Does 'altitude training' increase exercise performance in elite athletes?

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ABSTRACT

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The general practice of altitude training is widely accepted as a means to enhance sport performance despite a lack of rigorous scientific studies. For example, the scientific gold-standard design of a double-blind, placebo-controlled, cross-over trial has never been conducted on altitude training. Given that few studies have utilised appropriate controls, there should be more scepticism concerning the effects of altitude training methodologies. In this brief review we aim to point out weaknesses in theories and methodologies of the various altitude training paradigms and to highlight the few well-designed studies to give athletes, coaches and sports medicine professionals the current scientific state of knowledge on common forms of altitude training. Another aim is to encourage investigators to design well-controlled studies that will enhance our understanding of the mechanisms and potential benefits of altitude training.

INTRODUCTION

Since its popularisation in the late 1960s, altitude training has become a commonly accepted mode of training and spawned a worldwide industry. Altitude training is now widely endorsed by elite athletes, coaches and sports organisations as a crucial component of serious training regimes. Within the last few years, impressive altitude training facilities have been built around the globe to enhance elite performance in both endurance and strength/power sports. Hypoxic training facilities have also appeared in local fitness studios offering recreational athletes the opportunity to train in hypoxic conditions and promoting the general utility of altitude training. Unfortunately, the actual scientific justification for benefits of altitude training is not as strong as its general perception.

The scientific gold-standard design of a doubleblind, placebo-controlled, cross-over trial has never been conducted on altitude training. Despite some 100 altitude training studies in the published literature, few of these have included a control group, and even fewer studies have been performed in a double-blind and placebo-controlled manner. This is unfortunate since parameters related to exercise performance may be influenced by placebo or similar effects.^{1 2} In fact a recent meta-analysis on the topic concluded that the performance gains that may be observed with altitude training could be related to a placebo or nocebo effect.³ In the end, however, it could be argued that if seen from an athletic point of view it does not matter if altitude training increases performance by a placebo effect as long as performance is increased. We empathise with this standpoint, but from a scientific point of view believe that further research is needed to elucidate the true effect of altitude training. In this brief review, we have taken the role of 'devil's advocate' to present a critical evaluation of the current state of knowledge regarding the effects of altitude training to challenge researchers to incorporate more rigorous controls in future studies. We have focused on the best-controlled studies and follow a historical progression.

Live high-train high

This was the first type of altitude training (a.k.a. classical altitude training) adopted by western athletes following the dominance of Eastern African runners at the 1968 Olympic Games. East African athletes were known to live and train at moderate altitudes and thus may have conferred a competitive advantage through acclimatisation. Western athletes guickly adopted this form of altitude training to (1) induce an altitude acclimatisationdependent increase in red blood cell volume (RCV) and at the same time (2) superimpose an additional training stimulus due to tissue hypoxia (see also the LLTH section). A high RCV in athletes is well documented⁴ and correlates well with overall exercise performance in elite athletes,⁵ and thus an attempt to increase RCV in order to increase performance seems valid. Whether training in hypoxia actually imposes an additional training stimulus is unknown. There exist numerous anecdotal reports on world class athletes who incorporate this type of altitude training into their preparations but actual well-controlled studies investigating the effects of LHTH on sea-level performance are scarce. Furthermore, almost all older studies have only included very few subjects. It is obvious that this type of altitude training is virtually impossible to blind from the participating athletes, and a placebo effect can hence never be completely ruled out.

The seemingly best controlled, but unfortunately largely ignored study was published by Mellerowicz in 1970.⁶ Before exposing 22 East German police officers (unfortunately only with moderate VO₂max's of ~50 ml/kg) to either a 4-week altitude (2020 m) or sea-level training intervention with a rigorously controlled exercise training programme, all volunteers were subjected to a 6 week long lead-in trial at sea level to assure stabilisation of fitness. Running performance (3000 m) and VO₂max were greatly increased in the altitude group compared to the sea-level control group for up to 2 weeks after termination of the intervention. These results support the notion that LHTH improves performance and VO_2max , yet the study does not explain if the hypothesis that altitude training would improve performance was revealed to the subjects prior to or during the study. This is important because subjects in the altitude group may have been positively influenced by the placebo effect, while subjects in the sea-level group may have been adversely affected by the nocebo effect.

