Application of Altitude/Hypoxic Training by Elite Athletes

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ABSTRACT

WILBER, R. L. Application of Altitude/Hypoxic Training by Elite Athletes. *Med. Sci. Sports Exerc.*, Vol. 39, No. 9, pp. 1610–1624, 2007. At the Olympic level, differences in performance are typically less than 0.5%. This helps explain why many contemporary elite endurance athletes in summer and winter sport incorporate some form of altitude/hypoxic training within their year-round training plan, believing that it will provide the "competitive edge" to succeed at the Olympic level. The purpose of this paper is to describe the practical application of altitude/hypoxic training as used by elite athletes. Within the general framework of the paper, both anecdotal and scientific evidence will be presented relative to the efficacy of several contemporary altitude/hypoxic training models and devices currently used by Olympic-level athletes for the purpose of legally enhancing performance. These include the three primary altitude/hypoxic training models: 1) live high + train high (LH + TH), 2) live high + train low (LH + TL), and 3) live low + train high (LL + TH). The LH + TL model will be examined in detail and will include its various modifications: natural/terrestrial altitude, simulated altitude via nitrogen dilution or oxygen filtration, and hypobaric normoxia via supplemental oxygen. A somewhat opposite approach to LH + TL is the altitude/hypoxic training strategy of LL + TH, and data regarding its efficacy will be presented. Recently, several of these altitude/hypoxic training strategies and devices underwent critical review by the World Anti-Doping Agency (WADA) for the purpose of potentially banning them as illegal performance-enhancing substances/methods. This paper will conclude with an update on the most recent statement from WADA regarding the use of simulated altitude devices. **Key Words:** HYPOBARIC HYPOXIA, INTERMITTENT HYPOXIC TRAINING, LIVE HIGH–TRAIN LOW, NITROGEN DILUTION, NORMOBARIC HYPOXIA, SUPPLEMENTAL OXYGEN

Elite athletes have used altitude/hypoxic training for several years. Although the efficacy of altitude/hypoxic training relative to sea-level performance remains controversial from a research perspective, athletes continue to use it in preparation for elite level competition. Figure 1 outlines the different methods of altitude/hypoxic training currently used by elite athletes. The original method of altitude/hypoxic training was one in which athletes lived and trained at moderate altitude (1500–4000 m), for the purpose of increasing erythrocyte volume and ultimately enhancing sea-level maximal oxygen uptake ($\dot{V}O_{2max}$) and endurance performance. Live high + train high (LH + TH) altitude training is still used today by sea-level athletes who complete altitude training camps at specific times during the training year, and of course by altitude residents, such as the Kenyan and Ethiopian runners. It is not the purpose of this paper to review the extensive literature relative to LH + TH; however, the interested reader can access that information via comprehensive review articles (7,16,17,34,71).

One major conclusion drawn from both the anecdotal and scientific evidence regarding LH + TH altitude training was that endurance athletes did not seem able to train at an equivalent of near-equivalent training intensity (e.g., running velocity) as compared with sea-level training. Many runners and swimmers reported that they seemed to lose "race fitness/form" and "turnover" as a result of LH + TH altitude training. Indeed, in one of the original LH + TH altitude training studies conducted by Buskirk et al. (12), the results suggested that collegiate distance runners who completed 63 d of LH + TH (4000 m) returned to sea level in a *detrained* state, as evidenced by 3–8% decrements in time trial performance in the 880-yd, 1-mile, and 2-mile runs. More recently, it was demonstrated that absolute training intensity during base and interval workouts was significantly compromised at moderate altitude (2500 m) versus sea level in well-trained competitive distance runners (37,43).

LIVE HIGH + TRAIN LOW

As a potential solution to the training-intensity limitation that seems inherent in the LH + TH altitude training model, the live high + train low (LH + TL) model was developed in the early 1990s by Drs. Benjamin Levine and James Stray-Gundersen of the United States (34,36). Essentially, LH + TL is based on the premise that athletes can simultaneously experience the benefits of altitude/hypoxic

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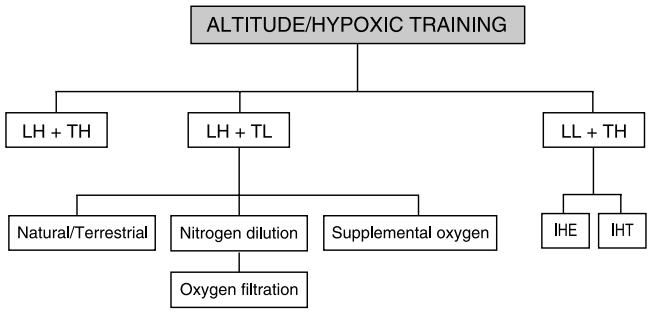

FIGURE 1—Contemporary altitude training models. IHE, intermittent hypoxic exposure; IHT, intermittent hypoxic training; LH + TH, live high + train high; LH + TL, live high + train low; LL + TH, live low + train high.

acclimatization (i.e., increased erythrocyte volume) *and* sea-level training (i.e., maintenance of sea-level training intensity and oxygen flux), thereby resulting in positive hematological, metabolic and neuromuscular adaptations. Athletes who use LH + TL live and/or sleep at moderate altitude (2000–3000 m) and simultaneously train at low elevation (< 1500 m). This can be accomplished using a number of methods and devices.

LH + TL via natural/terrestrial altitude. Initial implementation and scientific evaluation of the LH + TL model was conducted in the "natural/terrestrial" altitude environment of the Wasatch Mountains in the state of Utah, United States. The seminal research study by Levine and Stray-Gundersen (37) evaluated the efficacy of LH + TL among 39 American female and male collegiate distance runners who were initially matched according to fitness level and then randomly assigned to one of three experimental groups (LL + TL, LH + TL, and LH + TH). After a 4-wk baseline period at sea level (Dallas, TX), the LH + TL runners ($N = 13$) completed a 28-d training period in which they lived at 2500 m (Deer Valley, UT) for approximately 22 $h \cdot d^{-1}$ and trained at 1250 m (Salt Lake City, UT) for approximately 2 $h \cdot d^{-1}$. Training consisted of alternate workouts of base training and interval training. Thirteen fitness-matched female and male collegiate runners, serving as a control group (LL + TL), followed the same training program at sea level at 150 m (San Diego, CA), as did another group of 13 female and male runners who followed a conventional LH + TH regimen at 2500 m (Deer Valley). Compared with prealtitude values, postaltitude sea-level tests conducted on the third day after altitude training indicated significant improvements in the LH + TL group for erythrocyte volume (5%), hemoglobin concentration (9%), and treadmill $\dot{V}O_{2max}$ (4%). Similar changes in erythrocyte volume, hemoglobin concentration, and $\dot{V}O_{2max}$ were observed in the LH + TH runners, whereas no improvements in these parameters were seen in the sea-level control group. In terms of running performance, an average 1% improvement ($P < 0.05$) in postaltitude 5000-m run time was observed in the LH + TL group, an improvement that was equivalent to 13.4 s. Performance in the 5000-m run for the LH + TL runners was similar on days 7, 14, and 21 postaltitude compared with day 3 postaltitude, suggesting that the beneficial effects of LH + TL altitude training on running performance seem to last for up to 3 wk postaltitude. In contrast, neither the sea-level control group nor the conventional LH + TH group demonstrated any significant improvements in 5000-m run performance at any time after the 28-d altitude training period. Collectively, these results (37) suggest that living at moderate altitude (2500 m) resulted in significant increases in erythrocyte volume and hemoglobin concentration in both the LH + TH and LH + TL runners. However, simultaneous training at a lower elevation (1250 m) allowed the LH + TL athletes to achieve running velocities and oxygen flux similar to sea level, purportedly inducing beneficial metabolic and neuromuscular adaptations. When the runners returned to sea level, the LH + TL group was the only one that demonstrated significant improvements in both $\dot{V}O_{2max}$ and 5000-m run time. These results were attributed to positive hematological ("live high"), as well as metabolic and neuromuscular adaptations ("train low") resulting exclusively from 4 wk of LH + TL altitude training (37).

