Chapter 27

Altitude and hypoxic training in swimming

Martin J. Truijens,¹ Ferran A. Rodríguez²

¹Faculty of Human Movement Sciences, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands
mj.truijens@fbw.vu.nl

²Institut Nacional d’Educació Física de Catalunya, Universitat de Barcelona, Spain
farodriguez@gencat.cat

Key Words
Altitude training, hypoxia, hypoxic training, intermittent hypoxia

Abstract
Altitude/hypoxic training is nowadays a common practice among swimmers although its benefits are still controversial in scientific literature. Traditional altitude training (“live high-train high”) is still the most frequently used method in swimming, even though from a physiological perspective the “live high-train low” strategy appears to be a more promising alternative. While acute hypoxia deteriorates swimming performance, chronic hypoxia may induce acclimatization effects, mainly through the acceleration of red blood cell production, which could improve aerobic capacity and therewith performance upon return to sea level. Other potential benefits such as improved exercise economy, enhanced muscle buffer capacity and pH regulation, and improved mitochondrial function have also been postulated. In order to get a better picture of the potential usefulness of altitude and hypoxic training in swimming this chapter will (i) briefly review the acute and chronic effects of hypoxia, (ii) describe traditional and current methods of altitude/hypoxic training, (iii) discuss the scientific evidence on the effects of altitude/hypoxic training on sea level swimming performance, and (iv) give some practical guidelines for altitude/hypoxic training.

Introduction
Checking the records of several swimming organizations and altitude/hypoxic training facilities around the world it becomes clear that altitude/hypoxic training plays an important role in preparing elite and subelite swimmers all over the world [59, 57, CAR Sierra Nevada 2008 (personal communication)]. They devote considerable amounts of time, effort, and material resources to train in real or simulated altitude, with the expectation of improved performance at sea level.

Unfortunately, there is a remarkable lack of controlled studies on altitude training in swimming in the scientific literature, and the scientific evidence supporting most approaches to altitude/hypoxic training in general is inconclusive [59, 57]. Moreover, in spite of the important amounts of research carried out over the last decades the physiological mechanisms through which altitude/hypoxic training should be effective
in enhancing performance are still controversial. Field observations and also research studies show that altitude/hypoxic training may work for some athletes and not for others [8].

It has been estimated that an Olympic swimmer should improve his or her performance by about 1% within the year leading up to the Olympics to stay in contention for a medal [37] interestingly, a recent forthcoming meta-analysis concluded that the expectable performance benefit from altitude/hypoxic training for elite athletes can be as high as 1.6% [6]. Perhaps a worthy strategy if a medal is just some tenths of a second away. The right question today may not be if altitude training works, but how, when, and for whom it works. This chapter aims to provide the reader with an overview of peer-reviewed scientific research on altitude training in swimming for the improvement of performance at sea-level, together with a physiological rationale for altitude training in general.

Hypobaric and normobaric hypoxia
Altitude is defined as ‘the circumstance of a reduced partial oxygen pressure in ambient air’. This condition can be created by a decrease in barometric pressure, leading to a reduction in the inspired partial pressure of oxygen, known as hypobaric hypoxia [5], or by a decrease in the inspired oxygen fraction without changes in barometric pressure; this is known as normobaric hypoxia [4]. Hypobaric hypoxia can be obtained by 1) an ascent to natural/terrestrial altitude, and 2) at sea level using a hypobaric chamber. Examples of devices that provide normobaric hypoxia are nitrogen houses, hypoxic tents, and special breathing apparatuses.

Noteworthy, recently it has been shown that these two types of hypoxia do not evoke identical physiological responses. Hypobaric hypoxia leads to greater hypoxemia (decrease of partial O$_2$ pressure in the blood), hypocapnia (less CO$_2$ partial pressure), blood alkalosis (increase pH), and a lower O$_2$ arterial saturation, compared to normobaric hypoxia. These physiological differences could be the consequence of an increase in pulmonary dead space ventilation, probably related to the barometric pressure reduction [47].

