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Bachelor's thesis in Human Movement Science
Supervisor: Jørgen Danielsen
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Abstract

Formål: Denne litteraturstudien har som formål å undersøke sammenhengen mellom fysiologiske tilpasninger forårsaket av høydetrening og utholdenhetsprestasjon i lavlandet.

Metode: De inkluderte studiene hadde en høydeperiode på 3-4 uker mellom 1500 og 3000 meter. 8 originalartikler ble inkludert.

Resultat: De inkluderte studiene undersøkte aerob prestasjon, hematologiske tilpasninger og/eller ikke-hematologiske tilpasninger. Resultatene for alle de tre kategoriene var blandede.

Konklusjon: Høydetrening kan være fordelaktig for utholdenhetsprestasjon i lavlandet, men resultatene var ikke konkluderende og det er fortsatt usikkert hvilke fysiologiske aspekter som bidrar mest.

Nøkkelord: Høydetrening - Utholdenhetsprestasjon - Fysiologiske tilpasninger

Purpose: The aim of this literature review is to examine the relation between physiological adaptations caused by altitude training and sea level endurance performance.

Methods: The included studies had an altitude intervention lasting 3-4 weeks at 1500-3000 meters above sea level. 8 original articles were included.

Results: The included studies examined aerobic performance, hematological adaptations and/or non-hematological adaptations. Across all three categories the results were mixed.

Conclusion: Altitude training might be beneficial for endurance performance at sea level, but the results were not conclusive, and it is uncertain which physiological effects are contributing the most.

Keywords: Altitude training - Endurance performance - Physiological adaptations

Introduction

Altitude training is widely used by endurance athletes across multiple sports (1–3). There are several methods used for altitude training which can be subdivided into three main categories: Live high train low (LHTL), Live high train high (LHTH) and a combination of the two known as Live high train high, and low (LHTHTL). In this approach athletes live and perform low-intensity training at altitude, while high-intensity sessions are performed near sea level (4). Another noteworthy point is the difference between staying and/or exercising at actual altitude compared to staying and/or exercising at simulated altitude in a hypoxic chamber (5).

In endurance sports altitude training is often incorporated into an athlete's training program for one of two different reasons: either for acclimatization in preparation for a competition at a certain altitude or to induce adaptations that will lead to improved performance at sea level as well (4). The research literature, along with empirical evidence, seems to agree that altitude acclimatization is beneficial for performance in competitions at altitude. However, it is less clear if altitude training leads to adaptations that also improve performance at sea level. In practice, many top-level athletes claim it is vital for their success, while others don't experience the same effect. In addition, the research literature is split on the merits of altitude training for performance at sea level (6,7).

To understand the logic behind altitude training and the reason for its relevance a brief explanation of the hypobaric environment might be helpful. As we ascend above sea level the weight of the upper atmosphere combined with changes in ambient temperature reduces the barometric pressure. The composition of the ambient air (20.93% oxygen, 0.03% carbon dioxide and 79.04% nitrogen) remains the same, but the partial pressure of the gasses is reduced relative to the amounts at sea level. In addition, the ambient temperature is reduced at altitude by a rate of 1°C per 150 meters of ascent. This in turn reduces the amount of water vapor in the air relative to sea level which increases the risk of dehydration, especially during exercise (8).

There are several acute detrimental effects on athletic performance occurring at altitude. The reduced partial pressure of oxygen results in a more limited diffusion of oxygen between the capillaries and tissues. The body compensates for this reduced availability of oxygen by

increasing ventilation and cardiac output, mainly heart rate, both at rest and during exercise. There is also a decrease in blood plasma volume at altitude which causes stroke volume to fall during exercise, an effect that does not occur at sea level (8).