These initial findings by Levine and Stray-Gundersen (37) regarding LH + TL via natural/terrestrial altitude were subsequently supported in a similar study by Stray-Gundersen et al. (61) in elite athletes. American female and male national team distance runners demonstrated a significant 1% (5.8 s) prealtitude to postaltitude improvement in 3000-m time trial performance after 28 d of LH + TL altitude training in Deer Valley (2500 m) and Salt Lake City (1250 m), although this performance test was not referenced against a control group. More recently, Wehrlin et al. (69) evaluated the natural/terrestrial LH + TL model in conjunction with the training of Swiss national team orienteers. Compared with a fitness-matched control group, significant prealtitude versus postaltitude increments in erythrocyte volume (5%) and hemoglobin mass (5%) were reported in the LH + TL athletes, who completed a 24-d period during which they lived at 2500 m and trained at 1000 or 1800 m, depending on the goals of the specific training session. Although not referenced against a control group, significant prealtitude versus postaltitude improvements in treadmill $\dot{V}O_{2max}$ (4%) and 5000-m run time trial performance (2%) were also reported in the LH + TL orienteers (69).

An example of LH + TL via natural/terrestrial altitude training in elite sport is the U.S. national team in long-track speedskating, a group that initially used LH + TL in preparation for the 2002 Salt Lake City Winter Olympics. Three years before the Salt Lake City Olympics, the U.S. long-track speedskaters began living in the Deer Valley/Park City area at approximately 2500 m for the purpose of enhancing erythrocyte volume and to acclimatize at an elevation markedly higher than the altitude of their competition venue (1425 m) in the Salt Lake City area. The speedskaters used a modified LH + TL regimen in

which they performed moderate-intensity, dry-land training in Deer Valley/Park City (LH + TH *moderate* intensity) and completed high-intensity workouts in Salt Lake City (LH + TL *high* intensity). This LH + TH *moderate*-intensity + TL *high*-intensity model of altitude training had been previously evaluated by Stray-Gundersen et al. (61) and found to be as effective as the basic LH + TL strategy in bringing about significant increases in erythropoietic markers and $\dot{V}O_{2max}$, as well as improvements in 3000-m running performance in elite U.S. national team runners. During the year before the Salt Lake City Olympics, the speedskaters had access to the Olympic speedskating venue (Utah Olympic Oval; 1425 m), thereby gaining valuable experience and knowledge of the venue's ice conditions and aerodynamic characteristics. The U.S. long-track speedskaters enjoyed unprecedented success in the 2002 Salt Lake City Winter Olympics, with six athletes winning eight medals, including three gold medals and two world records (68). The U.S. national long-track speedskating team continued to use LH + TL via natural/terrestrial altitude in the quadrennium before the 2006 Torino Winter Olympics, during which time they established themselves as one of the best and most consistent teams in the world according to World Cup and World Championship performances. Similar to the 2002 Salt Lake City Olympics, U.S. long-track speedskaters performed very well in the 2006 Torino Olympics, capturing three gold, three silver, and one bronze medal.

LH + TL via natural/terrestrial altitude was also used effectively by U.S. national team marathon runners in preparation for the 2004 Athens Olympics. These athletes employed a LH + TH *moderate*-intensity + TL *high*-intensity model similar to the one used by the U.S. national long-track speedskaters. The marathon runners lived and completed their moderate intensity training at 2440 m (Mammoth Lakes, CA), whereas high-intensity workouts were done at 1260 m (Bishop, CA). The marathoners also employed heat/humidity preacclimatization strategies while living and training in the relatively moderate-temperature, low-humidity environment of the Sierra Nevada. These preacclimatization strategies served to prepare them very effectively for the harsh environmental conditions (30–35°C; 30–40% relative humidity) they eventually faced in Athens during the Olympics. U.S. Olympic team marathon runners enjoyed unprecedented success at the Athens Olympics, winning a bronze medal in the women's event and a silver medal in the men's race.

LH + TL via nitrogen dilution. *Nitrogen apartment/house* is a term used to describe a normobaric hypoxic apartment that simulates an altitude environment. The nitrogen apartment was developed by Dr. Heikki Rusko in Finland in the early 1990s for the purpose of simulating an altitude environment in relatively low-elevation Finland, thereby allowing Finnish elite athletes to LH + TL without having to travel abroad to do so. The nitrogen apartment simulates elevations equivalent to approximately 2000–3000 m via dilution of the oxygen concentration within the apartment. A ventilation system pulls in ambient air (~20.9% oxygen, ~79.0% nitrogen), and a gas composed of 100% nitrogen is simultaneously introduced into the ventilation system, resulting in an internal gas composition of approximately 15.3% oxygen and 84.7% nitrogen. This normobaric hypoxic environment simulates an altitude of approximately 2500 m.

Since the development of the nitrogen apartment by the Finns in the early 1990s, elite athletes in other Scandinavian countries, as well as Australian elite athletes have utilized nitrogen apartments in conjunction with LH + TL altitude training. Typically, these athletes live/sleep in the simulated altitude environment of the nitrogen apartment for $\geq$ 12 $h \cdot d^{-1}$ for $\geq$ 4 wk, and perform their training in natural/terrestrial sea level, or near-sea-level conditions.

Several studies have evaluated the efficacy of the nitrogen apartment on endurance athletes in Australia (2–6, 14,19,30,31,39,51,56,64), Finland (33,40,44,54,55), and Sweden (46). The details of these investigations can be reviewed in Table 1 and elsewhere (70,72). Within this group of studies, a more limited number were conducted on elite athletes from the Australian national team (3,39,56) and Finnish national team (44,54). The results of this limited number of studies on elite athletes have been equivocal. Whereas some researchers have reported significant increases in erythropoietic indices (54), others have not been able to replicate those results (3,56), or did not report erythropoietic data (39,44). However, several of these investigations on national team athletes reported significant improvements in sea-level performance after various "doses" of LH + TL via nitrogen dilution (39,44,56).

Thus, although limited, the empirical evidence suggests that LH + TL via nitrogen dilution may enhance sea-level performance in elite athletes, provided a sufficient dose of simulated altitude is applied, that is, $\geq$ 12 to 16 $h \cdot d^{-1}$ for $\geq$ 4 wk at an elevation of 2500–3000 m. It is not clear, however, whether the performance-enhancing effects of LH + TL via nitrogen dilution are attributable to accelerated erythropoiesis (54) or to beneficial changes in running economy (56), skeletal muscle buffering capacity (19), hypoxic ventilatory response (64), and/or skeletal muscle Na^+-K^+-ATPase activity (5,6).

