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ALTITUDE TRAINING IN ENDURANCE ATHLETES: PHYSIOLOGICAL ADAPTATIONS, TRAINING MODELS, AND CLINICAL RISKS

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ABSTRACT

Background and Objectives: Altitude training is widely used by endurance athletes to improve aerobic performance through haematological and non-haematological adaptations. Optimal training models and determinants of individual response remain debated. The aim of this review is to summarise current evidence on the physiological adaptations, training models, and clinical risks associated with altitude training in adult endurance athletes.
Methods: Literature from 2019 to 2025 was reviewed using PubMed/MEDLINE, Google Scholar, Scopus, and Web of Science. A total of 42 sources were included.
Key Findings: Altitude training at ≥2000 m for ≥3 weeks consistently increases haemoglobin mass and haemoglobin concentration, an effect replicated across multiple randomised trials [1, 6]. Improvements in VO_2max are variable and not always statistically significant [6]. Living at altitude while training at lower elevation has the strongest evidence base among available models [14]. Iron status critically moderates the response: athletes with low pre-camp ferritin show blunted adaptations regardless of altitude dose [7]. Sex differences in hypoxic physiology remain an important and understudied area [8, 19].
Conclusions: Altitude training, when properly implemented, is one of the more reliable tools available to endurance athletes. The data supports its use, but only if iron status is optimized beforehand, protocols are personalized to the individual, and clinical risks are managed carefully. More standardized studies are still required, especially in female athletes.

KEYWORDS

Altitude Training; Hypoxic Training; Haemoglobin Mass; Erythropoietin; VO_2max; Endurance Performance

CITATION

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1. Introduction

Altitude training has been used as a performance enhancement tool for over half a century. The 1968 Olympic Games in Mexico City, held at 2250 metres above sea level, sparked lasting interest in hypoxic training - athletes who had acclimatised to altitude performed noticeably better than their lowland counterparts in endurance events [1]. The live high–train low (LHTL) concept was created in 1997 after Levine and Stray-Gundersen showed that living at altitude while training at lower elevation gave better outcomes than the traditional method of doing both at height [1]. Since then, altitude training has become a central part of preparation for elite endurance athletes across disciplines including distance running, cycling, triathlon, cross-country skiing, and swimming [2].

The physiological basis of altitude training lies in the body's response to hypoxia - reduced oxygen availability at the tissue level. Above about 2000 m, the body senses the drop in available oxygen and begins responding at multiple levels - from hormones to red blood cells to the muscles themselves [3]. Reduced oxygen levels stabilize HIF-1α, a transcription factor that activates the EPO gene in renal cells, causing the kidneys to produce erythropoietin into the bloodstream. Elevated EPO then stimulates the bone marrow to produce red blood cells, increasing the oxygen-carrying capacity of the blood, which is a key determinant of aerobic performance [4]. Altitude training also produces non-haematological adaptations, including improved mitochondrial density, oxidative enzyme activity, exercise economy, and angiogenesis [3]. The training model used will most likely determine which of these mechanisms dominate.

Three models are most commonly applied in practice: live high–train high (LHTH), where athletes both reside and train at altitude; live high–train low (LHTL), where athletes live at 1800–3000 m but descend for high-intensity sessions; and intermittent hypoxic training (IHT), which involves repeated hypoxic exposures during exercise, typically in a normobaric chamber, without prolonged altitude residence [5]. Each model has a distinct physiological mechanism, logistical requirements, and risk profile.

Despite decades of research, several important questions remain. The magnitude of improvement in VO_2max is inconsistent across studies [6], and individual responses vary considerably depending on iron status [7], training history, sex [8], and genetic factors [3]. Clinical risks such as acute mountain sickness (AMS) and high-altitude pulmonary oedema (HAPE) are also rarely addressed in athlete preparation guidelines.

The aim of this review is to summarise current evidence on the physiological adaptations to altitude training in adult endurance athletes, with particular emphasis on haematological responses, training model efficacy, and individual determinants of response. Additionally, it examines potential problems - clinical risks that are frequently overlooked in training manuals and identifies areas where there is still little data. The aim is to provide practitioners with a practical, evidence-based reference.