A few years' later, Adams *et al*⁷ enrolled 12 competitive track runners (2 miles in ~9 min) to a 3-week-long altitude (2300 m) or sea-level training programme in a cross-over study design with concomitantly controlled training. Altitude training decreased 2 mile running time by 7 s, but no statistical differences could be obtained for this or for VO₂max, and hence the results were not as promising as those previously reported by Mellerowicz. Although the applied cross-over design is unmatched as of today, the conclusions were somewhat limited since training at altitude was performed at the same relative exercise intensity as at sea level, and hence at a lower absolute intensity. This study's major contribution was thus to raise concerns regarding the effect of altitude on training intensity and subsequent performance.

The weakness of Adam's study was, in part, addressed by Levine and Stray-Gundersen⁸ 20 years later. They subjected 39 college runners to 2 weeks of lead-in training and 4 weeks of controlled sea-level training where after the subjects were randomly assigned to 4 weeks of either living at 2500 m and training at 2500-2700 m (LHTH), living and training at sea level (Control), or living at 2500 m while training at lower altitudes between 1200 and 1400 m (live high-train low, LHTL). Following the various training camps, VO₂max was increased with LHTH and LHTL, but 5000 m running performance was only significantly increased in the LHTL group. The authors speculated that the reason for the lack of improvement in running performance with LHTH could be related to a reduction in peak running speeds do to an altitude-induced reduction in VO2max, yet could also not rule out potential placebo/ nocebo effects. This study subsequently led to a large number of follow-up LHTL studies, which will be discussed in the LHTL section.

Other LHTH studies including a control group have not found an increase in sea-level VO₂max following 4 weeks of altitude exposure to 1500–2000 m⁹ or in VO₂max and 3.2 km running performance after 4 weeks at 1740 m.¹⁰ These altitudes were likely too low to elicit a potential response.¹¹ Indeed, albeit in much less controlled studies, no increases in performance have been reported in recent studies at altitudes below 1900 m¹² ¹³ whereas sea-level time trials (or similar) have been reported to be increased after approximately 3 weeks at altitudes between 2100 and 2650 m.¹⁴ ¹⁵

On the basis of the present literature, it is impossible to provide a clear-cut conclusion concerning LHTH and potential gains in performance at sea level; however (1) LHTH may increase sea level performance in some, but not all, individuals (2) based on current knowledge it appears that athletes should live at an altitude at or above 2000 m to confer potential benefits from altitude training and (3) the duration of exposure should not be less than 3-4 weeks. We would highly recommend scientist with an interest in LHTH conduct their studies using elite athletes with controlled designs, since a major limitation in most studies is the inclusion of *trained subjects* (such as in the otherwise very nicely conducted Millerovicz study) rather than *elite athletes*. This may represent a problem since elite athletes may not be as responsive to a given stimulus as

healthy volunteers. A recent analysis concludes that athletes with an already high RCV may not increase their RCV any further with altitude training whereas an increase may be possible if RCV is low to begin with (figure 1B). The cross-over design may be the most feasible approach with elite athletes since blinding is nearly impossible.

Live or sleep high-train low

The general idea with live (or sleep) high-train low is to increase performance at sea level through an altitude-induced augmentation of red blood cell mass and thus oxygen carrying capacity. Athletes sleep at moderate altitudes to stimulate an increase in RCV, but avoid the problems associated with reduced VO₂max and training intensity at altitude by training at sea level. However, it should be recognised that at moderate altitudes relevant for altitude training, VO₂max increases over time with acclimatisation^{6 16} and it could be *speculated* that the relative importance of *training low* should hence decrease with