LH + TL via oxygen filtration. Similar to the method of nitrogen dilution, a normobaric hypoxic environment can also be simulated via oxygen filtration. This method of LH + TL via oxygen filtration can take the form of an apartment/house, or a commercially available *hypoxic tent*. LH + TL via oxygen filtration uses an oxygen-filtration membrane that reduces the molecular concentration of oxygen in ambient air drawn from outside the apartment/tent. The oxygen-reduced air is pumped by generator into the apartment/tent, resulting in a normobaric hypoxic living and sleeping environment. There are several sites worldwide that employ LH + TL via oxygen filtration in conjunction with the training of elite athletes. These include the U.S. Olympic Training Center (Chula Vista, CA), Nike

Oregon Project (Portland, OR), Pettit National Ice Center (Milwaukee, WI), Japan Institute of Sports Science (Tokyo, Japan), Centre National de Ski Nordique (Premanon, Jura, France), English Institute of Sport (Twickenham, UK), New Zealand Academy of Sport (Auckland, New Zealand), Canadian Sport Centre (Calgary, Alberta, Canada), and Aspire Dome (Doha, Qatar).

The key research findings relative to the efficacy of LH + TL via oxygen filtration are found in Table 2, which is organized according to studies that have evaluated hypoxic apartments (top panel) and hypoxic tents (bottom panel). All of the hypoxic apartment investigations were conducted on elite endurance athletes from the French national team (athletics, biathlon, Nordic ski, swimming), whereas none of the hypoxic tent studies evaluated elite athletes. Collectively, the research findings regarding LH + TL via oxygen filtration are equivocal regarding erythropoietic effect, with two studies (10,49) reporting significant increases in erythrocyte volume and/or total hemoglobin mass, whereas others (24,25,41,50) found no significant erythropoietic response after LH + TL via oxygen filtration. In addition, the effect of LH + TL via oxygen filtration on performance is unclear. Significant postaltitude improvements have been reported in $\dot{V}O_{2max}$ (10), cycling peak power output (60), cycling power output at the respiratory compensation point (60), and 800- to 3000-m run time (24). In contrast, no significant enhancement of $\dot{V}O_{2max}$ (50,60), treadmill run time to exhaustion (50), or 2000-m swim time (49) have been demonstrated after LH + TL via oxygen filtration. Thus, although elite athletes continue to use LH + TL via oxygen filtration to enhance performance, it seems to be supported as much by anecdotal versus empirical evidence according to the current literature.

A final note regarding the potential negative effects of using LH + TL via oxygen filtration: Brugniaux et al. (11) recently evaluated the safety and efficacy of oxygen-filtration technology in elite athletes (5–6 d at 2500 m + 8–12 d at 3000–3500 m; $\geq 11\ h \cdot d^{-1}$). Although they report that cardiac function and symptoms of acute mountain sickness were not negatively affected at any elevation, immune status was compromised at 3500 m, as evidenced by a significant decrease in leukocyte count (11). Similar results have been demonstrated by Tiollier et al. (63), who report a significant depletion of secretory immunoglobulin A (sIgA) in French national team athletes living at a simulated altitude of 3500 m. These investigations (11,63) were taken into consideration by the World Anti-Doping Agency (WADA) in their recent evaluation of simulated altitude devices. On the basis of these studies, WADA concluded that there were potential negative health effects associated with the use of simulated altitude (http://altitudeforall.info/index.html). However, WADA's conclusion was subsequently challenged by the research group that conducted these investigations (11,63), in which they argued that their findings had been misinterpreted by WADA, and that there were minimal and physiologically insignificant health effects resulting from the use of simulated altitude via oxygen filtration (http://altitudeforall.info/index.html).

LH + TL via supplemental oxygen. Another modification of LH + TL altitude training is one in which athletes live in a natural, hypobaric hypoxic environment but train at simulated "sea level" with the aid of supplemental oxygen (LH + TLO_2). LH + TLO_2 is used effectively at the U.S. Olympic Training Center in Colorado Springs, CO, where U.S. national team athletes live at approximately 2000–3000 m in the foothills of the Rocky Mountain range. The average barometric pressure (P_B) in Colorado Springs is approximately 610 torr, which yields a partial pressure of inspired oxygen (P_IO_2) of approximately 128 torr. By inspiring a certified medical-grade gas with a fraction of inspired oxygen (F_IO_2) of approximately 0.26, athletes can complete high-intensity training sessions in a simulated sea-level environment at a P_IO_2 equivalent to approximately 159 torr. In addition to U.S. national team athletes at the U.S. Olympic Training Center in Colorado Springs, the previously mentioned U.S. long-track speedskating team uses LH + TLO_2 in conjunction with high-intensity training sessions done at the Utah Olympic Oval (1425 m) in Salt Lake City.

Only a few studies have evaluated the efficacy of LH + TLO_2 on athletic performance (13,42,73–75). Wilber et al. (73) evaluated the acute effects of supplemental oxygen on physiological responses and exercise performance during a high-intensity cycling interval workout (6 × 100 kJ; work:recovery ratio = 1:1.5) in trained endurance athletes who were altitude residents (1800–1900 m). Compared with a control trial (F_IO_2 0.21), average total time for the 100-kJ work interval was 5 and 8% ($P < 0.05$) faster in the F_IO_2 0.26 and F_IO_2 0.60 trials, respectively (Fig. 2A). Consistent with improvements in total time were increments in power output equivalent to 5% in the F_IO_2 0.26 trial and 9% in the F_IO_2 0.60 trial ($P < 0.05$) (Fig. 2B). Whole-body $\dot{V}O_2$ ($L \cdot min^{-1}$) was higher by 7 and 14% ($P < 0.05$) in the F_IO_2 0.26 and F_IO_2 0.60 trials, respectively, and was highly correlated with the improvement in power output ($r = 0.85$; $P < 0.05$). Arterial oxyhemoglobin saturation (S_pO_2) was significantly higher by 5% (F_IO_2 0.26) and 8% (F_IO_2 0.60) in the supplemental oxygen trials.

In a subsequent study, Wilber et al. (75) used near-infrared spectroscopy (NIRS) and report that hemoglobin/myoglobin (Hb/Mb)-deoxygenation of m. vastus lateralis was 8 and 12% less at blood lactate threshold and $\dot{V}O_{2max}$, respectively, during an F_IO_2 0.60 trial versus a control trial (F_IO_2 0.21) (Fig. 3), suggesting that supplemental oxygen enhances the availability of oxygen at the level of the capillary bed of the working skeletal muscle. Finally, Wilber et al. (74) report that there was no significant difference in cellular oxidative stress during exercise when comparing supplemental oxygen trials (F_IO_2 0.26, F_IO_2 0.60) with a control trial (F_IO_2 0.21), as determined by serum measurements of lipid hydroperoxides (LOOH) and

TABLE 1. Summary of current research findings relative to the use of live high + train low (LH + TL) via nitrogen dilution.