Acute and chronic effects of altitude/hypoxic exposure and training
Having the definition of altitude in mind the obvious problem the human body has to overcome when at altitude is the maintenance of an acceptable high scope for aerobic metabolism in the face of reduced oxygen availability in the atmosphere. In general, on acute exposure to hypoxia the human body reacts immediately with an integrated reaction of both the autonomic nervous system and cardiovascular system to overcome the drop in arterial oxygen content. Increased ventilation, sympathetic neural activity, cardiac output, and diuresis, among other mechanisms, constitute the acute response to hypoxia. Maximal values for heart rate and cardiac output are either similar to sea level values or slightly reduced. Maximal anaerobic capacity is reported to be unchanged.

If the hypoxic stimulus is maintained (i.e. chronic hypoxia) a complex multisystemic response is developed, leading to complete or partial acclimatization to hypoxia within a few days or weeks (figure 1). The most prominent adaptation that has been observed with continuous altitude exposure that has the clearest link to improved sea-level performance is an increase in red blood cell mass (RCM), which increases the oxygen-carrying capacity of the blood and improves aerobic power [29]. Although some studies in elite athletes have failed to show an increase in RCM with chronic
altitude exposure [2], the sum of experimental evidence in favor of this response is quite compelling. Several other adaptations to long term hypoxic exposure have been reported in literature.

Conflicting evidence exists for changes in anaerobic capacity with altitude acclimatization. Some studies have reported that buffer capacity of skeletal muscle may be increased [31], even with discontinuous altitude exposure [18], which may lead to improvements in anaerobic capacity, whereas other studies reported no change in anaerobic capacity after acclimatization [28]. Furthermore, it is suggested that hypoxic training could induce local adaptations at the molecule (augmented transcription for HIF1-alpha as well as increased mRNA for myoglobin and vascular endothelial growth factor) and muscle level (increased myoglobin and oxidative enzymes) that would be beneficial for performance [51].

Figure 1. Summary of the purported physiological mechanisms involved in the use of hypoxia for performance enhancement. Modified from Rodriguez 2007 [44].

The above mentioned adaptations and the degree in which they take place depend on several factors, some characterizing the “dose” of hypoxia (e.g. degree of hypoxia, duration of the exposure to hypoxia), some related to training (e.g. training goals, training program, normoxic or hypoxic training), and some related to nutrition and clinical status (e.g. iron stores, diet, oxidative stress, immune function). This disparity makes research on this topic particularly complex. Another important limitation is the fact that not all subjects respond the same to a certain combination of factors. In fact,
some authors have investigated the effects in “responders” and “nonresponders”, to underline the wide individual response to altitude training [8].

How high? Optimizing the level of altitude for altitude training
It is well-known that the erythropoietin (EPO) response to acute altitude is proportional to the degree of hypoxic stress, i.e. the higher the altitude, the more EPO is produced [12]. Therefore, in terms of the EPO response, the general rule for altitude training would be “the higher, the better”. However, living at higher altitudes increases the likelihood of suffering symptoms of acute mountain sickness (AMS): headaches, poor sleep, fatigue, nausea, vomiting, dizziness and loss of appetite, being characteristic symptoms. The incidence of symptoms of AMS increases dramatically when unacclimatized individuals ascend to altitudes above 3,000 m [21]. Moreover, highly trained individuals may even be more susceptible to the onset of AMS than moderately fit individuals [48]. In general, moderate AMS symptoms in swimmers may appear at or above 1,800 m, increasing in frequency and severity above 2,400 m (personal observations).

On the other hand, the study conducted by Ri Li et al. is of great interest [38]. They looked at the change in erythropoietin concentration ([EPO]) at four different altitudes, 1,780, 2,085, 2,454 and 2,800m, during the first 24 hours of exposure (figure 2). The report concluded that the altitude-induced increase in [EPO] is “dose” dependent, 2,100–2,500 m appearing to be a threshold for stimulating sustained EPO release in most subjects. Noteworthy, most altitude training sites for swimming are located at altitudes between 1,700 and 2,500m.