The theory behind altitude training is that, despite these acute detrimental effects, a prolonged sojourn will encourage beneficial physiological adaptations for endurance performance at altitude as well as at sea level. Perhaps the most discussed adaptation is the release of the hormone erythropoietin (EPO). EPO stimulates the production of red blood cells which in turn increases the hemoglobin (Hb) concentration of the blood. In theory this means that more oxygen can be transported per unit of blood which should be beneficial for endurance performance (8). But why is the literature split if it's this straightforward? Also, studies on highlanders in Tibet, Andes and Ethiopia suggest that other physiological adaptations than Hb_{mass} might be crucial as well (9). Tibetans and Ethiopian highlanders do not show a significant increase in Hb_{mass} when compared to lowlanders, while the Andean highlanders do (10).

Also, the acute detrimental effects occurring at altitude often leads to athletes exercising differently than they would at sea level (4). They may be more cautious and meticulous around both their exercise and recovery to cope with these effects. Also the effect of simply traveling to a different location, where exercise and recovery might be easier to take fully seriously than in normal everyday life, might be beneficial both physiologically and mentally (2). Are "training camp effects" like these the actual reason for the presumed effect of altitude training on sea level performance, or are there any genuine lasting physiological benefits, such as increased Hb_{mass} as well? Based on all of this we have formulated the following research question: can endurance performance at sea level be improved by physiological adaptations induced by altitude training?

Methods

Initially we needed to assert some exclusion criteria to limit the search to more relevant studies. We decided to exclude studies which were conducted in a period shorter than two weeks, studies that were mainly performed at an altitude below 1500 meters, and studies that were conducted on animals. The following search words were utilized in EbscoHost; "sea level performance" AND "altitude training" AND "physiology OR physiological". In

PubMed the search words that were utilized were “live high, train low”. In Google Scholar the search consisted of; “endurance performance”, “altitude training” and “sea level”. Lastly, “endurance performance” and “altitude training” were utilized in Web of Science. The research phase lasted from Tuesday 14th of February until Friday 3rd of March. We then thoroughly read through and selected the 8 original articles that were most relevant to our research question.

Results

The included studies examined elite, collegiate and junior level athletes across three different sports: running, swimming and cycling. Some used hypoxic chambers, while others were completed at natural altitude. Although related, the included studies investigated slightly different aspects of adaptations induced by altitude training. For the purpose of clarity, the findings have been divided into three separate categories: aerobic performance, hematological adaptations and non-hematological adaptations. The first category contains results from aerobic performance tests and the rest are physiological adaptations.