Author	Subjects	Design	Key Results
Aughey et al. (6)	Well-trained cyclists and triathletes	LH + TL continuous (N = 12) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 20 d Train at 600 m, normobaric normoxia LH + TL intermittent (N = 10) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} 4 x 5 d (2 d at 600 m, between each 5 d) Train at 600 m, normobaric normoxia Control (N = 11) Live and train at 600 m, normobaric normoxia	↓ Skeletal muscle maximal Na^+-K^+-ATPase activity in LH + TL "continuous" after 5 d and remained unaltered ($P < 0.05$) ↓ Skeletal muscle maximal Na^+-K^+-ATPase activity in LH + TL "intermittent" after 5 d but returned to baseline values after 4 x 5 d ($P < 0.05$)
Aughey et al. (5)	Well-trained cross-country skiers, cyclists, and triathletes	LH + TL (N = 6) Live at 3000 m, normobaric hypoxia 8–10 h·d^{-1} for 23 d Train at 600 m, normobaric normoxia Control (N = 7) Live and train at 600 m, normobaric normoxia	↓ Skeletal muscle maximal Na^+-K^+-ATPase activity ($P < 0.05$)
Kinsman et al. (30)	Trained endurance athletes	LH + TL continuous (N = 7) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 15 d Train at 600 m, normobaric normoxia LH + TL intermittent (N = 7) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} 3 x 5 d (2 d at 600 m, between each 5-d) Train at 600 m, normobaric normoxia Control (none reported)	↓ SpO_2 in LH + TL "continuous" and LH + TL "intermittent" vs pre ($P < 0.05$) ↑ Sleep arousal and RDI in LH + TL "continuous" and LH + TL "intermittent" vs pre ($P < 0.05$) ↑ REM sleep in LH + TL "continuous" and LH + TL "intermittent" vs pre ($P < 0.05$)
Kinsman et al. (31)	Well-trained cyclists	Crossover design (N = 10) LH + TL Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 1 d Train at 600 m, normobaric normoxia Control Live and train at 600 m, normobaric normoxia	HVR not correlated with sleep quality Stratified RDI may help identify potential sleep disturbances
Clark et al. (14)	Well-trained cyclists and triathletes	LH + TL continuous (N = 12) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 20 d Train at 600 m, normobaric normoxia LH + TL intermittent (N = 10) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} 4 x 5 d (2 d at 600 m, between each 5 d) Train at 600 m, normobaric normoxia Control (N = 11) Live and train at 600 m, normobaric normoxia	↓ Lactate production in high-intensity cycling in LH + TL "continuous" ($P < 0.05$) NSD skeletal muscle lactate metabolism LH + TL "continuous" and LH + TL "intermittent" NSD skeletal muscle buffer capacity LH + TL "continuous" and LH + TL "intermittent"
Saunders et al. (56)	Australian NT runners	LH + TL (N = 10) Live at 2000–3100 m, normobaric hypoxia 9–12 h·d^{-1} 4 x 5 d (2 d at 600 m, between each 5 d) Train at 600 m, normobaric normoxia Control (N = 13) Live and train at 600 m, normobaric normoxia	NSD Hb mass Improvement in running economy ($P < 0.05$)

Roberts et al. (51)	Well-trained cyclists	LH + TL (N = 19) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 5, 10, or 15 d Train at 600 m, normobaric normoxia Control (N = 19) Live and train at 600 m, normobaric normoxia	NSD $\dot{V}O_{2max}$ Improvement in $MMPO_{4\ min}$ ($P < 0.05$) Improvement in MAOD ($P < 0.05$)
Martin et al. (39)	Australian NT cyclists	LH + TL (N = 5) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 12 d Train at 600 m, normobaric normoxia Control (N = 5) Live and train at 600 m, normobaric normoxia	Improvement in $MMPO_{4\ min}$ ($P < 0.05$)
Townsend et al. (64)	Well-trained cyclists and triathletes	LH + TL continuous (N = 12) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 20 d Train at 600 m, normobaric normoxia LH + TL intermittent (N = 10) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} 4 x 5 d (2 d at 600 m, between each 5 d) Train at 600 m, normobaric normoxia Control (N = 11) Live and train at 600 m, normobaric normoxia	↑ HVR in LH + TL "continuous" and LH + TL "intermittent" ($P < 0.05$)
Gore et al. (19)	Well-trained cross-country skiers, cyclists, and triathletes	LH + TL (N = 6) Live at 3000 m, normobaric hypoxia 8–10 h·d^{-1} for 23 d Train at 600 m, normobaric normoxia Control (N = 7) Live and train at 600 m, normobaric normoxia	↑ Skeletal muscle buffer capacity ($P < 0.05$) ↓ Submaximal $\dot{V}O_2$ ($P < 0.05$) Improvement in cycling efficiency ($P < 0.05$)
Ashenden et al. (4)	Well-trained runners	LH + TL (N = 6) Live at 2650 m, normobaric hypoxia 8–11 h·d^{-1} 3 x 5 d (3 d at 600 m, between each 5 d) Train at 600 m, normobaric normoxia Control (N = 5) Live and train at 600 m, normobaric normoxia	↑ sEPO ($P < 0.05$) NSD reticulocyte parameters NSD Hb concentration
Nummela and Rusko (44)	Finnish NT 400-m runners	LH + TL (N = 8) Live at 2200 m, normobaric hypoxia 16–17 h·d^{-1} for 10 d Train at 10 m, normobaric normoxia Control (N = 10) Live and train at 10 m, normobaric normoxia	Improvement in 400-m-run TT ($P < 0.05$)
Ashenden et al. (2)	Well-trained cross-country skiers, cyclists, and triathletes	LH + TL (N = 6) Live at 3000 m, normobaric hypoxia 8–10 h·d^{-1} for 23 d Train at 600 m, normobaric normoxia Control (N = 7) Live and train at 600 m, normobaric normoxia	NSD reticulocyte parameters NSD Hb mass
Ashenden et al. (3)	Australian NT cyclists	LH + TL (N = 6) Live at 2650 m, normobaric hypoxia 8–10 h·d^{-1} for 12 d Train at 600 m, normobaric normoxia Control (N = 6) Live and train at 600 m, normobaric normoxia	NSD reticulocyte parameters NSD Hb mass

(Continued on next page)

TABLE 1. (Continued)

Author	Subjects	Design	Key Results
Rusko et al. (55)	Well-trained cross-country skiers and triathletes	LH + TL (N = 12) Live at 2500 m, normobaric hypoxia 12–16 h·d^{-1} for 25 d Train at 10 m, normobaric normoxia Control (N = 12) Live and train at 10 m, normobaric normoxia	↑ sEPO ($P < 0.05$) ↑ RBC mass ($P < 0.05$) ↑ $\dot{V}O_{2max}$ ($P < 0.05$)
Piehl-Aulin et al. (46)	Trained endurance athletes	LH + TL (N = 15) Live at 2000–2700 m, normobaric hypoxia 12 h·d^{-1} for 10 d Train at 10 m, normobaric normoxia Control (N = 5) Live and train at 10 m, normobaric normoxia	↑ sEPO ($P < 0.05$) ↑ Reticulocyte parameters ($P < 0.05$) NSD Hb mass NSD $\dot{V}O_{2max}$
Mattila and Rusko (40)	Well-trained cyclists	LH + TL (N = 5) Live at 3000 m, normobaric hypoxia 18 h·d^{-1} for 11 d Train at 10 m, normobaric normoxia Control (none reported)	↑ sEPO ($P < 0.05$) ↑ Reticulocyte parameters ($P < 0.05$) Improvement in 40-km cycle TT ($P < 0.05$)
Laitinen et al. (33)	Trained runners	LH + TL (N = 7) Live at 2500 m, normobaric hypoxia 16–18 h·d^{-1} for 20–28 d Train at 10 m, normobaric normoxia Control (N = 6) Live and train at 10 m, normobaric normoxia	↑ sEPO ($P < 0.05$) ↑ RBC mass ($P < 0.05$)
Rusko et al. (54)	Finnish NT cross-country skiers	LH + TL (N = 6) Live at 2500 m, normobaric hypoxia 14 h·d^{-1} for 11 d Train at 10 m, normobaric normoxia Control (none reported)	↑ sEPO ($P < 0.05$) ↑ Reticulocyte parameters ($P < 0.05$)

↑ increase; ↓ decrease; Hb, hemoglobin; HVR, hypoxic ventilatory response; MAOD, maximal accumulated oxygen deficit; $MMPO_{4\ min}$, maximal mean power output in 4 min; NSD, no significant difference ($P > 0.05$); NT, national team; RBC, red blood cell; RDI, respiratory disturbance index; REM, rapid eye movement; sEPO, serum erythropoietin; SpO_2, oxyhemoglobin saturation measured via pulse oximetry; TT, time trial; $\dot{V}O_{2max}$, maximal oxygen uptake.