![Figure 2](image)

Figure 2. Individual values for percent change in EPO from baseline (after 6 and 24 h of exposure) in subjects at different simulated altitudes in a hypobaric chamber. Note marked individual variability that increases with increasing altitude. From Ri-Li et al, 2002 by permission [38].

How long? Optimizing the duration of altitude exposure
Although EPO concentration ([EPO]) increases within hours of exposure to a significant level of hypoxia, substantially more time is necessary to obtain an increase in red cell
mass sufficient to improve VO$_2$max and exercise performance. To obtain more insight in this area Levine et al. monitored [EPO] before, during and after a four-weeks altitude training camp (2,500m) [28]. Plasma [EPO] almost doubled after one night but returned to normal, prealtitude levels after three to four weeks of exposure. Moreover, at the end of the four weeks RCM was significantly increased demonstrating successful altitude acclimatization. These data indicate that in terms of an erythropoietic response at least three and preferably four weeks is the typically recommended duration for an altitude training camp at 2,500m. However it remains unknown whether this period of three to four weeks is optimal for other adaptations (for example, changes in muscle morphology or metabolism) as well.

Altitude/hypoxic training: current methods and strategies

Variations in the combination of exposure to hypoxia (degree and duration of hypoxia) and exercise (in normoxia or hypoxia), and therewith variations in the use of acclimatization and training effects, have led to the different altitude training strategies applied in sports practice today (see table 1).

Conventional altitude training consists in living and training in hypoxia (“living high, training high”, LH-TH). More recently, Levine and Stray-Gundersen introduced the strategy of living at moderate altitude (2,500 m) and training at a lower altitude (“living high-training low”, LH-TL). From this standpoint, hypoxic training can be described as a “living low, training high” (LL-TH) strategy. Finally, hypoxia can be achieved by living or staying in a hypoxic environment on a continuous basis or intermittently, i.e. combining periods of hypoxia and normoxia. Table 1 presents a summary of the hypoxic methods and strategies most commonly used in sports.

Table 1. Hypoxic methods commonly used in sports. Modified from Rodríguez (2002) [46].

<table>
<thead>
<tr>
<th>Method</th>
<th>Physical principle</th>
<th>Type of hypoxia</th>
<th>Facilities / Common strategies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate altitude</td>
<td>Natural reduction of atmospheric and O$_2$ pressure (↓PO$_2$)</td>
<td>Hypobaric, continuous or intermittent</td>
<td>Altitude resorts at the mountains (altitude training centers) Continuous or intermittent sojourns at altitude (LH-TH, LH-TL, LL-TH)</td>
</tr>
<tr>
<td>Hypobaric chamber</td>
<td>Artificial reduction of atmospheric and O$_2$ pressure (↓PO$_2$)</td>
<td>Hypobaric, intermittent</td>
<td>Hypobaric chambers (decompression or low pressure chambers) Intermittent exposure, passive (LH-TL) or combined with training exercise (LL-TH)</td>
</tr>
<tr>
<td>Hypoxic gas mixtures</td>
<td>Artificially decreased O$<em>2$ concentration in inspired air (↓F$</em>{I_O2}$)</td>
<td>Normobaric, intermittent</td>
<td>Hypoxic gas mixture (cylinders) Intermittent exposure, usually with training exercise (LL-TH)</td>
</tr>
<tr>
<td>Hypoxic houses, portable chambers and tents</td>
<td>Artificial ↓F$_{O2}$ by N$_2$ addition to atmospheric air</td>
<td>Normobaric, intermittent</td>
<td>Houses, portable chambers or tents with external N$_2$ addition to atmospheric air Intermittent exposure, usually during sleep or resting time (LH-TL)</td>
</tr>
<tr>
<td>Respiratory hypoxic devices</td>
<td>Artificial ↓F$_{O2}$ by O$_2$-filtering membranes in atmospheric air</td>
<td>Normobaric, intermittent</td>
<td>Portable respiratory devices (with breathing masks) producing a hypoxic gas mixture Intermittent exposure, usually combined with training exercise (LL-TH)</td>
</tr>
</tbody>
</table>