2. Methodology

The literature search covered publications from 2019 to 2025 across PubMed/MEDLINE, Google Scholar, Scopus, and Web of Science. The search incorporated both free-text terms and Boolean combinations (AND, OR), including: “altitude training”, “hypoxic training”, “live high train low”, “LHTL”, “LHTH”, “intermittent hypoxic training”, “IHT”, “normobaric hypoxia”, “hypobaric hypoxia”, “haemoglobin mass”, “erythropoietin”, “VO_2max”, “iron status”, “endurance athletes”, “altitude acclimatisation”. Boolean operators were applied to refine and expand the search where appropriate. The search was restricted to peer-reviewed studies available in English. In addition, a manual screening of reference lists from included studies was performed to identify any relevant publications not captured through database searches.

2.1 Eligibility Criteria

To ensure relevance and scientific quality, the following inclusion criteria were applied:

- peer-reviewed original research articles or review papers published in English,
- studies evaluating altitude or hypoxic training in the context of endurance sport, haematological adaptation, or performance outcomes,
- research involving adult athletes (≥18 years) exposed to natural or simulated altitude ≥1800 m for ≥3 consecutive days.

Studies were excluded if they involved non-athlete clinical populations, exposures shorter than three days, or were purely case reports without quantitative physiological outcomes.

2.2 Study Selection and Data Extraction

All identified records were screened in a two-step process: titles and abstracts were first assessed for relevance, followed by a full-text review of potentially eligible articles. For each included study, the following data were extracted: study design, sample size, participant characteristics, training model, altitude and duration of exposure, primary outcomes, and key findings. Due to the methodological variability across studies, a narrative synthesis was performed rather than a meta-analysis. Extracted data were grouped thematically into four domains: haematological and physiological adaptations; comparative efficacy of training models; individual determinants of response; and clinical risks and adverse effects. A total of 42 studies and reviews met the eligibility criteria and were included in the analysis.

3. Results

3.1 Altitude Training Models

Altitude training, also referred to as hypoxic training, involves athletes training either at high altitudes or within artificially induced hypoxic environments. Different models combine different living and training altitudes, and the differences are relevant.

Live High, Train Low (LHTL) is currently the most widely used and best-supported approach [1, 5]. Athletes sleep and rest at altitude, typically between 1800 and 3000 metres, but travel to lower elevations for hard training sessions. The body adapts to the low-oxygen environment during rest, producing more red blood cells and improving oxygen transport, while training at lower altitude preserves the ability to work at high intensity. Among all hypoxic training variants, LHTL with low-altitude training has the highest probability of improving VO_2max [14]. A separate network meta-analysis comparing seven hypoxic training models identified intermittent hypoxic interval training (IHIT) as most effective for VO_2max in trained athletes specifically [15]. The altitude at which athletes train, not the altitude at which they sleep, is the primary determinant of efficacy - a conclusion supported by the superiority of LHTL over LHTH in network meta-analyses [14]. Effective LHTL implementation requires at least 12–14 hours of hypoxic exposure per day for

at least 3–4 weeks, accumulating more than 300 cumulative hours to produce significant haematological adaptation [1].

Live High, Train High (LHTH) is the older, classical model in which athletes both live and train at altitude. It reliably stimulates red blood cell production, but at the cost of reduced training quality: at altitude, maximum aerobic power is lower, forcing athletes to train at reduced absolute speeds and power outputs. This eventually minimizes the metabolic and neuromuscular changes brought about by high-intensity exercise [1]. LHTH is therefore most appropriate for base-building phases rather than competition preparation. Even so, a 21-day LHTH camp above 2000 m has been shown to produce a 4.9% increase in haemoglobin concentration alongside a 2.4% improvement in VO_2max in endurance runners [11].

Intermittent Hypoxic Training (IHT) offers a more logistically accessible option: athletes live at sea level and train in a normobaric hypoxic chamber a few times per week. Daily hypoxic exposure is too brief and infrequent to substantially increase haemoglobin mass, and current evidence does not support the superiority of aerobic IHT over normoxic training for VO_2max or endurance performance [31]. The exception is repeated sprint training in hypoxia (RSH), which consistently improves anaerobic capacity in addition to moderate aerobic gains through non-haematological pathways [15, 30, 31]. Among live-low, train-high approaches, RSH has shown consistent improvements in repeated-sprint capacity and anaerobic performance [30, 31]

Post-camp maintenance using combined intermittent hypoxic exposure (IHE) plus continuous hypoxic training (CHT) has emerged as an effective way to extend haematological benefits after a completed altitude camp. Athletes who combined IHE plus CHT every third day after returning from a 27-day camp maintained their elevated haemoglobin mass significantly longer than controls [17].