TABLE 2. Summary of current research findings relative to the use of live/sleep high + train low (LH + TL) via oxygen filtration using either a hypoxic apartment (top panel) or hypoxic tent (bottom panel).

Author	Subjects	Design	Key Results
Brugniaux et al. (10)	French NT runners	LH + TL ($N = 5$) Live 14 $h \cdot d^{-1}$ at 2500 m (6 d) + 3000 m (12 d) Train at 1200 m Control ($N = 5$) Live and train at 1200 m	↑ sTfR ($P < 0.05$) ↑ Total Hb mass (mmol) ($P < 0.05$) NSD RBC volume (L) ↑ $\dot{V}O_{2max}$ ($P < 0.05$)
Brugniaux et al. (11)	French NT Nordic skiers, swimmers, and runners	LH + TL ($N = 21$) Live minimum 11 $h \cdot d^{-1}$ at 2500 m (5–6 d) + 3000–3500 m (8–12 d) Train at 1200 m Control ($N = 20$) Live and train at 1200 m	No AMS symptoms Cardiac function not affected HVR enhanced ($P < 0.05$) Immune status impaired ($P < 0.05$) at 3500 m
Robach et al. (49)	French NT swimmers	LH + TL ($N = 9$) Live 16 $h \cdot d^{-1}$ at 2500 m (5 d) + 3000 m (8 d) Train at 1200 m Control ($N = 9$) Live and train at 1200 m	↑ RBC volume (L) ($P < 0.05$) ↑ Total Hb mass (mmol) ($P < 0.05$) NSD 2000-m-swim TT
Robach et al. (50)	French NT Nordic skiers	LH + TL ($N = 6$) Live 11 $h \cdot d^{-1}$ at 2500 m (6 d) + 3000 m (6 d) + 3500 m (6 d) Train at 1200 m Control ($N = 5$) Live and train at 1200 m	NSD RBC volume (L) NSD total Hb mass (mmol) NSD $\dot{V}O_{2max}$ NSD TM time to exhaustion
Schmitt et al. (60)	French NT Nordic skiers, swimmers, and runners	LH + TL ($N = 20$) Live minimum 11 $h \cdot d^{-1}$ at 2500 m (5–6 d) + 3000–3500 m (8–12 d) Train at 1200 m Control ($N = 20$) Live and train at 1200 m	NSD $\dot{V}O_{2max}$ ↑ Cycling PPO ($P < 0.05$) ↑ Cycling power at RCP ($P < 0.05$)
Tiollier et al. (63)	French NT Nordic skiers	LH + TL ($N = 6$) Live 11 $h \cdot d^{-1}$ at 2500 m (6 d) + 3000 m (6 d) + 3500 m (6 d) Train at 1200 m Control ($N = 5$) Live and train at 1200 m	Compromised immune function as evidenced by ↓ sIgA ($P < 0.05$)
McLean et al. (41)	Healthy ♂	LH + TL ($N = 6$) Sleep 8 $h \cdot d^{-1}$ for 3 d at individualized elevations eliciting SpO_2 81 ± 2% Control ($N = 5$) Sleep 8 $h \cdot d^{-1}$ for 3 d in normobaric normoxic tent	↑ sEPO ($P < 0.05$) NSD RBC count, Hb concentration or Hct
Hinckson and Hopkins (24)	Runners and triathletes	Crossover design ($N = 11$) LH + TL Sleep 8 $h \cdot d^{-1}$ for 25 d at 2500–3000 m Control Sleep 8 $h \cdot d^{-1}$ for 25 d in normobaric normoxic tent	NSD Hb concentration or Hct 1–2% improvement in 800- to 3000-m runs
Pedlar et al. (45)	Recreational athletes	Double-blind, crossover design ($N = 11$) LH + TL Sleep 8 $h \cdot d^{-1}$ for 1 d at 2500 m Control Sleep 8 $h \cdot d^{-1}$ for 1 d in normobaric normoxic tent	↓ SpO_2 ($P < 0.05$) ↑ RDI ($P < 0.05$) ↓ BFW ($P < 0.05$)
Hinckson et al. (25)	Competitive runners	LH + TL ($N = 10$) Sleep 10 $h \cdot d^{-1}$ for 24 d at 2500–3000 m Control ($N = 10$) Sleep 8 $h \cdot d^{-1}$ for 24 d in normobaric normoxia in home environment	NSD Hb concentration NSD run HLa TH NSD run TTE

↑ increase; ↓ decrease; AMS, acute mountain sickness; BFW, behavior following waking; Hb, hemoglobin; Hct, hematocrit; HLa TH, blood lactate threshold; HVR, hypoxic ventilatory response; NSD, no significant difference ($P > 0.05$); NT, national team; PPO, peak power output; RBC, red blood cell; RCP, respiratory compensation point; RDI, respiratory disturbance index; sEPO, serum erythropoietin; sIgA, secretory immunoglobulin A; sTfR, serum transferrin receptor concentration; SpO_2, oxyhemoglobin saturation measured via pulse oximetry; TM, treadmill; TT, time trial; TTE, time to exhaustion; $\dot{V}O_{2max}$, maximal oxygen uptake.

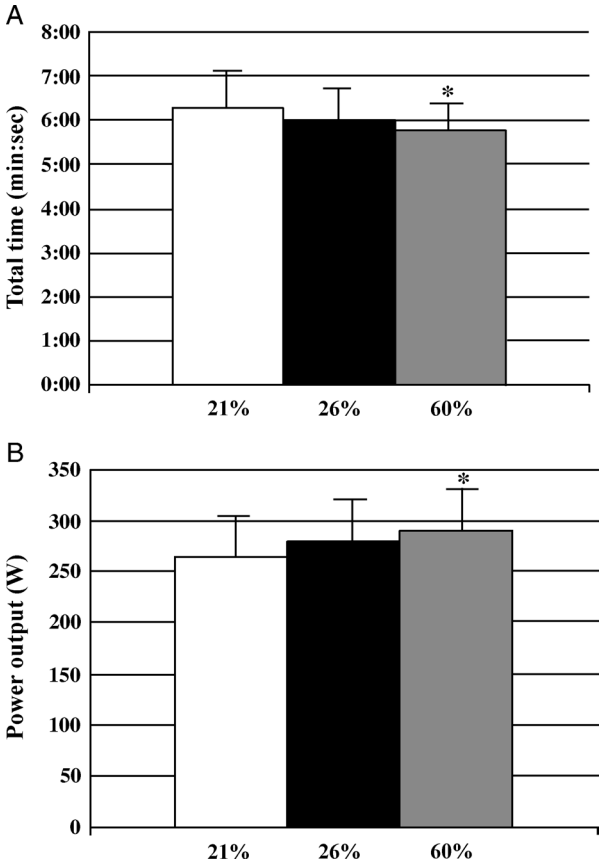

FIGURE 2—Total time (*A*) and power output (*B*) averaged over a 6 × 100-kJ bicycle ergometer interval session performed by trained cyclists at moderate altitude (1860 m) with F_IO_2 0.21 ($P_IO_2 \approx$ 128 torr), 0.26 ($P_IO_2 \approx$ 159 torr), and 0.60 ($P_IO_2 \approx$ 366 torr). Values are the group means ± standard deviations. * Significantly different vs F_IO_2 0.21 ($P <$ 0.05). Reprinted by permission from R.L. Wilber et al. (73).