Table 1: Overview of original studies

Authors	Title	Participants	Methods	Results
Levine and Stray-Gundersen (1997)	“Living high-training low”: Effect of moderate-altitude acclimatization with low-altitude training on performance	39 Collegiate runners	<p>LHTH: N=13 living and training at 2500m. LHTL: N=13 living at 2500m, training at 1250m. Control: N=13 living and training at 150 m.</p> <p>4w altitude camp for all groups. Before the altitude camp all groups spent 6w at SL to normalize iron stores (with iron supplementation). First 2w to familiarize subjects with testing and training protocols, followed by 4w SL training camp.</p>	<p>Altitude groups: Increased $\dot{V}O_2$ max (5%) in direct proportion to an increase in RCM (9%; $r = 0.37$, $P < 0.05$) for both LHTH and LHTL. 5000m time improved similarly ($22.3 \pm 5s$) after SL camp for all groups, but only significant for LHTH. Only LHTL improved further after altitude ($13.4 \pm 10s$), in direct proportion to increase in $\dot{V}O_2$ max ($r = 0.65$, $P < 0.01$). Both other groups performed worse. LHTH improved 5000m time 2-3w after camp, but not significantly. No significant change in the running economy for any group.</p> <p>Control: No significant changes. Non significant decrease in $\dot{V}O_2$ max.</p>
Stray-Gundersen et al. (2001)	“Living high-training low” altitude training improves sea level performance in male and female elite runners	22 Elite runners	<p>LHTHTL: Living 27 days at 2500 m, performing high-intensity, and high-velocity training at 1250 m. All other training took place at 1250-3000 m.</p> <p>Athletes were assessed at SL the week before and after the altitude sojourn. All athletes received oral liquid iron supplementation (Feo-Sol, 9 mg elemental iron/ml) with dose adjusted on the basis of plasma SF concentration.</p>	<p>3000m performance at SL significantly improved. Improved TT performance by 5.8s (95% confidence limits 1.8–9.8 s) or 1.1% (95% confidence limits 0.3–1.9%) in correlation to $\dot{V}O_2$ max significantly increasing by 3% (72.1 ± 1.5 to 74.4 ± 1.5 ml · kg⁻¹ · min⁻¹). Significant correlation between $\dot{V}O_2$ max and 3000m TT ($r = -0.48$, $P = 0.02$). EPO nearly doubled 20h after ascent (8.5 ± 0.5 to 16.2 ± 1.0 IU/ml). Hb_{mass} increased on acute ascent to altitude, remained high throughout the camp and was significantly elevated on SL return. Hematocrit was significantly elevated on day 19 at altitude and remained elevated on SL return.</p>
Saunders et al. (2009)	Improved running economy and increased hemoglobin mass in elite runners after extended moderate altitude exposure	18 Elite runners	<p>LHTL: N=9 sleeping in hypoxic chamber (2860m) for 46±8 nights. 5 nights/w over 12w period. Training near SL (600m). Training volume 127 ±31 km/w. Control: N=9 living and training near SL (600m). Training volume 113 ±38 km/w.</p> <p>Testing performed pre and 2 days post intervention at SL.</p>	<p>LHTL improved running economy compared to controls. RE measured as decreased submaximal $\dot{V}O_2$ at 14, 16 and 18 kph on treadmill, (-3.2%, 90% confidence intervals, -1.0% to -5.2%, $p = 0.02$). Increased Hb_{mass} (4.9%, 2.3–7.6%, $p = 0.01$). Decreased submaximal heart rate (-3.1%, -1.8% to -4.4%, $p = 0.00$). Non-significant increase in $\dot{V}O_2$ max (1.5%, -1.6 to 4.8, $p = 0.41$). Trivial correlation between change in Hb_{mass} and $\dot{V}O_2$ max ($r = 0.04$, $p = 0.93$). Hypoxic exposure of ~400h was sufficient to improve Hb_{mass}. O_2 carrying capacity improved, though the mechanism(s) to explain were not identified.</p>