Table 1. provides a structured comparison of the four principal models.

Table 1. Overview of principal altitude and hypoxic training models.

Model	Athlete lives at	Athlete trains at	Key limitation
LHTL (natural)	1800–3000 m	< 1500 m / sea level	Travel cost, logistics
LHTL (simulated)	Altitude tent/room	Normoxia	Equipment cost, comfort
LHTH	1800–3000 m	Same altitude	Reduced training intensity
IHT / RSH	Sea level	Normobaric hypoxic chamber	Minimal haematological effect
IHE + CHT (post-camp)	Sea level	Normoxia ± hypoxia	Compliance, scheduling

3.2 Physiological Adaptations

3.2.1 Haematological Adaptations: Hb, EPO, Reticulocytes, Iron

At altitude, reduced oxygen availability stabilises HIF-1α - a protein that switches on several key genes in response to hypoxia, including the one that codes for erythropoietin (EPO). EPO is produced by oxygen-sensing cells in the kidney and released into the bloodstream, where it stimulates the bone marrow to produce more red blood cells. Serum EPO begins rising within 2 hours of arriving at altitude, peaks at 24–48 hours, and then gradually returns to baseline as haemoglobin concentration rises [3, 10]. Despite this EPO decline, red blood cell production continues for weeks because the precursor cells already committed to differentiation continue maturing [3].

Among the haematological outcomes of altitude training, total haemoglobin mass (Hbmass) is considered the most reliable marker of adaptation. Unlike haemoglobin concentration, which fluctuates with acute changes in plasma volume, Hbmass reflects the true amount of oxygen-carrying capacity available to the athlete [4]. Evidence from multiple randomised trials confirms that altitude training produces significant haematological gains, with pooled increases in both Hbmass and haemoglobin concentration [6]. The magnitude of adaptation is dose-dependent: Hbmass increases by approximately 1% per 100 hours of altitude exposure [1], and a 21-day camp above 2000 m has been shown to produce a 4.9% rise in haemoglobin concentration [11].

Reticulocytes, immature red blood cells that have just been discharged from the bone marrow, are the first detectable sign that erythropoiesis is active. Their count begins rising within 3–5 days of altitude arrival

and serves as a practical early indicator that the camp is producing the intended haematological stimulus [10]. After returning to sea level, Hbmass declines gradually rather than dropping off immediately. It often remains significantly elevated for at least 10 days after camp, owing to the strong erythropoiesis that occurs throughout the camp, which produces a group of young red blood cells, keeping the total cell population fresher and longer-lived than under normal circumstances [13]. This is why competition is generally best planned for 2–4 weeks after descent, when haematological adaptation is still near its peak.

Iron plays a central role in haemoglobin synthesis, and its availability ultimately determines how effectively the body can respond to altitude. Hypoxia partially compensates for increased iron demand by suppressing hepcidin - a hormone that normally limits iron absorption in the gut and restricts its release from storage sites. This process is further supported by erythroferrone (ERFE), which amplifies hepcidin suppression and helps channel more iron toward red blood cell production [9, 12]. The system works well when iron stores are adequate but breaks down when they are not. Athletes who arrive at altitude with low serum ferritin consistently show a weaker haematological response, no matter how long or how high they train [7]. This is not a minor concern: iron deficiency affects between 35 and 57% of elite endurance athletes, with women, vegetarians, and high-mileage runners facing the greatest risk [23].