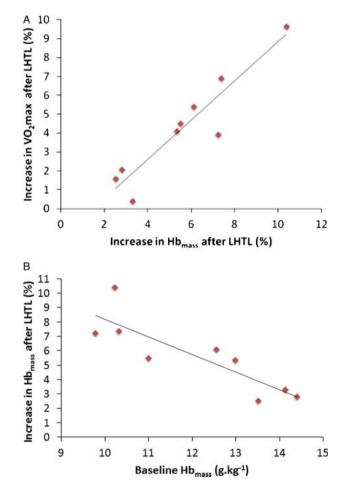


Figure 1 (A) The correlation between the relative gain in Hb_{mass} following live high-train low (LHTL) and the corresponding increase in V0₂max and B: The correlation between baseline total haemoglobin mass (Hb_{mass}, body-weight adjusted) measured prior to LHTL intervention, and the relative increases in Hb_{mass} following LHTL. The present analysis is based on nine previously published LHTL studies conforming to an appropriate 'dose' of hypoxia, that is, an altitude > 2000 m and a daily exposure to hypoxia > 12 h. Each point corresponds, for a given LHTL study, to the mean value (baseline or LHTL-induced change) reported by the authors for the LHTL group. Body-weight adjusted Hb_{mass} was either reported directly from the published data, or calculated by using the available mean body weight values. Reproduced from ⁴⁶ with permission.

acclimatisation, and perhaps vanish if sufficient acclimatisation is allowed. This could limit the logistic strains of LHTL to the first few weeks of an altitude training programme.

The use of LHTL was shown effective in increasing sea-level performance in college runners (VO₂max<65 ml/kg) by Levine and Stray-Gundersen.⁸ As mentioned above, VO_2max was increased following LHTH and LHTL, but running performance was only increased in the LHTL group.8 To what extent a potential placebo effect may have affected these results remains unknown, and it should also be noted that the response to LHTL varied greatly among individuals (figure 2). In support of the study by Levine, RCV, VO₂max and 5000 m running times were recently demonstrated to decrease following living at 2500 m with concomitant training at lower altitudes (1000-1800 m) for 24 days.¹⁷ In that study, however, subjects in the intervention group were orienteer's (five men and five women) whereas the control group were cross-country skiers (three men and four women), and unfortunately they were studied at different stages in their respective seasons. On the basis of these two non-blinded studies it seems (1) living at 2100-2800 m for approximately 3 weeks may increase RCV and (2) if at the same time training intensity can be maintained by descending to lower altitudes for training, then sea-level endurance performance is increased. The mechanism by which performance is improved appears related to either increased RCV¹⁸ or increased skeletal muscle efficiency.¹⁹ Although this remains unresolved, it may be seen from figure 1A that the changes in performance following LHTL is tightly correlated to an increase in RCV, albeit challenged by others.²⁰ A recent study including more than 100 subjects concludes that altitude exposure and/or altitude training does not change exercise economy,²¹ and that even continuous altitude exposure does not induce changes in skeletal muscle mitochondrial efficiency.²²

For practical reasons, it may not be convenient for athletes to spend time at natural altitude. To surpass this potential problem, studies have been conducted substituting altitude

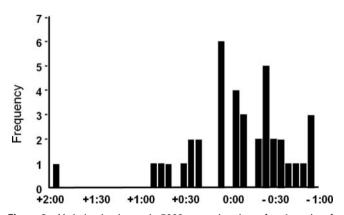


Figure 2 Variation in change in 5000 m running time after 4 weeks of altitude training in 39 college runners who were divided into 'responders' (\geq 14.1 s improvement, n=17: 8=ligh-low, 6=high-high-low, 3 high-high) and 'non-responders' (\leq 0 s improvement, n=15: 4=high-low, 4=high-high-low, 7=high-high). The responders experienced an 8% increase in estimated red cell volume (Evans blue) following the 4 weeks of altitude training whereas the nonresponders did not increase their red cell volumes. High-low=living at altitude (2500 m) and all training at low altitude (1200–1400 m); high-high-low=living at high altitude (2500–3000 m) and high-intensity 'interval' training at low altitude (1200–1400 m) and high-high=living and all training at high altitude (2500–3000 m). Reprinted from²⁹ with permission.