Mohammadi Mirzaei, R & Mohammadi Mirzaei, M (2020)	Responses to altitude training in terms of hematological parameters and performance in elite endurance runners	32 Elite runners	<p>Four equal LHTHTL groups (n=8) living and training at 1500m, 2000m, 2350m and 2500m respectively, with hard sessions at 1400m.</p> <p>4w of training and assessment at SL, followed by 4w altitude camp. 3w SL to retake tests. All runners performed similar training, including strength and plyometrics. 1500m group ran 100km/w, 2000m and 2350m did 120km and 2500 did 140km.</p>	<p>Significant improvement (2-3%) in 3000m TT for the groups living at 1500m, 2000m and 2350m ($P \leq 0.05$). 2500m group improved similarly to 1500m group, but results were not significant. No significant changes in HIF-1 alpha levels observed ($P \geq 0.05$) compared to baseline or between groups. Significant increase in PGC-1 alpha and EPO after altitude intervention for all groups ($P \leq 0.05$), effect more pronounced the higher the altitude.</p>
Friedmann et al. (2005)	Individual variation in the erythropoietic response to altitude training in elite junior swimmers	16 Junior swimmers	<p>EPO levels were measured before and after 4h exposure to altitude.</p> <p>3w at 2100-2300 m. 60-70 km swim training/w, mainly endurance training. About 30% training concentrating on improving swimming style.</p>	<p>Increased EPO(10-185%) after 4 hour exposure to altitude in hypoxic chamber. Large inter-individual variation. Significant correlation ($p < 0.001$) with acute EPO increase during altitude training. No correlation with change in total Hb_{mass}. Hb_{mass} increased significantly (6% average). Change in SL performance not related to change in total Hb_{mass}.</p>
Rodriguez et al. (2015)	Altitude training in elite swimmers for sea level performance (Altitude Project)	54 Elite swimmers	<p>LHTH: 4w living and training at 2320 m (n=11) LHTH3: 3w living and training at 2320 m (n=11) LHTHTL: 4w living at 2320 m, and training at 2320 m and 690 m (n=11). Control: LLTL 4w living and training at SL (190 m or 655 m) (n=11).</p> <p>Swim time trials for all groups: TT50, TT400 and TT100/TT200 for sprinters/non sprinters. $\dot{V}O_2$ max and HR measured with incremental 4x200m test. Tests were performed pre and post trial and once weekly after return to SL (PostW1 to PostW4). Hb_{mass} was measured pre and weekly during the trial.</p>	<p>TT100/TT200 unchanged or worse at post, but improved by 3.5% regardless of altitude or SL after at least one week of SL recovery. LHTHTL improved the most 2 (5.3%) and 4w (6.3%) after the trial. LHTHTL also improved the most in TT50 and TT400 2 (5.2% and 4.2%) and 4w (5.5% and 4.7%) after the trial.</p> <p>No significant changes in $\dot{V}O_2$ max in either group. Control (1.9%; $\pm 1.5\%$), LHTH (1.1%; $\pm 2.6\%$), LHTH3 (1.5%; $\pm 2.5\%$) and LHTHTL (1.3%; $\pm 1.4\%$) ($\Delta\%$ from PRE, $P > 0.05$). Performance improvement not linked with changes in $\dot{V}O_2$ max or Hb_{mass}. Trivial and insignificant correlation between change in Hb_{mass} and change in $\dot{V}O_2$ max $r=0.04$ ($p=0.93$). Hb_{mass} increased by 6.2% (at W4, 6.2%; 90% CI, $\pm 1.1\%$; $P < 0.001$) in LHTH and 3.8% (at W3, 3.8%; ± 2.3; $P = 0.08$) in LHTH3. No significant changes in Hb_{mass} in LHTHTL (at W4, 1.3%; ± 1.8; $P = 0.71$) or Control. ($\Delta\%$ compared with PRE).</p>

Garvican et al. (2012)	Time course of the hemoglobin mass response to natural altitude training in elite endurance cyclist: Altitude training and hemoglobin mass	13 Elite cyclists	<p>Altitude: N=8, living at 2760 m and training between 1000-3000 m.</p> <p>Control: N=5, living and training at SL (600m).</p> <p>Subjects were monitored for 5w, with a 3w training camp. Both groups did 500-600 km/w of cycling.</p> <p>Hb_{mass} and EPO were measured before training camp, day 5-6, day 10-11, day 19-21, when returning to SL and 10 days after returning.</p>	<p>Altitude group increased mean Hb_{mass} at D11 (2.9% ± 2.0%) compared to baseline, and possibly greater compared with controls (+2.6%, 90% CL=-2.4 to 7.7%). Mean Hb_{mass} likely to be greater than controls at end of altitude period (D19: +3.3%, 0.2-6.5%). Differences in Hb_{mass} between groups were unclear at D31 (+0.8%, -1.7 to 3.3%).</p> <p>EPO increased 64.2% (± 18.8%) after 2 nights at altitude and was almost certainly higher than controls. On return to SL EPO decreased (41.1 ± 31.8% below baseline). By D31 EPO increased towards baseline, but remained likely lower (-22.9 ± 59.6%). Controls had no substantial change.</p>
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Siebenmann et al. (2012)	“Live high-train low” using normobaric hypoxia: a double-blinded, placebo-controlled study	17 Elite cyclists	<p>LHTL: N=10 living in normobaric hypoxia (3000m) 16 h/day for 4w and training at <1200 m.</p> <p>Control: Placebo group (n=6) living and training at the same place as LHTL in normobaric normoxia.</p> <p>First 2w lead-in period, familiarizing with environment and baseline testing. Training followed personal training habits with constant intensity and volume. All subjects received oral iron supplementation throughout intervention period.</p>	<p>LHTL: Hb_{mass} was not affected and remained unchanged. EPO was higher after 2-6 days of LHTL exposure (P < 0.05), but returned to normal values further into the trial. $\dot{V}O_2$ max did not increase in any of the LHTL subjects. Increase in 26.15km TT performance by 2% (P = 0.12). No effect on cycling economy (Average of $\dot{V}O_2$ at 200 watts in normoxia and $\dot{V}O_2$ at 150 watts in hypoxia).</p> <p>No statistically significant difference in Hb_{mass}, hematocrit, cycling economy, TT performance or $\dot{V}O_2$ max compared to Control.</p> <p>Control: No changes in Hb_{mass}, EPO or $\dot{V}O_2$ max. 5% increase in TT performance (P=0.12). No effect on cycling economy.</p>
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Aerobic performance