reduced glutathione (GSH), as well as urinary measurements of malondialdehyde (MDA) and 8-hydroxy-deoxygenase (9-OHdG). On the basis of these results (73–75), it was concluded that LH + TLO_2 results in significant increases in arterial oxyhemoglobin saturation and greater unloading of oxygen at the level of the capillary bed of the working muscle, contributing to significant increases in power output and exercise performance, without inducing additional cellular oxidative stress. In terms of practical application, these results provide support for elite athletes to use LH + TLO_2 as an altitude training strategy that allows them to effectively live/sleep high and train low with minimal travel or inconvenience.

The long-term training effects of LH + TLO_2 were evaluated by Morris et al. (42). U.S. national team junior cyclists completed a 21-d training period during which they lived and performed their moderate-intensity workouts at 1860 m (Colorado Springs) and performed their high-intensity interval training at simulated sea level using supplemental oxygen (F_IO_2 0.26; P_IO_2 159 torr). Interval workouts were done 3 $d \cdot wk^{-1}$, and each interval workout required the athletes to complete 5 × 5-min cycling efforts at 105 to 110% of maximal steady-state heart rate. A control group of fitness-matched teammates completed the same training program at 1860 m using normoxic gas (F_IO_2 0.21; P_IO_2 128 torr). Athletes using supplemental oxygen were able to train at a significantly higher percentage of their altitude-determined lactate threshold (126%) versus their counterparts who trained in normoxic conditions (109%). After the 21-d training period, the athletes performed a 120-kJ cycling performance time trial in simulated sea-level conditions (F_IO_2 0.26; P_IO_2 159 torr). Results of the cycling performance test showed improvements of 2 s ($P > 0.05$ vs pretraining) and 15 s ($P < 0.05$ vs pretraining) for the normoxic-trained and LH + TLO_2-trained cyclists, respectively (42). In agreement with Wilber et al. (73), the results of Morris et al. (42) demonstrate that high-intensity workouts at moderate altitude (1860 m) are enhanced through the use of supplemental oxygen. Further, Morris et al. (42) were the first to show that sea-level endurance performance in elite athletes can be improved as a result of LH + TLO_2.

LIVE LOW + TRAIN HIGH

The live low + train high (LL + TH) model of altitude training is one in which athletes live in a natural, normobaric normoxic environment, and are exposed to discrete and relatively short intervals (5–180 min) of simulated normobaric hypoxia or hypobaric hypoxia. Normobaric hypoxia can be simulated via nitrogen dilution (e.g., Altitrainer 200 hypoxicator), oxygen filtration (e.g., Go2Altitude hypoxicator), or inspiration of hypoxic gas. LL + TH can be used by athletes in the resting state (intermittent hypoxic exposure; IHE) or during formal training sessions (intermittent hypoxic training; IHT). It is purported that IHE/IHT can enhance athletic performance by stimulating an increase in serum erythropoietin (sEPO) and erythrocyte volume (32,47,59), and can augment skeletal muscle mitochondrial density, capillary-to-fiber ratio, and fiber cross-sectional area (15,67) via upregulation of hypoxia-inducible factor 1α

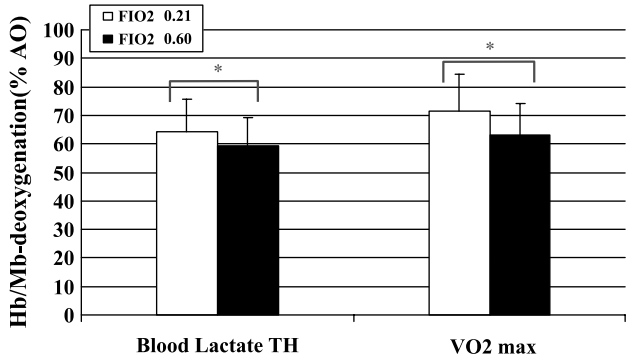

FIGURE 3—Hemoglobin (Hb)/myoglobin (Mb)-deoxygenation (% arterial occlusion (% AO)) at blood lactate threshold and maximal oxygen uptake ($\dot{V}O_{2max}$) during a progressive exercise test to exhaustion performed by trained cyclists and triathletes at moderate altitude (1860 m) with F_IO_2 0.21 ($P_IO_2 \approx$ 128 torr) and F_IO_2 0.60 ($P_IO_2 \approx$ 366 torr). Values are the group means ± standard deviations. * Significant difference between F_IO_2 0.21 and F_IO_2 0.60 ($P <$ 0.05). Reprinted by permission from R.L. Wilber et al. (75).

TABLE 3. Summary of current research findings relative to the use of live low + train high (LL + TH) using either intermittent hypoxic exposure (IHE) or intermittent hypoxic training (IHT).