3.2.2 Skeletal Muscle Adaptations

Beyond hematological alterations, altitude training causes skeletal muscle to undergo structural and metabolic changes that may last regardless of red cell mass and directly improve performance [3]. Hypoxia activates HIF-1α, which upregulates vascular endothelial growth factor (VEGF), which is a key driver of angiogenesis in skeletal muscle. After sustained altitude exposure, capillary density increases following altitude exposure, improving oxygen diffusion from red blood cells to working muscle fibres [3]. HIF-1α also promotes mitochondrial biogenesis by activating PGC-1α - a signalling protein that instructs muscle cells to produce more mitochondria, the organelles responsible for aerobic energy generation. A greater mitochondrial density allows the muscle to function harder for longer periods of time before becoming fatigued. Evidence from muscle biopsy studies backs this up: after altitude training, key enzymes involved in aerobic energy production - citrate synthase (CS) and succinate dehydrogenase (SDH), show measurably higher activity, pointing to a real improvement in how well the muscle can use oxygen [3, 26].

A study using four weeks of continuous hypoxic training (CHT, with oxygen levels reduced to approximately 15%) found that muscle oxygen saturation improved significantly across all exercise intensities compared to normal-oxygen training [35]. The muscles were also better at regulating blood flow - responding faster and more efficiently to the demands of exercise. These results suggest that hypoxic training improves performance via altering muscle structure and metabolism in addition to haematological adaptation.

3.2.3 Respiratory Adaptations

Altitude exposure also induces meaningful adaptations in the respiratory system, some of which continue long after the first phase of acclimatization.

The first and most immediate response to altitude is hyperventilation - an increase in breathing rate driven by chemoreceptors in the carotid bodies that detect falling arterial oxygen saturation. This reflex partly offsets the drop in alveolar oxygen pressure but also causes a reduction in arterial carbon dioxide, leading to respiratory alkalosis. The kidneys compensate over the following 24–48 hours by excreting bicarbonate, restoring blood pH at the cost of reduced buffering capacity [33].

With continued exposure, this ventilatory response itself adapts. The hypoxic ventilatory response (HVR) - the sensitivity of the breathing control system to falling oxygen levels - strengthens progressively with continued altitude exposure [3, 32]. This means the body becomes increasingly reactive to even small drops in oxygen, and this heightened sensitivity does not fully disappear after returning to sea level. In parallel, acclimatization can improve exercise economy and shift metabolic thresholds, allowing athletes to sustain higher intensities before lactic acid begins to accumulate - a shift driven by improved oxygen delivery and greater buffering capacity in the muscles [1, 33]. Female athletes tend to exhibit a stronger HVR than males, an effect likely modulated by estrogen and progesterone acting on peripheral chemoreceptors [8, 19].

3.2.4 Cardiovascular Adaptations

The cardiovascular system undergoes its own set of adaptations in response to altitude exposure. In the acute phase, cardiac output rises substantially, driven primarily by an increase in heart rate [32, 34]. As haematological adaptations develop over the following weeks, cardiac output normalises, and the system operates with greater efficiency. Early altitude exposure also triggers a diuresis that reduces plasma volume, temporarily concentrating the red cell fraction and elevating haematocrit. This haemoconcentration reflects dehydration rather than genuine erythropoiesis, which is why haemoglobin concentration alone is an unreliable marker of true haematological adaptation, and why Hbmass remains the preferred measure [4].

Some athletes experience a drop in blood oxygen saturation during maximal exercise even at sea level - a phenomenon known as exercise-induced arterial hypoxaemia (EIAH). At altitude, these athletes tend to struggle more than others, showing greater performance decrements for the same workload [27]. For this reason, measuring SpO_2 during a maximal effort test before a camp can help identify who may need a more cautious approach to altitude load. Despite decades of physiological research, official guidelines in competitive sport still offer only general acclimatisation advice rather than clear, sport-specific recommendations - leaving coaches and medical staff with considerable room for interpretation [41].

3.3 Performance Adaptations

3.3.1 VO_2max and Aerobic Capacity

VO_2max is the most widely used marker of aerobic fitness, yet it is also one of the most inconsistent outcomes in altitude training research. Haemoglobin mass increases reliably across studies, but VO_2max does not always follow - at least not in the pooled data [6]. Several factors help explain this discrepancy.

Elite athletes with very high VO_2max values are simply close to their biological ceiling - there is not much room left to improve, regardless of the training stimulus [6]. Studies also measure VO_2max using different equipment and protocols, making direct comparisons between them unreliable [1]. And many studies test athletes too soon after returning from altitude, before the haematological benefits have fully translated into performance gains - the optimal window tends to be 2–4 weeks post-descent [1].