Both Siebenmann et al. and Stray-Gundersen and Levine (7,11) looked at exercise economy, and found that it was unchanged in all groups, while Saunders et al. (6) found an improvement in running economy in LHTL compared to the controls. All the groups in these studies (2,3,7,12) improved their time trial (TT) performance. In the study by Levine and Stray-Gundersen (11) only LHTL improved their TT performance while LHTH and controls failed to achieve a similar result. Siebenmann et al., Saunders et al. and Rodriguez et al. (2,6,7) found no significant increase in the athletes' $\dot{V}O_2\text{max}$. Levine and Stray-Gundersen (11) and Stray-Gundersen et al. (3) found increased $\dot{V}O_2\text{max}$, and the increase was in direct proportion with both the increase in red cell mass (RCM) and TT performance.

Hematological Adaptations

Most of the studies found an increase in EPO, Hb_{mass} or RCM for the altitude groups (1,3,6,11,12). Siebenmann et al. (7) found no significant difference in EPO, Hb_{mass} or hematocrit between LHTL and the controls. Rodriguez et al. (2) found an improvement in Hb_{mass} for LHTH and LHTH3 at the end of the trial, but no significant changes for LHTL or the controls. Friedmann et al. (5) concluded that a 4 hour exposure to hypoxia could predict the increase of EPO at natural altitude, however the increase in EPO was not related to a final change in Hb_{mass} .

Non-hematological Adaptations

Mohammadi Mirzaei and Mohammadi Mirzaei (12) confirmed that all four LHTL groups significantly increased the enzyme PGC-1, but not hypoxia inducible factor (HIF-1). Rodriguez et al. (2) did not measure any non-hematological factors, but hypothesized that other factors than erythropoiesis (production of red blood cells) must be playing a role in explaining the effects of altitude training.

Discussion

Aerobic performance

In general, almost all the groups, regardless of altitude, improved their TT performance. In the study by Levine and Stray-Gundersen (11) only LHTL improved their TT performance after the altitude training camp while LHTH and controls failed to achieve a similar result. When they introduced the LHTL approach to altitude training Stray-Gundersen et al. (3)

found that the athletes improved TT performance by 5.8 seconds on average. Mohammadi Mirzaei and Mohammadi Mirzaei (12) found that LHTHTL improved TT performance for all groups, although results were not significant for the 2500m group. Rodrigues et al. (2) found that all groups improved their TT performance, but LHTHTL improved the most. Siebenmann et al. (7) did not find any significant difference in TT performance between LHTL and the controls. Although most of the groups improved their TT performance it is still unclear if the improvement was caused by the altitude or just the exercise in general.