* L = live; T = train; H = high; L = low (e.g. “Live High-Train Low” = LH-TL)

Hypoxic training: “live low-train high” and controlled breathing

Combining the physiology of swimming, in most events being a high intensity sport requiring high rates of anaerobic metabolism, and the proposed effect of hypoxic
exercise training, enhanced anaerobic metabolism, Truijens et al. hypothesized that high intensity intermittent hypoxic training would improve sea level swimming performances more than equivalent training at sea level [55]. Sixteen well-trained collegiate and master swimmers were matched for gender, performance level and training history, and assigned to either hypoxic (simulated altitude of 2,500 m) or normoxic (sea-level) interval training in a randomized, double blind, placebo controlled design. All subjects completed a five week training program, consisting of three high intensity training sessions in a swimming flume and supplemental low to moderate intensity sessions in a pool each week. Although both groups of athletes improved performance (100 and 400 m freestyle) and VO\(\text{2}\)max, no differences between groups could be demonstrated. Moreover, neither swimming economy nor anaerobic capacity improved with this training. Interestingly, using a similar training regimen Ogita and Tabata found a 10% increase in anaerobic capacity, as measured by the accumulated oxygen deficit (AOD) after only two weeks of hypoxic training in nine competitive Japanese male swimmers [34]. However, no control group was included. Therefore, the question remains whether this improvement was an effect of the added hypoxic stimulus or solely an effect of the training itself.

The key issue in the interpretation of the results of hypoxic training studies seems to be the control of training intensity. In order to make an honest comparison between hypoxic and normoxic exercise training and draw conclusions regarding the occurrence of hypoxia specific effects, both groups should train at similar relative intensities. The study of Truijens et al. demonstrated that when both groups train at similar relative intensities the hypoxic group trained at significantly lower swimming speeds and thus lower power outputs compared to the normoxic controls [46]. At the metabolic level this was indicated by significantly lower VO\(\text{2}\) in the hypoxic (71.5%) compared to the normoxic group (91.8% of pre-test VO\(\text{2}\)max) (see figure 3). Thus, although hypoxic exercise may feel harder, the power output generated by the muscle is less, and the stimulus for muscle hypertrophy and myosin synthesis must be equivalently less. Moreover, although in the study of Truijens et al. both groups significantly improved their flume training speeds, this improvement tended to be smaller in the hypoxic group compared to the normoxic group [46]. This suggests that in the long run hypoxic exercise training might even lead to a relative state of detraining.
Controlled frequency breathing (CFB) is a frequently used training technique in which a swimmer voluntarily restricts breathing which, theoretically, limits oxygen availability and may stimulate anaerobic metabolism. In fact, breath-holding or reduced breathing frequency during exercise has been reported to result in alveolar hypoxia, increased arterial pressure of carbon dioxide (hypercapnia), substantial hypertension, and heart rate depression (bradycardia) [1] Very high intensity cycling exercise has also shown significant hypoxemia and arterial oxygen desaturation [30].

However, during tethered swimming training (4:4 min intervals at three different loads), estimated saturation of arterial blood with oxygen was found essentially undiminished, so that the major response to exercise was hypercapnia, rather than real hypoxemia [11]. Research has not confirmed either higher blood lactate accumulation or accelerated glycolytic metabolic activity as compared to high-intensity training [22]. In a recent study [56], during swimming graded exercise (3-min trials at 55, 65, 75, and 85% of peak intensity), CFB reduced ventilation, VO₂, and heart rate when compared to normal breathing, but it did not alter blood lactate concentration.

