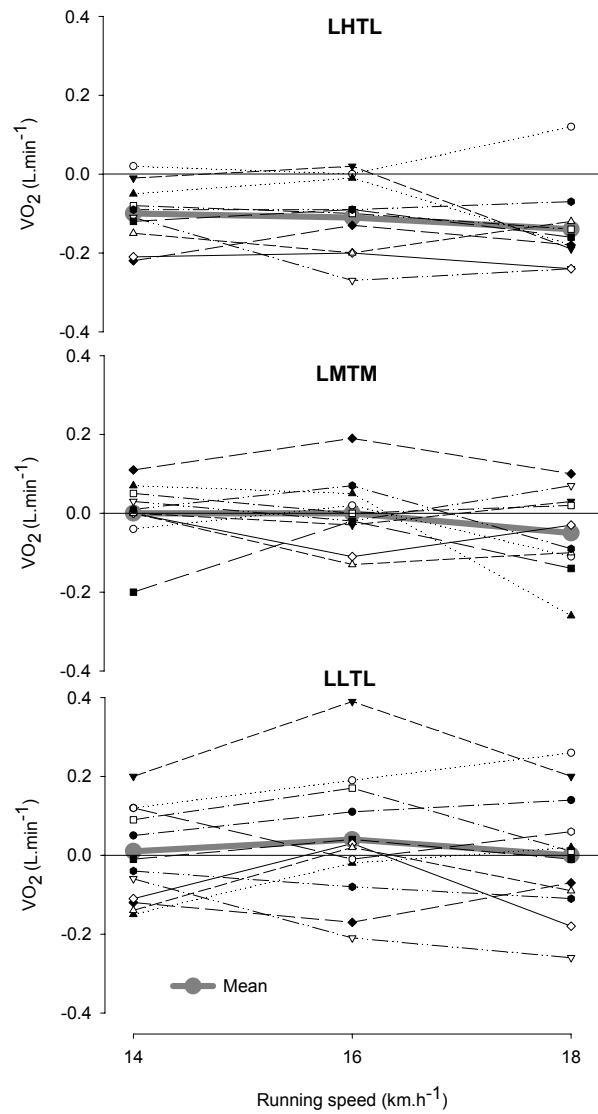


Fig. 2. Change in absolute oxygen consumption ( $L \cdot min^{-1}$ ) at 3 running speeds (14, 16 and 18  $km \cdot h^{-1}$ ) after 20 days altitude exposure. Values are individual responses as well as the mean response for each intervention, mean values are represented with a thicker line and different shade to individuals;  $n=10$  (LHTL),  $n=10$  (LMTM),  $n=13$  (LLTL). LHTL, live-high train-low at simulated high altitude; LMTM, live and train at natural moderate altitude; LLTL, live and train at natural low altitude (600 m).



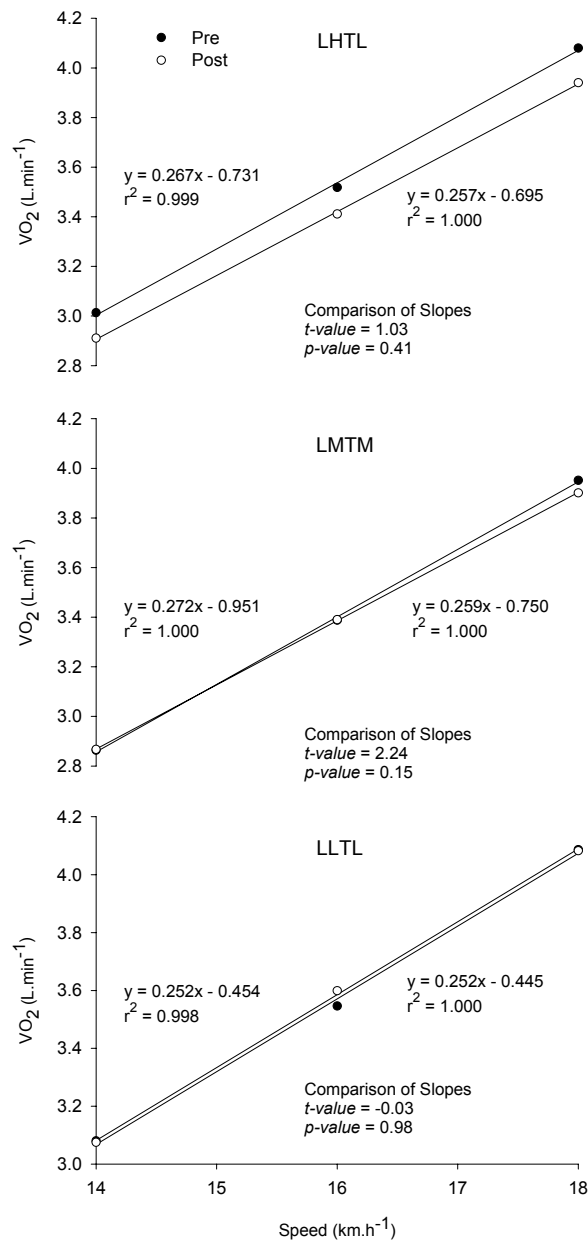


Fig. 3. Comparison of absolute oxygen consumption ( $\text{L}\cdot\text{mn}^{-1}$ ) with speed ( $\text{km}\cdot\text{h}^{-1}$ ) between pre- and post-test for the three intervention groups. Slope equations and  $r^2$  values are given for each slope as well as the  $t$ -value and  $p$ -value for the comparison of pre- and post-test slopes for the three groups. LHTL, live-high train-low at simulated high altitude; LMTM, live and train at natural moderate altitude; LLTL, live and train at natural low altitude (600 m).

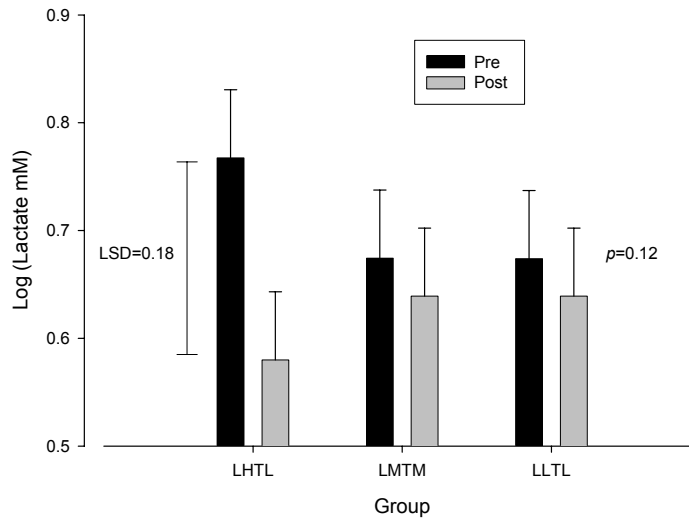


Fig. 4. Log of blood lactate concentration pre and post 20 days of altitude exposure, averaged across 3 running speeds (14, 16 and 18 km.h<sup>-1</sup>). Values are means  $\pm$  SE; n=10 (LHTL), n=10 (LMTM), n=13 (LLTL). LSD, least significant difference which is two times the standard error of the differences, any pair of means differing by more than the LSD are considered significant ( $p < 0.05$ ) changes between pre- and post-tests amongst the 3 treatment groups. LHTL, live-high train-low at simulated high altitude; LMTM, live and train at natural moderate altitude; LLTL, live and train at natural low altitude (600 m).