When more performance-sensitive outcomes are used, such as time-trial results, critical power, or velocity at the lactate threshold, the evidence is considerably more consistent. In a controlled study, three weeks of simulated LHTL led to meaningful improvements in 3000-m time-trial performance and running economy in competitive runners - even though changes in VO_2max were modest [16].

3.3.2 Sport-Specific Considerations and Exposure Duration

The performance benefits of altitude training are most clearly seen in sports that place high demands on oxygen transport - distance running, road cycling, cross-country skiing, triathlon, swimming, and rowing [5]. The underlying physiology is broadly similar across these disciplines, but the way altitude training is structured and applied needs to be tailored to each sport.

When it comes to endurance sports, the evidence is consistent and well-established. Olympic and World Championship medallists across events from 800 m to marathon have consistently linked their performances to structured LHTL camps at 2000–2750 m [1], and meaningful haematological gains have been documented in elite swimmers [13], rowers [28], and cross-country skiers [21] following similar protocols. What differs between sports is the training content, recovery needs, and tolerance to altitude load - a swimmer's technical demands at altitude are very different from a marathon runner's.

Beyond traditional endurance sports, altitude training appears to offer benefits that go beyond haematology. Three weeks of strength training at altitude in elite judo athletes produced meaningful neuromuscular adaptations compared with sea-level controls [39] - a finding that suggests combat and power sport athletes may also have something to gain through mechanisms unrelated to red blood cells.

The question of how long to stay at altitude does not have a single answer. Elite endurance athletes generally need at least 3-4 weeks to generate meaningful haematological adaptation, with cumulative exposure exceeding 300 hours linked to greater gains in Hbmass [1]. Team-sport athletes, who tend to start with lower baseline haemoglobin mass, can see proportionally larger responses from shorter camps of 10–14 days [1]. For simulated altitude using tents or chambers, at least 12–14 hours of daily exposure are considered the minimum to sustain acclimatisation - shorter exposures do not provide a sufficient hypoxic stimulus [1, 34]. Competition performance tends to peak 3–5 weeks after returning to sea level, with a transient decline around week 2 as the body readjusts to normoxia [1].

3.4 Individual Variability in Response to Altitude Training

Not all athletes respond equally to altitude training. Even within groups following identical programmes at the same altitude, individual haemoglobin mass responses can range from no change to increases of more than 8% [20]. Understanding what drives this variability is essential for personalising altitude training.

Iron status is the most important and controllable factor. Athletes with low pre-camp serum ferritin show blunted haematological responses, independent of altitude dose, because without enough iron the body simply cannot produce new haemoglobin [7]. At least two to three weeks before the start of the camp, iron stores should be restored to adequate levels, if needed by supplementation [1]. Female athletes and long-distance runners need particular attention here: menstrual losses and foot-strike haemolysis both accelerate iron turnover [19].

Training level and baseline Hbmass create a ceiling effect. Athletes who already have high haemoglobin mass from years of endurance training have limited further capacity for improvement. Altitude training therefore tends to produce larger relative gains in developing athletes and team-sport players than in world-class endurance specialists who have already adapted extensively.

Individual EPO responses vary considerably. Some athletes mount a large EPO surge within the first 24 hours of altitude exposure (fast/high responders), while others show a delayed or blunted response (slow/low responders). Athletes are commonly categorised as high, medium, or low responders based on the magnitude of their Hbmass gain, with regular blood monitoring used to adjust the programme accordingly [1]. Some athletes who appear to be non-responders may instead be slow responders, achieving comparable Hbmass improvements when tested several weeks later [1]. Genetic factors also contribute - polymorphisms in genes encoding the EPO receptor and HIF pathway regulators influence how strongly an athlete responds [3].

The hormonal environment adds another layer of complexity. In professional cyclists, altitude training has been linked to significant individual differences in androgenic hormone profiles, including testosterone and cortisol dynamics, which may influence both adaptation and recovery capacity [40]. This dimension of altitude training is still poorly understood, but it likely matters for how athletes are loaded and recovered.

Year-to-year variability in the same athlete is also well documented. Non-physiological factors such as illness during camp, sleep quality, calorie availability, and training load all have a significant impact on how much an athlete benefits from a specific camp [20]. This is why individual monitoring during each block matters more than relying on how an athlete responded previously [1, 34].