Three of the studies investigated changes in movement economy measured as $\dot{V}O_2$ during submaximal workloads. Levine and Stray-Gundersen (11) found that it was unchanged in all groups, while Saunders et al. (6) saw an improvement in running economy in LHTL compared to the controls. In the study by Siebenmann et al. (7) neither LHTL nor the controls improved cycling economy compared to baseline and there was no significant difference between the groups either. The studies by Saunders et al. (6) and Siebenmann et al. (7) were both executed in hypoxic chambers, yet they found complete opposite results. The two studies differ in two key aspects; Saunders et al. examined runners as opposed to cyclists and their study lasted for 12 weeks rather than 4 weeks. The total amount of hours spent in hypoxia was similar across the two studies, 415 and 450 hours respectively. The runners however spent only 9 hours, 5 nights a week in the hypoxic chamber, while the cyclists spent 16 hours every day in the hypoxic chambers. Based on these results it might be beneficial to prolong the duration of the hypoxic exposure without necessarily increasing the total amount. Overall, these conflicting results suggest that there is no clear relationship between altitude training and improvements in movement economy.

Several of the studies also measured the $\dot{V}O_2$ max of the subjects. Neither Siebenmann et al. (7) nor Saunders et al. (6) found any significant difference between LHTL and the controls after the intervention. Also, there were no significant improvements in $\dot{V}O_2$ max in either of the four groups in the study by Rodriguez et al. (2). However, Levine and Stray-Gundersen (11) measured a significant increase in $\dot{V}O_2$ max for LHTL and LHTH, but not in the controls. Stray-Gundersen et al. (3) also found significant improvements in $\dot{V}O_2$ max for LHTHTL. For both studies the increase was in direct proportion to improved TT performance. The obvious difference between the studies that found an effect and the ones that did not, apart from Rodriguez et al, is the use of natural altitude vs hypoxic chambers.

This might possibly explain the disparity in the results but needs to be studied further to determine an effect more clearly. Again, the findings are contradictory indicating that there is no clear relation between altitude training and increased $\dot{V}O_2$ max.

Hematological Adaptations

The two studies that used hypoxic chambers over a prolonged period found contradictory results in the hematological response to altitude training. Siebenmann et al. (7) found no significant difference in EPO, Hb_{mass} or hematocrit between LHTL and the controls. Saunders et al. (6) on the other hand concluded that LHTL in a hypoxic chamber was beneficial for an increase in Hb_{mass} . These contradictory results might be explained by differences that were discussed previously in this study. Friedmann et al. (5) concluded that a 4 hour exposure to altitude in a hypoxic chamber could predict the increase of EPO at natural altitude, however the increase in EPO was not related to a final change in Hb_{mass} . This result demonstrates that individuals react differently to the hypoxic environment.

The studies that utilized natural altitude generally found positive results when examining the hematological response to altitude training. Garvican et al. (1) measured an increase in Hb_{mass} in the altitude group compared the controls. Levine and Stray-Gundersen (11) found that both LHTL and LHTH increased RCM while the controls did not. The increased RCM was in proportion with improved TT performance. Stray-Gundersen et al. (3) documented a significant increase in Hb_{mass} and hematocrit values for LHTHTL. Rodriguez et al. (2) found an improvement in Hb_{mass} in LHTH and LHTH3 at the end of the trials, while there were no significant changes in Hb_{mass} for LHTHTL or the controls. Changes in Hb_{mass} was not linked with changes in $\dot{V}O_2$ max or TT performance. These results suggest that athletes are likely to experience an increase in Hb_{mass} after a training camp at natural altitude, but whether the increase is meaningful for sea level performance remains unclear.

Non-hematological adaptations

Mohammadi Mirzaei and Mohammadi Mirzaei (12) measured the levels of HIF-1 and PGC-1 in the subjects. The 2500m group experienced a non-significant increase in HIF-1 levels that was not present in the other groups. The levels of PGC-1 on the other hand increased in all four groups, and the changes overlap with the increase of mitochondrial biogenesis that occurs while training at altitude. The authors explain that PGC-1 coordinates the upregulation

of muscle mitochondrial content, as well as accounting for metabolism and oxidative phosphorylation. Furthermore, mitochondrial activity increases after altitude training because of increased PGC-1 which in turn stabilizes HIF-1. In total these physiological adaptations should be beneficial for endurance performance, but the results from this study are not conclusive (12).