exposure with the use of 'nitrogen housing', where indoor living areas are flushed with N₂, or use of molecular oxygen sieves to decrease F_1O_2 and thus stimulate exposure to high altitude. At present, is remains unexplored if normobaric and hypobaric hypoxic exposure exert different responses with regard to accliamatory effects, and although the likelihood for such potential differences to affect, for example, RCV in a manner relevant for exercise performance seems minimal, studies investigating this need to be conducted. Many studies have been conducted using normobaric hypoxia as a stimulus and they have been reviewed elsewhere.²³ In general, the results are congruous with natural altitude training studies provided that the degree and duration of hypoxia are similar to the recommendations above. Recently, the first double-blinded and placebo-controlled study has been published with regard to aerobic²⁴ and anaerobic²⁵ performance following LHTL. In one of these studies Siebenmann²⁴ could not demonstrate any effects on any haematological or performance parameters in elite cyclists following 16 h/day at 3000 m normobaric hypoxia for four full weeks. As mentioned above, the absence of a positive response could be related to the already high RCV values of these athletes, which were greater than those of the subjects enrolled by Levine and Stray-Gundersen⁸ and Brugniaux²⁶ who reported the largest increases in VO2max following LHTL (figure 1). Thus, although the general recommendations for LHTL (>2000 m>12 h/day^{11 23}) may increase the performance of lower-end athletes, this is not necessarily the case for higherlevel athletes. In elite runners however, LHTL has been suggested to increase performance.²⁷ In this particular study, however, not even a control group was included and placebo and/or training camp effects cannot be ruled out to have contributed to the results.

An important but often neglected issue with regard to altitude training is the individual variation and reproducibility. This was recently addressed in a normobaric LHTL study²⁸ conducted by Christopher Gore's group. In that study male (VO₂max=73.1) and female (VO₂max=64.4) runners completed 2×3-week blocks of 14 h/day, 3000 m LHTL in a controlled but non-blinded manner. It was concluded that there is a large *individual* variation in the change in physiological and performance measures (as also noted by others,^{29 30} but that normobaric LHTL induces reproducible mean improvements in VO₂max and RCV Changes in time trial performance varied considerably more. The specific reason for the individual variation still remains to be elucidated, and further research in line with this is encouraged and could include studies on the potential interactions between nHb and [Hb], central circulatory changes including a hypoxia-dependent reduction of maximal heart rate,³¹ differences in protein synthesis and degradation³² and differences in buffer capacity.^{33 34}

To advance our understanding of LHTL, we encourage all future studies, at the very least those using normobaric hypoxia to include a placebo-controlled, double-blinded study design. There is no value in conducting additional uncontrolled studies as these will not enhance our understanding of LHTL, and the next logical step with regard to LHTL would be to repeat the experimental set-up applied by Siebenmann²⁴ to validate these data. Yet another point that could deserve further research is the degree of altitude of exposure. Today it is recommended not to surpass 3000 m (or an equivalent normobaric reduction in F_1O_2) since sympathetic stimulation above 3000 m is believed to have deleterious effects on among others sleep quality and hence recovery. Recent data however suggest that sleep quality is rapidly increased with acclimatisation to even

4559 m altitude,³⁵ and perhaps athletes could also cope with higher elevations as compared with current recommentations, and thereby reduce some of the individual responses seen today.

Live low-train high

Of all the hypoxic training protocols live low-train high (LITH) is the easiest one to commercialise, and some of the drive behind LITH may come more from industry than from actual science. However, according to the proponents of LLTH, during exercise at sea level one of the main stimuli for training-induced adaptations could be tissue hypoxia, although this remains unresolved (for review on the topic see 36). By performing training sessions in hypoxic conditions, it is speculated that the oxygen partial pressure in muscle tissue will be further lowered to provide an additional training stimulus and hence greater magnitude in training response. Investigating gene expression at the mRNA or protein level in muscle tissue clearly demonstrates that hypoxia evokes rapid cellular responses via hypoxia inducible factor,³⁷ but these studies cannot prove that training in hypoxia improves performance without interventional studies in athletes. Despite the relative ease to blind subjects to such hypoxic interventions, this has only been done in one study by Levine's group.³⁸ They observed no performance gains following 5 weeks of high-intensity hypoxic (15.3% O₂) training, refuting the LITH hypothesis. One factor which somewhat limits these conclusions is that the subjects were not elite athletes, although it seems difficult to see why including such athletes into the study would change the outcome as athletes in general need a more powerful stimulus to induce changes as compared with recreational athletes. In contrast to LHTH and LHTL, is seems safe to conclude that LLTH does not increase exercise performance at sea level in endurance athletes any more than simply training at sea level.