Hence, based on the available information it seems unlikely that breath-holding during exercise will enhance the effects of training or provide a physiological advantage that may enhance performance. On the other hand, the cardiovascular and neural responses elicited by breath-holding may be potentially hazardous to general health, as it may result in a decrease in cerebral circulation (with sudden loss of consciousness), acutely high blood pressure or arrhythmia.

Altitude training: “live high-train high” and “live high-train low”
The traditional and most common altitude training strategy among swimmers is LH-TH. The prevailing paradigm which supports this practice is that living and training at
moderate altitude (about 2,000-3,000 m) is directly linked to an accelerated production of red blood cells, which leads to an increase in VO$_2$max, ultimately resulting in improved endurance performance. However, it is important to note that controlled studies of LH-TH have not shown to improve sea level performance. This failure has been attributed to reduced training loads at altitude, particularly concerning intensity [29]. Only two studies have tested LH-TH for sea level performance in swimmers. In the only study with a sea-level control group, 10 male and female Korean elite swimmers lived and trained for 21 days at 1,890 m [9]. Although statistical analysis was not reported, the little increase in performance in 100- and 200-m races (0.1-0.7%) fell within inter-race variability for swimmers (~0.5%). In the second uncontrolled study male collegiate swimmers spent 14 days at 2,300 m [13], failing to show any significant changes in 200- and 500-yd swimming performance.

A more recent approach, first developed by Levine and Stray-Gundersen and known as "living high-training low" (LH-TL), has been shown to improve sea level performance in runners of different levels over events lasting 8-20 minutes [28, 49, 50]. In essence, LH-TL allows athletes to "live high" for the purpose of facilitating altitude acclimatization, while simultaneously allowing athletes to "train low" for the purpose of replicating sea-level training intensity and oxygen flux [28]. Analysis of 15 LH-TL studies has recently indicated that moderate altitude exposure of more than 12 h per day increases hemoglobin mass by about 1% per 100 hours of exposure [20]. This would imply a minimal duration of 21 days of intermittent exposure to attain about 5% an increase in hemoglobin.

Concerning the LH-TL strategy, proven useful in endurance trained athletes which performance time is over 8 minutes, it has never been successfully applied to swimming, in which most events last for less than 5 min. In fact, the only controlled study in which swimmers were tested, red cell production was effectively stimulated (+8.5%) after 13 days at 2,500-3,000 m, 16 h per day of simulated altitude, but neither VO$_2$max nor 2,000-m performance improved [40]. In another, uncontrolled study, even if total haemoglobin mass significant increases by 6% on average, the change in sea level performance after altitude training was not related to this hematological change [14].

Consequently, based on available research, altitude/hypoxic training has failed to prove useful for the enhancement of sea level performance in swimmers [57]. This fact is particularly remarkable bearing in mind that swimmers are among the most frequent users of high altitude training facilities.

**Intermittent hypoxia and sea-level training**

Another promising strategy is the use of intermittent hypoxia (IH), artificially achieved in a hypobaric or in a normobaric environment (nitrogen houses, hypoxic tents, or hypoxic breathing apparatuses) (see table 1). The practical reasons for IH being an alternative to conventional altitude training would be the availability of an artificially created environment located at low altitude areas, and the shorter hypoxic stimuli needed, which can be more compatible with normal living conditions and lower risk of acute mountain sickness in unacclimatized subjects.

IH combined with sea-level training should theoretically induce physiological adaptations without hampering training workload, thus allowing a comparison with the LH-TL paradigm. In a series of studies a short-term IH model with a higher degree of hypoxia (1.5 to 5 h at 4,000 to 5,500 m of simulated altitude) and shorter duration of the
chronic exposure (2-3 weeks) has been investigated. A significant increase in exercise time at sea level related to lower lactate accumulation during incremental exercise [7, 42], and improved ventilatory threshold [7, 45] in trained individuals have been reposted, with no significant changes in VO₂max.

