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Altitude training: Exploring the benefits and risks for athletic performance and health

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ABSTRACT

The approach of altitude training has since become a popular method for improving athletic performance by taking advantage of the body's inherent response in a low-oxygen setting. These physiological adaptations, including increased red blood cell production, improved oxygen transport, and enhanced endurance, are due to exposure to hypoxia. Multiple altitude training protocols have been developed to maximize the beneficial effects of altitude, minimize possible performance impairments, and abate altitude acclimatization-related issues, including "Live High, Train Low" (LHTL). Outside of sports, altitude training holds medical potential. Studies indicate controlled hypoxia can bolster cardiovascular and respiratory health, promote neuroprotection, and stimulate metabolic function. But altitude exposure involves risks as well—such as acute mountain sickness and cardiovascular and cognitive strain—that require personalized protocols and greater vigilance. This review is focused on the physiological mechanisms involved in altitude training, its benefits for athletes and clinical populations, and the risks associated with hypoxia. By summarizing the existing research, we hope to inspire further optimization of training strategies and their applications within sports and medicine.

Keywords: Altitude training, intermittent hypoxia, endurance athletes, acute mountain sickness

1. INTRODUCTION

Altitude training has proved revolutionary for endurance athletes seeking to boost their performance. The body adapts by producing greater amounts of red blood cells, which facilitate better oxygen delivery and increase athletic performance-upgrading overall aerobic capacity by training or living at high altitudes, where oxygen levels are low. One of the most attractive strategies is called "Live High, Train Low" (LHTL), which allows athletes to take advantage of hypoxic adaptations while maintaining training intensity at sea level (Płoszczyca et al., 2018). However, outside of sports, altitude training is entering the medical dialogue. Studies suggest that regulated exposure to low oxygen levels may benefit people with cardiovascular, respiratory, and neurological conditions.

Low-dose intermittent hypoxia, for instance, has been associated with better cardiovascular health, improved lung function, and some cognitive benefits (Navarrete-Opazo and Mitchell, 2014). However, altitude training is not without its risks. Serious health risks include acute mountain sickness, high-altitude pulmonary edema, and cognitive impairments. That is why personalized training plans and close monitoring are important (Arnaud et al., 2023; Imray et al., 2010). This review explores the science of altitude training, including how it works, why it is valuable, and what risks should be weighed. Covering both athletic and medical applications, we seek to offer a fair review of how altitude training can be utilized for both performance and health benefits.

2. METHODOLOGY

This review analyzed literature from January 2015 to August 2024 on the benefits and risks of altitude training among athletes and patients. The methodology included Database Search: PubMed, Scopus, and Google Scholar were searched using keywords such as "altitude training", "intermittent hypoxia", "endurance athletes", and "acute mountain sickness" (Figure 1).