Rodriguez et al. (2) did not measure any non-hematological factors but hypothesize that other factors than erythropoiesis must be playing a role in explaining the effects of altitude exposure. Their results seem counterintuitive with no clear relation between changes in $\dot{V}O_2$ max, Hb_{mass} or TT performance. The authors list several plausible factors that might explain the effects of altitude training; improved muscle efficiency at mitochondrial level, greater muscle buffering, ability to tolerate accumulating lactate and improved O_2 flux to exercising muscles. Gore et al. (13) discuss several of these factors of altitude training in their review study. They specify that the response to hypoxia is multifaceted and that a deeper understanding of non-hematological factors is required to fully understand its role in the adaptations related to altitude training. These non-hematological factors might explain the possible effects of altitude training for sea level performance and should be studied further.

Optimal altitude training paradigm

LHTL, LHTH and LHTHTL are the three altitude training paradigms discussed in this study, but what is the optimal altitude strategy based on the results of the included studies? Because of the acute detrimental effects caused by the hypobaric environment, training intensity should be somewhat reduced at altitude. However, a reduction in the intensity of high-intensity workouts can lead to a reduced mechanical stimulus which can in turn be detrimental for competitions at sea level. To bypass this negative effect high-intensity workouts should probably be performed near sea level. For low-intensity workouts this effect is less of a concern, and these workouts can therefore be performed at altitude both for practical reasons and to prolong the altitude exposure (4). Based on these findings LHTHTL might prove to be the most efficient strategy for altitude training.

The results are not conclusive regarding how high it is most beneficial to reside, but it seems that the optimal natural altitude lies somewhere between 2000 meters and 2500 meters (2,3,11,12). For hypoxic chambers on the other hand this is not necessarily the case. For

practical reasons it might be most beneficial to only sleep in the hypoxic chamber at night. This way the athletes are not confined to a small and uncomfortable space for the whole training camp. In an approach like this it seems necessary to increase the hypoxic exposure beyond the 2500 meter threshold that might exist at natural altitude, to around 3000 meters (6). If this increase in altitude is beneficial because of some difference between natural altitude and hypoxic chambers is not known. It might simply be that the total amount of hours is lower in an approach like this, giving the athletes time to “recover” in normoxia.

Methodological critique

Finally, several of the included studies have a clear statistical weakness that needs to be addressed. Due to a number of statistical phenomena, such as regression to the mean (14), it is paramount that between-group differences are compared rather than within-group differences in randomized controlled trials. This is the most correct way to estimate the “true” effect, if one exists, of an intervention. Most of the included studies are randomized controlled trials of some form, yet almost all of them fail on this point and rely only, or heavily, on within-group differences. By doing this the results that are produced can be terribly misleading and involve a true alpha level of 0.5 rather than 0.05 (15,16).

In addition, the number of participants in most of the studies is probably too small to clearly determine the effect of an altitude intervention. If a clear and sizable effect is measured, then only a small number of subjects are needed to produce statistically significant results. If the measured effect is small and unclear on the other hand, a greater number of subjects are needed if such results are to be produced (17). Based on the included studies the effect of altitude training, if it exists, seems to be quite limited. Therefore, larger sample sizes are needed to produce statistically significant results. Because of these fundamental statistical errors, it is hard to determine if there is a causal relationship between altitude training and endurance performance at sea level based on the included studies.

Conclusion

Can endurance performance at sea level be improved by physiological adaptations induced by altitude training? Possibly yes, but the results appear ambiguous. If there is an effect of altitude training, it seems to account for a tiny percentage of the total endurance performance at sea level. Additionally, it is uncertain which adaptations are contributing the most to an

improvement in performance at sea level. Erythropoiesis and increased Hb_{mass} might contribute, but other non-hematological adaptations can possibly prove to be more important factors and need to be studied further.

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