Author	Subjects	Design	Key Results
Gore et al. (20)	Well-trained swimmers and runners	IHE (N = 11) ~4000–5500 m, hypobaric hypoxia 3 h·d^{-1} 5 d·wk^{-1} for 4 wk Control (N = 12) ~0–500 m, normobaric normoxia	↑ sEPO ($P < 0.05$) ↑ sTfR ($P < 0.05$) NSD RBC volume (mL) or Hb mass (g)
Bonetti et al. (9)	New Zealand NT sprint kayakers	Crossover design (N = 10) IHE F_IO_2 0.12–0.10, normobaric hypoxia 60 min·d^{-1} (5 min on + 5 min off) 5 d·wk^{-1} for 2 wk Control F_IO_2 0.21, normobaric normoxia	↑ Hb concentration ($P < 0.05$) ↑ Paddling PPO ($P < 0.05$) NSD $\dot{V}O_{2max}$ NSD 500-m paddling TT
Roels et al. (53)	Well-trained cyclists and triathletes	IHT (N = 11) ~3000 m, normobaric hypoxia 115 min·d^{-1} IHT cycle training session 2 d·wk^{-1} for 7 wk Control (N = 11) ~0 m, normobaric normoxia	NSD sEPO, RBC count, Hb concentration, or Hct NSD $\dot{V}O_{2max}$ NSD 10-min cycling TT
Abellan et al. (1)	Well-trained triathletes	IHE (N = 8) ~4000–5500 m, hypobaric hypoxia 3 h·d^{-1} 5 d·wk^{-1} for 4 wk Control (N = 8) ~0–500 m, normobaric normoxia	↑ sEPO ($P < 0.05$) ↑ sTfR ($P < 0.05$) NSD RBC volume (mL) or Hb concentration
Hamlin and Hellemans (22)	Trained endurance athletes	IHE (N = 12) F_IO_2 0.13–0.10, normobaric hypoxia 90 min·d^{-1} (5 min on + 5 min off) 5 d·wk^{-1} for 3 wk Control (N = 10) F_IO_2 0.21, normobaric normoxia	↑ Hb concentration and Hct ($P < 0.05$) Improvement in 3000-m-run TT ($P < 0.05$)
Katayama et al. (28)	Trained runners	IHE (N = 8) F_IO_2 0.12, normobaric hypoxia 3 h·d^{-1} for 14 d Control (N = 8) F_IO_2 0.21, normobaric normoxia	NSD sEPO NSD RBC count, Hb concentration, or Hct NSD $\dot{V}O_{2peak}$ Improvement in running economy ($P < 0.05$) Improvement in 3000-m-run TT ($P = 0.06$)
Rodriguez et al. (52)	Well-trained swimmers and runners	IHE (N = 11) ~4000–5500 m, hypobaric hypoxia 3 h·d^{-1} 5 d·wk^{-1} for 4 wk Control (N = 12) ~0–500 m, normobaric normoxia	NSD $\dot{V}O_{2max}$ NSD 100- and 400-m-swim TT NSD 3000-m-run TT
Glyde-Julian et al. (18)	U.S. NT runners	IHE (N = 7) ~5000 m, normobaric hypoxia 70 min·d^{-1} (5 min on + 5 min off) 5 d·wk^{-1} for 4 wk Control ~0 m, normobaric normoxia	NSD sEPO NSD RBC count, Hb concentration, or Hct NSD $\dot{V}O_{2max}$ NSD 3000-m-run TT
Hendriksen and Meeuwsen (23)	Well-trained triathletes	IHT (N = 16) ~2500 m, hypobaric hypoxia 2 h·d^{-1} IHT cycle training session 1 IHT session per day for 10 d Control (N = 8) ~0 m, normobaric normoxia	NSD Hb concentration NSD $\dot{V}O_{2max}$ Improvement in cycling PPO ($P < 0.05$) Improvement in anaerobic power ($P < 0.05$) and anaerobic capacity ($P < 0.05$) on a 30-s Wingate test

(Continued on next page)

TABLE 3. (Continued)

Author	Subjects	Design	Key Results
Katayama et al. (27)	Trained runners	IHE (N = 6) ~4500 m, hypobaric hypoxia 90 min·d^{-1} 3 d·wk^{-1} for 3 wk Control (N = 6) ~0 m, normobaric normoxia	NSD sEPO NSD RBC count, Hb concentration, or Hct NSD $\dot{V}O_{2max}$ Improvement in running economy ($P < 0.05$) Improvement in 3000-m-run TT ($P < 0.05$)
Truijens et al. (65)	Trained swimmers	IHT (N = 8) F_IO_2 0.15, normobaric hypoxia 12.5 min·d^{-1} IHT swim training session 3 d·wk^{-1} for 5 wk Control (N = 8) F_IO_2 0.21, normobaric normoxia	NSD Hb concentration NSD $\dot{V}O_{2max}$ NSD 100- and 400-m-swim TT
Ventura et al. (66)	Well-trained cyclists	IHT (N = 7) ~3200 m, hypobaric hypoxia 30 min·d^{-1} IHT cycle training session 3 d·wk^{-1} for 6 wk Control (N = 5) ~560 m, normobaric normoxia	NSD RBC count, Hb concentration, or Hct NSD $\dot{V}O_{2max}$ NSD cycling PPO NSD 10-min cycling TT
Karlsen et al. (26)	Norwegian NT junior cyclists	Crossover design (N = 8) IHT ~3000 m, normobaric hypoxia 2 h·d^{-1} IHT cycle training session 3 d·wk^{-1} for 3 wk Control ~0 m, normobaric normoxia	NSD sEPO NSD RBC count, Hb concentration, or Hct NSD $\dot{V}O_{2max}$ NSD 30-min cycling TT
Katayama et al. (29)	Trained runners	IHT (N = 7) ~4500 m, hypobaric hypoxia 30 min·d^{-1} IHT cycle training session 5 d·wk^{-1} for 2 wk Control (N = 7) ~0 m, normobaric normoxia	NSD $\dot{V}O_{2max}$ NSD HVR
Hahn et al. (21)	Australian NT rowers	IHT (N= 8) F_IO_2 0.15, normobaric hypoxia 60-min IHT rowing training session 3 IHT sessions per day for 19 d Control (N = 8) F_IO_2 0.21, normobaric normoxia	NSD Hb concentration NSD $\dot{V}O_{2max}$ NSD 2500-m rowing TT
Terrados et al. (62)	Professional cyclists	IHT (N = 4) ~2300 m, hypobaric hypoxia 1–2 h·d^{-1} IHT cycle training session 4–5 d·wk^{-1} for 3–4 wk Control (N = 4) ~0 m, normobaric normoxia	NSD Hb concentration NSD $\dot{V}O_{2max}$ NSD cycling TWC ($P < 0.05$) Improvement in cycling PPO ($P < 0.05$)

This table is limited to investigations that 1) examined the effects of IHE/IHT on trained athletes and 2) included a fitness-matched control group in the research design.
↑ increase; ↓ decrease; Hb, hemoglobin; Hct, hematocrit; HVR, hypoxic ventilatory response; NSD, no significant difference ($P > 0.05$); NT, national team; PPO, peak power output; RBC, red blood cell; sEPO, serum erythropoietin; sTfR, serum transferrin receptor concentration; TT, time trial; TWC, total work capacity; $\dot{V}O_{2max}$, maximal oxygen uptake.

(HIF-1α) (67). Because of its convenience, LL + TH via IHE/IHT is used by elite athletes in several countries.

The key research findings relative to the efficacy of IHE/IHT are found in Table 3. It should be noted that Table 3 is limited to studies that evaluated IHE/IHT in athletes only (recreational to elite), and included a fitness/training-matched control group in the research design. Collectively, the empirical evidence regarding the efficacy of IHE/IHT on erythropoietic response and athletic performance is not extremely compelling. Only a minimal number of well-designed, well-controlled studies on trained or elite athletes have reported increments in hemoglobin concentration (9,22), and to this author's knowledge none have evaluated or reported any increases in robust erythropoietic markers such as soluble transferrin receptor (sTfR), erythrocyte volume, and/or hemoglobin mass. Furthermore, no IHT study has demonstrated improvements in $\dot{V}O_{2max}$, and only 31% have reported that athletic performance was enhanced after IHT (9,23,27,28,62), possibly from improvements in efficiency/economy (27,28). In contrast, several studies have failed to demonstrate significant alterations in erythropoietic acceleration, $\dot{V}O_{2max}$ or post-IHT performance (1,18,20,21,26,29,52,53,62,65,66). One possible explanation for the preponderance of negative results in IHE/IHT studies may be related to the relatively short-duration hypoxic doses administered in the various protocols used (Table 3). It has been argued that for altitude/hypoxic acclimatization to be effective in accelerating erythropoiesis and ultimately enhancing performance, the hypoxic dose must be equivalent to an altitude of 2000–2500 m for $\geq$ 4 wk at a daily hypoxic exposure of $\geq$ 22 $h \cdot d^{-1}$ (34,38), as described in the paper presented in this symposium by Drs. Levine and Stray-Gundersen. That argument has been countered by those who contend that the mechanism by which IHE/IHT enhances performance is nonhematological, and may be due to beneficial changes in skeletal muscle mitochondrial density, capillary-to-fiber ratio, and fiber cross-sectional area (15,67), which have been demonstrated in untrained individuals. It is apparent that further research is needed in the area of LL + TH via IHE/IHT, particularly as it relates to elite athletes. Future investigations should focus on potential IHE/IHT-induced changes in these skeletal muscle parameters, along with continued evaluation of the more conventional measures of sEPO, erythrocyte mass, $\dot{V}O_{2max}$, and performance.