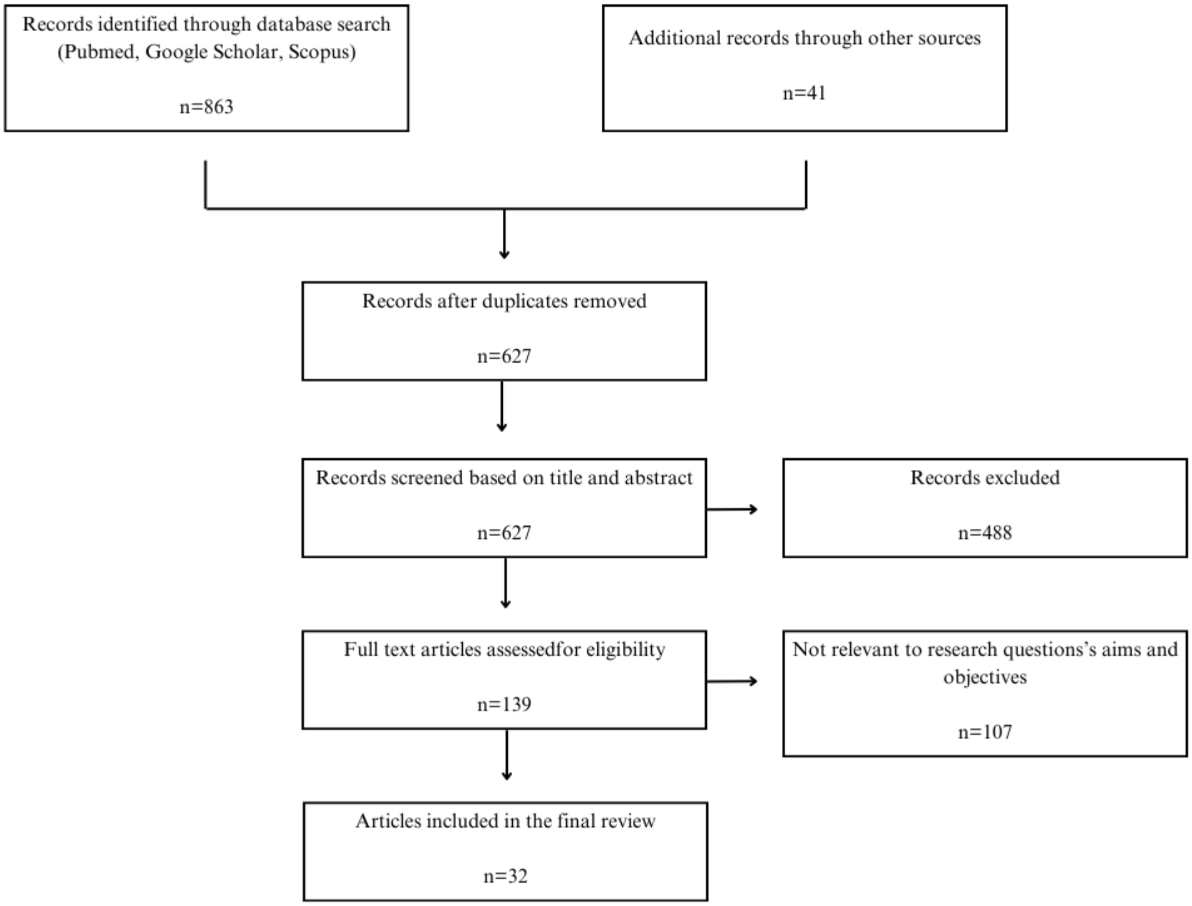

Figure 1 Consort chart

Inclusion Criteria: We included clinical research, systematic reviews, and meta-analyses published in English that focused on the benefits, risks, and physiological effects of altitude training and involved human participants.

Exclusion Criteria: We excluded studies with methodological weaknesses such as small sample sizes, lack of control groups, inadequate statistical analysis, and studies published in languages other than English.

All studies included in this review adhered to ethical guidelines, and relevant institutional review boards approved studies involving human participants.

3. RESULTS AND DISCUSSION

Physiology of altitude training

Hypoxic Conditions and the Human Body

Hypoxia is the lack of oxygen delivered to the human body's tissues, which triggers several physiological reactions. Such adaptations are attempts to preserve oxygen homeostasis. Nevertheless, chronic or extreme hypoxia can induce pathological states. Numerous factors are expressed in acute hypoxia (minutes to hours), stabilizing the hypoxia-inducible factors (HIFs). HIFs then trigger the production of hormones and messengers involved in the body's reaction to hypoxia, including vascular endothelial growth factor, erythropoietin, and glucose transporters (Richalet et al., 2024).

In many aerobic sports, altitude training has become a routine training regimen to improve sea-level exercise capacity or to acclimate before altitude events or altitude ascent. Athletes may perform better at sea level due to the many adaptations brought about by abrupt exposure to a hypoxic environment or prolonged elevation. These processes are typically linked to the effects of altitude training on the heart, lungs, or hematology. Altitude training, however, can also result in increased myoglobin concentration, capillary density, muscle mitochondrial volume, improved muscle buffering capacity, and higher glycolytic enzyme activity (Table 1) (Płoszczyca et al., 2018).

Firstly, hypoxia affects blood vessels in various parts of the human body. Low oxygen causes peripheral capillaries to dilate, whereas the pulmonary vasculature's vessels contract to divert blood from the area with inadequate ventilation, balancing ventilation, and perfusion. The rapid reaction known as hypoxic pulmonary vasoconstriction occurs in both pulmonary veins and arteries but is most pronounced in small resistance arteries. It starts when one or more K+ channels are inhibited, determining the membrane potential. It is inherent to the smooth muscle cells of the pulmonary vasculature. Voltage-gated Ca2+ channels are activated by the ensuing depolarization, increasing the cytosolic calcium level and causing myocyte constriction (Michiels, 2004).

While hypoxic pulmonary vasoconstriction raises pulmonary artery pressure, acute hypoxia triggers tachycardia and activates the adrenergic system. The autonomic nervous system adjusts, and tachycardia lessens after a few days of exposure to low oxygen concentrations, protecting the myocardium from excessive energy use (Richalet et al., 2024). Despite significant alterations in systemic hemodynamics, physiological research has demonstrated that mean arterial pressure barely changes during acute hypoxia (simulated altitude). This is because direct hypoxia vasodilation causes a decrease in peripheral vascular resistance, which contrasts with a marked increase in cardiac output, primarily caused by a sympathetic-mediated rise in heart rate (stroke volume stays essentially unchanged).