Two IH studies have been performed with swimmers. In a first study [43], 8 swimmers of high national level combined sea-level training with exposure to IH over 2 weeks (3 h/d) at a simulated altitude of 4,000-5,500 m, and were compared to a control group following an identical training program. The IH swimmers significantly improved swimming performance in a 200-m trial (-1.3 s), associated with an increase in VO₂peak in the same distance (+9.3%), and VO₂max measured at a 400-m all-out test (+5.4%).

In a second, double blind, placebo controlled study (Rodriguez et al., 2007) 4 weeks of 4,000-5,500 m IH was administered to 23 subjects, 13 swimmers and 10 runners, distributed in two groups (IH, and controls). Swimmers performed duplicated 100 and 400 m time trials, and VO₂max tests on a swimming flume within three weeks before, and during the first and third week after the intervention. No significant changes in time trials or physiological markers of performance were observed for either the runners or swimmers. However, when runners and swimmers were considered separately, IH swimmers, and not controls, showed a significant increase in VO₂ at the ventilatory threshold (VT, +8.9) and maximal minute ventilation (+10.6%) immediately after the intervention, and also a significant increase in VO₂max relative to body mass (+7.5%) and VO₂ at VT (12.1%) two weeks after, following a pre-competition taper (figure 4).

Intriguingly, these changes could not be attributed to increased red blood cell or haemoglobin mass [19], submaximal swimming economy [54], nor to hypoxic and hypercapnic ventilatory control changes [53], neither of which changed in this experimental model. The hypothesis was raised that these changes could have been the combined effect of IH and tapering, thus suggesting that this strategy can be useful immediately before competition.
Figure 4. Changes in VO$_2$max relative to body weight (left panel) and VO$_2$ at ventilatory threshold following intermittent hypoxia and sea-level training (HYPO group: 3 h/d, 5 d/wk during 4 weeks at 4,000-5,500 m) or sea-level training controls (NORM group) in competitive swimmers (n=13). See text for details. Redrawn from Rodríguez et al. 2007 [44].

In a different approach, also short-term moderate normobaric IH has been investigated in athletic subjects. Some indications of erythropoietic stimulation were observed, such as increased serum EPO, 2,3 DPG and transferrin receptor concentration, and reticulocyte counts [25]. However, no significant changes in hematological parameters such as hemoglobin concentration or hematocrit were found, nor enhancement of performance indicators were reported. Altogether, the efficacy of various strategies of IH in swimmers deserves further investigation.

When to compete after returning from altitude?
The scientific literature on the optimal time of competition after altitude training is scarce. However, just as the process of acclimatization to altitude occurs as soon as exposure to altitude begins, the process of deacclimatization occurs immediately upon return to sea level. In the athlete population both acclimatization and deacclimatization always occur in combination with exercise training, and thus the optimal time to compete after an altitude training camp depends on the response of the individual athlete and the training design upon return to sea level. The increased oxygen carrying capacity of the blood and VO$_2$max that are observed as a result of altitude acclimatization will start to diminish upon return to sea level as red cell mass returns to its sea level equilibrium. However, these adaptations may also allow higher training work loads during the first few days at sea level [8]. This suggests that the overall advantage of altitude training for sea level performance, when achieved, can be maintained as long as the positive effects of the improved post altitude sea level training response cancel out the negative effects of deacclimatization. Interestingly, Levine et al. [28] observed no slowing of 5,000m running performance in the first four weeks after a four-week altitude training camp [28].

In contrast to relative small scientific evidence on altitude deacclimatization and sea level performance, there is a large amount of anecdotal evidence on this topic [10]. Overall, it is suggested that optimal performance can be reached as soon as a few days after cessation of altitude exposure, until about four weeks after return to sea level. Clearly, more research is necessary in this area.