A final note regarding LL + TH via IHE/IHT relative to elite athletes: a number of studies have found IHE/IHT to be an effective method of preacclimatization before ascending to high altitude (> 4000 m) (8,48,57,58), and those findings are presented in detail by Dr. Muza in a separate presentation in this symposium. Although those studies were conducted on mountaineers and soldiers, the findings certainly have implications for elite athletes. It seems that IHE/IHT may be used effectively by elite athletes either before competition at altitude (e.g., Mexico City, 2300 m) or before undergoing an extended altitude training block.

SIMULATED ALTITUDE: LEGAL AND ETHICAL ISSUES

Recently, the use of simulated altitude by elite athletes has come under review by WADA. The rationale behind the WADA review is related to the fact that WADA officials are concerned that some athletes who are exploiting illegal erythropoietic agents are making use of "utilization of simulated altitude" as a false explanation for their abnormally elevated hemoglobin and hematocrit levels, thereby circumventing WADA's Prohibited Substance/Method List. WADA considers "artificially induced hypoxic conditions" to include hypobaric hypoxia (barometric pressure chamber), normobaric hypoxia via nitrogen dilution (nitrogen apartment; Altitrainer 200 hypoxicator), or normobaric hypoxia via oxygen filtration (hypoxic apartment/tent; Go2Altitude hypoxicator).

For a substance/method to be placed on WADA's prohibited list, it must meet two of the following three criteria (35):

1. Scientific evidence or experience demonstrates that the method or substance has the potential to enhance, or enhances sport performance.
2. Medical evidence or experience suggests that the use of the substance or method represents an actual or potential health risk to the athlete.
3. The use of the substance or method violates the spirit of sport.

The WADA scientific, medical and ethics committees have thoroughly evaluated the evidence regarding "artificially induced hypoxic conditions" and reached the following conclusions in May 2006 (35):

1. Artificially induced hypoxic conditions can significantly enhance performance when properly applied, by increasing the endogenous production of EPO with a subsequent elevation of red blood cell production and a better oxygen transfer to the muscles.
2. Under proper medical supervision, when reliable equipment was used, and when moderate altitude simulation was reproduced, no significant signs of health risk were reported.
3. After consultations with the WADA ethics review panel, it was concluded unanimously that artificially induced hypoxic conditions should be considered as violating the WADA spirit of sport criterion.

Collectively, these conclusions made by the WADA scientific, medical and ethics committees indicated that criteria 1 and 3 had been satisfied, and therefore "artificially induced hypoxic conditions" were to be considered for inclusion on the WADA prohibited list for 2007. In response to these initial conclusions, WADA conducted additional consultations throughout the summer of 2006 with its stakeholders, as well as scientific experts in the area of altitude/hypoxic training. The debate was amplified when several members of the international scientific

community responded collectively in opposition to WADA's consideration of banning simulated altitude devices (http://altitudeforall.info/index.html).

The final decision regarding artificially induced hypoxic conditions was made in September 2006 by the WADA executive committee and announced by WADA Chairman Richard Pound as follows:

"In response to our stakeholders who requested that there be full consideration of hypoxic conditions in the context of the prohibited list, WADA performed a scientific and ethical review of the matter, and engaged in a thorough consultation with experts and stakeholders. While we do not deem this method appropriate for inclusion on the list at this time, we still wish to express the concern that, in addition to the results varying individually from case to case, use of this method may pose health risks if not properly implemented and under medical supervision." (http://altitudeforall.info/index.html)

This statement indicated that WADA does not prohibit the use of "artificially induced hypoxic conditions" by elite athletes, at least through 2007. However, it should be noted that all "hypobaric/hypoxic practices are [currently] prohibited" in Italy, as mandated by the Italian Health Ministry in June 2005 (Decree of the Italian Ministry of Health 13.04.2005. Section 5, subsection M.1, June 3, 2005) in response to an incident involving professional cyclists competing in the 2005 Giro d'Italia (stage 10; May 18, 2005). The Italian law regarding simulated altitude is totally independent of any current and future WADA rulings, and presently has judicial precedence over any WADA rulings in areas of Italian jurisdiction. Finally, the International Olympic Committee has prohibited the use of simulated altitude devices within the boundaries of the Olympic Village since the 2000 Sydney Olympics, and this mandate is expected to apply to all future summer and winter Olympic Games.

SUMMARY

Many contemporary elite endurance athletes in summer and winter sport incorporate some form of altitude/hypoxic training within their year-round training plan, believing that it will provide the competitive edge to succeed at the Olympic level. This paper has presented both anecdotal and scientific evidence relative to the efficacy of several contemporary altitude/hypoxic training models and devices currently used by Olympic-level athletes for the purpose of legally enhancing performance. "Live high + train low" altitude training is employed by elite athletes using: 1) natural/terrestrial altitude, 2) normobaric hypoxia via nitrogen dilution (e.g., nitrogen apartment) or oxygen filtration (e.g., hypoxic tent), and 3) hypobaric normoxia via supplemental oxygen. Research regarding several of these LH + TL strategies is either limited or equivocal, particularly regarding optimal LH + TL hypoxic dose, as well as the physiological mechanisms that potentially impact post-altitude performance. Regarding the safety and health aspects of LH + TL, recent evidence suggests that living at a simulated altitude > 3500 m may have an impact on immunocompetence, but this effect may not have physiologically significant consequences.

A somewhat opposite approach to LH + TL is the altitude/hypoxic training strategy of *live low + train high*, in which athletes live in a natural, normobaric normoxic environment, and train for brief intervals using simulated normobaric hypoxia via nitrogen dilution (e.g., Altitrainer 200 hypoxicator), oxygen filtration (e.g., Go2Altitude hypoxicator) or hypobaric hypoxia (barometric pressure chamber). LL + TH is used by athletes in the resting state (IHE) or during formal training sessions (IHT). Collectively, the empirical evidence regarding the efficacy of LL + TH via IHE/IHT on erythropoietic response and endurance performance is not overly persuasive, and additional research is needed in this area, especially among elite athletes. The current literature does suggest, however, that IHE/IHT may be an effective preacclimatization strategy for elite athletes prior to training or competing at altitude.

Recently, several of these altitude/hypoxic training strategies and devices underwent critical review by WADA for the purpose of potentially banning them as an illegal performance-enhancing substance/method. Ultimately, WADA decided to refrain from including artificially induced hypoxic conditions on the 2007 prohibited list. However, it should be noted that use of all hypobaric/hypoxic practices was outlawed in Italy in June 2005, and this Italian law has judicial precedence within the boundaries of Italy over any WADA rulings regarding simulated altitude. In addition, the International Olympic Committee has prohibited the use of simulated altitude devices within the boundaries of the Olympic Village since the 2000 Sydney Olympics, and this mandate is expected to apply to all future summer and winter Olympic Games.

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