Sex is an increasingly recognised source of variability. Women have lower absolute Hbmass, show different ventilatory responses to hypoxia, and may respond differently depending on menstrual cycle phase and hormonal contraception use [8]. Female athletes can achieve meaningful haematological and performance adaptations from normobaric hypoxic training [29]. In recreational women, intermittent hypoxic training has also been shown to improve functional fitness, with benefits persisting for up to four weeks after the intervention [25]. The optimal sex-specific protocols, however, remain undefined [19].

3.5 Clinical Risks and Adverse Effects

There are significant clinical risks associated with altitude training that are often overlooked in sports medical practice. The most common and clinically consequential is acute mountain sickness (AMS), characterized by headache combined with at least one of the following: gastrointestinal symptoms, fatigue, dizziness, or sleep disturbance [22].

The incidence of AMS increases with altitude: approximately 10–25% at 2000–3000 m, rising to 50–85% among those who ascend to 4500–5500 m [36]. AMS typically manifests 6–12 hours after ascent and resolves within 24–48 hours with acclimatization, however, if untreated, it can progress to the potentially fatal high-altitude cerebral oedema (HACE) or high-altitude pulmonary oedema (HAPE). A monitoring framework combining wearable sensors, physiological biomarkers, and individualised ascent protocols can reduce AMS incidence during altitude training camps [24].

HAPE represents a non-cardiogenic pulmonary oedema caused by increased pulmonary vascular permeability and excessive hypoxic pulmonary vasoconstriction. It is rare below 3000 m, but its incidence rises significantly with altitude [22]. High-altitude cerebral oedema (HACE) is a rarer but equally serious complication, characterised by cerebral swelling, ataxia, severe headache, and progressive alteration of mental status [42]. Both conditions represent medical emergencies: immediate descent is the primary intervention, with dexamethasone indicated for HACE and nifedipine for HAPE where available. Athletes with underlying cardiorespiratory conditions, anaemia, sleep apnoea, or a history of prior altitude illness are at elevated risk for both.

Immune suppression at altitude is a clinically underrecognized problem. Prolonged hypoxia combined with heavy training loads can reduce lymphocyte activity, impair natural killer cell function, and disrupt immunoglobulin levels - increasing susceptibility to upper respiratory tract infections (URTIs) that can shorten an altitude camp. Immune function abnormalities that occur during altitude training camps typically return to normal after the intervention is over [37]. This highlights the need for tracking immunological indicators throughout the camp rather than waiting for symptoms to appear.

Iron deficiency carries a double risk at altitude: it both limits the haematological response and impairs performance directly. Iron deficiency without anaemia reduces exercise performance by 3–4%, and supplementation at 100 mg of elemental iron per day can improve performance by 2–20% in deficient athletes [38]. Screening for iron deficiency before altitude exposure and initiating supplementation when serum ferritin is low is standard clinical practice [7, 38].

Energy deficiency is another underestimated concern. Hypoxia suppresses appetite through hormonal pathways, and when reduced hunger is combined with elevated metabolic demands, athletes may lose considerable lean mass and compromise their haematological adaptation [11]. Reducing training volume by up to 25% in the first week, alongside careful nutritional monitoring, helps manage this [1].

Finally, sleep disruption is near-universal in the first week at altitude. Periodic breathing during sleep - cycling between apnoea and hyperventilation due to low CO_2 levels - reduces sleep quality and leaves athletes fatigued in training. Sleep quality improves as acclimatisation progresses and can be tracked using heart rate variability and wellness questionnaires [33].

4. Discussion

4.1 Haematological vs. Non-Haematological Mechanisms

The primary explanation for why altitude training improves performance is haematological: more red blood cells mean greater oxygen delivery, which directly correlates to increased endurance capacity. This is well supported by the fact that haemoglobin mass is the most consistently improved marker across all training models [6]. The relationship is more nuanced, however. Even when Hbmass clearly increases, VO_2max does not consistently rise across trials [6], indicating that performance adaptations and haematological changes are not as closely related as previously thought.

Interestingly, the altitude at which athletes train appears to be a stronger predictor of performance improvement than the altitude at which they sleep [14]. This may help explain why LHTL, where training quality is preserved at lower elevation, tends to outperform models where athletes train at altitude. Continuous hypoxic training has also been shown to improve muscle microvascular function independently of any changes in red cell mass [35].