Health and nutritional issues in altitude/hypoxic training
Hypoxia is a remarkable physical and psychological stressor. Despite the scarcity of studies focused on the health and nutritional aspects of altitude/hypoxic training, it is well established that the cardiac output and the blood flow to skeletal muscles diminish, thus reducing the capacity of tolerating high intensity training [27]. This condition may deteriorate the athlete’s training status and performance capacity, besides increasing the risk of overtraining.

As mentioned before, one of the most frequent negative effects of altitude/hypoxic training is the early appearance of symptoms of acute mountain sickness, even if generally transient and of low or moderate intensity [28, 36, 39]. These symptoms typically include headache, sleep disturbances [24], and appetite and weight loss [23]. Their appearance may require medication or even the disruption of altitude exposure in
some cases. Also mental disturbances may affect well-being and performance at altitude. In fact, exercising and performing at altitude can produce a stress response characterized by increased negative mood and relatively poor performance [26].

Other potential negative effects are depression of the immune system and increased risk for infections, particularly of the upper respiratory airways [15, 16, 52], and cellular damage due to increased oxidative stress induced by exercise in hypoxia [35]. Active prevention measures include the use of antioxidants and, particularly, an adequate prescription of training intensity.

Another importance issue is iron balance and utilization. Since altitude/hypoxic training will optimally accelerate red blood cell production, iron stores should be determined prior to exposure to hypoxia, and eventually iron supplements should be prescribed by a physician before, during, and/or after it [27, 33, 41].

Conclusions and practical applications

Based on the available scientific evidence the following general guidelines can be drawn:

- There is no objective evidence for the conductance of low-moderate, short duration (less than 3 weeks) altitude training camps (LH-TH) in order to improve sea level performance in highly trained swimmers. Using traditional altitude training (LH-TH), at least 2,100 to 2,500 m of altitude for at least 3 to 4 weeks appears necessary to acquire a robust acclimatization response (primarily red cell mass) with lower risk of altitude disturbances in the majority of athletes.

- The optimal altitude training strategy for improvements in sea level swimming performances is likely to be the “living high-training low” (LH-TL) strategy, in which one “lives high” (i.e. 2,100-2,500 m) to get the benefits of altitude acclimatization and “trains low” (1,250 m or less) to avoid the detrimental effects of hypoxic exercise. Whether the performance benefits would be equally large for swimmers compared to cyclists and runners remains questionable and requires further research.

- When using intermittent LH-TL at real or simulated altitude it is likely that fewer hours of hypoxic exposure (i.e. at least 12 h/day at 2,100–3,000 m maintained during 3 to 4 weeks) may suffice to achieve significant similar erythropoietic effects; training is to be carried out at a lower altitude (1,250 m or less). Short duration exposures to more severe hypoxia (e.g. 4,000 to 5,500 m, 3 hours/day for 2 to 4 weeks) combined with sea-level training may enhance VO2max, ventilatory threshold and middle-distance swimming performance (e.g. 200 m) when combined with a pre-competition taper, although the mechanisms for this improvement are unclear.

- Hypoxic exercise (as in LH-TH or LL-TH) does not appear to provide any physiologic advantage over normoxic exercise, regardless of training intensity. Moreover, absolute work load and oxygen flux are reduced suggesting that, if anything, hypoxic exercise might be detrimental effect to sea level performance.

- The optimal time to compete after an altitude training camp depends on the individual deacclimatization response and the training design upon return to sea level. The scarce evidence available suggests that the obtained performance advantage, if attained, can be maintained up to four weeks after the altitude training camp.

- There is substantial individual variability in the outcome of every hypoxic/altitude strategy. Moreover, since none of these strategies has undoubtedly proven to enhance
swimming performance, more research is warranted to further unravel and optimize the individual dose-response relationship.

- In any case, altitude/hypoxic training needs to be implemented under medical and nutritional supervision in order to minimize the potential negative effects on health, nutritional status, training status, and performance. Particular attention should be paid to carefully designed training programs, appropriate rest, and nutrition; iron and antioxidants supplements may be required but should be prescribed by a physician.

References