Intermittent hypoxia at rest

The most recent additions to the array of hypoxic training regimes involve intermittent hypoxia at rest delivered by repeatedly switching between breathing hypoxic and normoxic air for relatively short durations (60-90 min). Because the hypoxic exposures are brief (in some examples 5 min) the severity of hypoxia can be high (4500–6000 m). The precise rationale for such an approach is less clear than LHTL and LLTH, and at present the mechanisms remain obscure. With short hypoxic exposures lasting 5-10 min, a strong ventilatory drive is elicited which causes mild (and sometimes severe) respiratory alkalosis, left shifting the oxygen dissociation curve of the haemoglobin. As a result, the arterial O_2 content may be maintained at high levels (~90%), even when PaO_2 remains below 60 mm Hg.³ Thus, this type of hypoxic exposure may not cause much of a reduction in tissue oxygenation from an arterial oxygen content perspective. In addition, hypoxia causes vasodilatation and despite the high level of hypoxia used during these brief exposures, O_2 delivery to most tissues is likely barely reduced, due to the combination of both mechanisms (vasodilatation and leftward shift of oxygen dissociation curve of the haemoglobin).

In contrast to the other altitude training modalities, a high number of well-controlled studies all including a double-blinded design have been performed. In one of the studies, 14 nationalclass distance runners completed a 4 week regimen (5:5-min hypoxia-to-normoxia ratio for 70 min, 5 times/week) of intermittent normobaric hypoxia or placebo control. Following the experimental period there were no significant differences in VO_2max or 3000-m time-trial performance.⁴⁰ Subsequently, the same research group performed a double-blind, randomised, placebo-controlled trial to examine the effects of 4 weeks of resting exposure to intermittent hypobaric hypoxia (3 h/day, 5 days/week at 4000-5500 m). No differences in VO₂max, performance⁴¹ or exercise economy were reported.⁴² Also others have reported similar results with similar protocols. During 15 consecutive days, 20 endurance-trained men were exposed each day to breathing either a gas mixture (11% O_2 on days 1–7 and 10% O_2 on days 8–15, or a normoxic control gas), six times for 6 min. followed by 4 min of breathing room air for a total of six consecutive cycles. The results of this study demonstrated that 1 h of intermittent hypoxic exposure for 15 consecutive days has no effect on aerobic or anaerobic performance.⁴³ In conclusion, the use of intermittent hypoxic exposure does not increase sea-level performance and is not recommend. Further research in this area with respect to improving endurance performance does not seem warranted.

CONCLUSION AND FUTURE GUIDELINES FOR RESEARCH

Although we admit to taking a sceptical perspective for this review, our overall conclusion is that LHTH and LHTL may increase exercise performance in some but certainly not in all athletes, and that the potential response seems to be reduced in athletes with an already high RCV. It could be speculated that LHTH or LHTL could increase RCV in elite athletes of sport disciplines where a high RCV is not necessarily a prerequisite. In such disciplines, an elevated haemoglobin mass could perhaps increase performance by increasing the blood buffer capacity rather than by increasing the oxygen transport capacity. LLTH as well as intermittent hypoxic breathing at rest do not seem to improve endurance capacity any more than normoxic training and therefore we cannot support further research on endurance athletes in this area.

Unfortunately, the scientific ground on which altitude training is recommended is not solid enough, particularly to make specific recommendation for elite athletes. The importance of ruling out placebo effects is highlighted by some excellently conducted recent studies on the effects of 'carbohydrate mouth rinse'⁴⁴ and beet root juice⁴⁵ on exercise performance. The applied study designs and methodology in these studies allows for solid conclusions and publication in high-ranking journals which is in contrast to most altitude training studies. To increase our understanding with regard to altitude training the study design of future studies is critical.

 ${\rm Contributors}~{\rm CL}$ drafted the manuscript. GPM, JAC, PB and AWJ edited and approved the final version of the manuscript.

Competing interests None.

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