4.2 The Inconsistent VO_2max Response

The lack of a consistent increase in VO_2max does not prove that altitude training is ineffective. It mostly suggests that VO_2max is an inadequate metric for elite athletes who are already nearing their maximum potential [6]. Additionally, different labs use different testing protocols, which complicates pooled comparisons [1]. Many studies test athletes in the first week back at sea level, right before the benefits peak [1]. The evidence becomes considerably more consistent when more sensitive outcomes are used instead of VO_2max, such as running economy, pace at the lactate threshold, or time-trial performance. For example, competitive runners' 3000-meter time-trial performance and running economy improved after three weeks of simulated LHTL, despite very slight increases in VO_2max [16].

4.3 Iron: The Most Controllable Factor

Going into an altitude camp, one of the most adjustable variables is iron status. The process is clear: EPO is increased by altitude, erythropoiesis is increased by EPO, and erythropoiesis requires more iron than normal food absorption can reliably supply [12]. The overall haematological response is lowered when iron reserves are reduced. Regardless of altitude dose, athletes with serum ferritin levels below approximately 30 ng/mL are unlikely to see significant Hbmass gains [7]. More than half of elite female endurance athletes and more than one-third of male athletes suffer from iron deficiency, which highlights the importance of this issue [18]. Ferritin should be checked at least 2–3 weeks before departure, and athletes below 35 µg/L should begin supplementation before the camp starts [7].

4.4 Sex Differences: An Underexplored Area

Most altitude training research has been conducted on male athletes, limiting how confidently its findings can be applied to women. Several important sex differences have been identified: women have a stronger ventilatory response to hypoxia, lower absolute Hbmass, and may respond differently depending on menstrual cycle phase and hormonal contraception use [8, 19]. Female athletes can achieve real haematological and performance adaptations from normobaric hypoxic training [29], but the optimal sex-specific protocols remain undefined. Developing altitude training guidelines tailored to female athletes should be a research priority.

4.5 Practical Recommendations

Before attending any altitude camp, a full blood count, serum ferritin, and transferrin saturation should be checked. Athletes with ferritin below 100 µg/L should begin iron supplementation at least 2–3 weeks before departure. Athletes with a history of HAPE, significant cardiorespiratory disease, or sleep apnea must have an individual medical evaluation before attending any altitude camp [22].

During the camp, 3–4 weeks at 2000–2500 m using the LHTL model is recommended where possible. Training volume should be reduced by up to 25% in the first week [1]. Daily monitoring with heart rate variability, SpO_2, and wellness questionnaires allows early identification of problems [33, 34]. The final 2–3 days should be kept lighter, so athletes arrive at sea level feeling refreshed. Competition performance typically peaks 3–5 weeks post-descent [1]. For athletes who cannot compete in that window, post-camp IHE combined with CHT sessions every third day can extend haematological benefits by up to 30 days [17].

5. Conclusion

The evidence accumulated over recent years presents altitude training as one of the most consistently successful performance-enhancing strategies available to endurance athletes. When properly implemented, at the right altitude, for long enough, with good iron status and careful load management, it consistently increases haemoglobin mass, improves oxygen delivery, and produces faster race times across distance running, cycling, swimming, rowing, and other aerobic disciplines. The LHTL model has the strongest evidence base and remains the most practical choice for most athletes. Beyond haematology, altitude training also improves muscle capillary density, mitochondrial function, and respiratory efficiency.

Altitude training is not a simple intervention. It requires careful planning, medical screening, nutritional support, and ongoing monitoring throughout the camp. Iron deficiency, acute mountain sickness, immune suppression, energy deficiency, and sleep disruption are real and preventable risks. Athletes who are well-prepared and supported by a team capable of recognising and managing problems as they arise are likely to benefit the most.

Future research should prioritise standardised VO_2max testing protocols, greater representation of female athletes, and longer follow-up periods after camps. The genetic determinants of individual response, optimal post-camp maintenance protocols, and the interaction between altitude and other environmental stressors such as heat are all promising areas for investigation. As the evidence base grows, altitude training will become more individualised in its application, and better integrated into clinical and coaching practice.

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