Thus, the overall impact on BP is almost zero. However, many studies that measured blood pressure through 24-hour ambulatory measurement showed a significant increase in blood pressure in response to hypoxia at high altitudes. The key mechanism of blood pressure rise is increased peripheral resistance. Evidence also suggests an impact of chemoreceptor-mediated sympathetic activation and an alteration in arterial baroreflex (Bilo et al., 2019). The ventilatory response, triggered by carotid body receptors, is linked to the renal reaction to hypoxia. Increasing breathing rate causes decreased alveolar CO2, hypocapnia, and respiratory alkalosis, inhibiting the central respiratory center (Khodaee et al., 2016).

Acute hypoxia has minimal effects on glomerular filtration rate and renal plasma flow. Urine flow rises, most likely due to adrenergic stimulation and RAAS inhibition. Hypocapnia and alkalosis, resulting from hypoxia-induced hyperventilation, profoundly affect renal physiology by generating a considerable rise in bicarbonate excretion. Hypoxia also increases cerebral blood flow. However, because hypoxia causes hyperventilation, the subsequent hypocapnia directly constricts blood vessels in the brain. Cerebral blood flow returns to normal sea-level levels after a few hours or days of exposure to hypoxia. Exercise stimulates various muscular vasodilatory activities through the production of NO and the blunting of sympathetic αadrenergic vasoconstriction.

This reaction counteracts the elevated sympathetic vasoconstrictor activity aimed at skeletal muscle during exercise in hypoxic environments. Additional hypothesized pathways include increased NO release via the exercising limb's β-adrenergic receptors, straight from the endothelium, or endothelial cells' activation by shear stress. Adenosine may also help control the blood flow in skeletal muscle by promoting the synthesis of prostaglandin and NO. The degree and length of hypoxic exposure, the intensity of the activity, and the recruited muscle mass may all affect these processes (Arnaud et al., 2023; Richalet et al., 2024; Shobatake et al., 2022).

Athletes at sea level may perform poorly due to immunosuppression, which has been linked to variations in serum immunoglobin levels, helper/suppression cell ratio, cell proliferation in response to mitogens, total leucocyte, granulocyte, monocyte, lymphocyte, natural killer cell, and T cell counts, and cell proliferation. Some researchers have reported physiological evidence for a less favorable modulation of immune function in vivo during acute and chronic exposure to hypobaric hypoxia. This could be explained by the stress of reducing the inspiratory oxygen concentration and the heavy training loads athletes use at altitude.

An increase in endogenous glucocorticosteroids and neuropeptide levels may cause this (Bailey and Davies, 1997). There is also evidence of intermittent hypoxia causing metabolic changes, such as increasing blood leptin concentrations, enhancing liver leptin expression, or increasing serum serotonin levels (Navarrete-Opazo and Mitchell, 2014). Hypoxia may also increase serum triglyceride levels. However, it is not well-documented in human studies (Morin et al., 2021).

Oxygen availability and the hypoxic environment

The low partial pressure of oxygen in the inspired air reduces exercise capacity at altitude, reducing muscle oxygen delivery in a manner directly proportional to the altitude at which effort is exerted. The following formula provides the oxygen pressure (PiO2) in the inspired air: The equation PiO2 = FiO2 × (Pb – PH2 O) is independent of altitude and currently equals 20.93%. FiO2 means the percentage of oxygen in the inspired air, Pb is the barometric pressure, and PH2 O is FiO2 (Bilo et al., 2019; Richalet et al., 2024).

Effects on hemoglobin concentration, erythropoiesis, and VO_2 max

Erythropoietin (EPO) release is one of the most well-documented physiological responses to a decreased partial pressure of oxygen, resulting in a temporary rise in the mass of red blood cells. Studies that artificially induced erythrocythaemia following either autologous blood reinfusion or subcutaneous injections of recombinant human erythropoietin have demonstrated the implications of secondary polycythemia to both submaximal and maximal indices of endurance performance. Iron deficiency may be risky for endurance athletes who exercise at altitude since hypoxia raises iron demand and mobilization. The absence of this essential erythropoietic component has been demonstrated to inhibit complete hematological adaptation (Bailey and Davies, 1997).

The EPO level dramatically rises during altitude training following the first two to three days/nights at altitude. After the peak, the level progressively declines, although it remains above the starting values for a few days to weeks. Following altitude training, there is also an increase in hemoglobin concentration, hematocrit value, red blood cell count, and reticulocyte percentage (Płoszczyca et al., 2018). The decrease in VO2max at altitude and VO2max at sea level are strongly correlated in the general population. This phenomenon can be explained at the lung level because there is a strong negative association between the drop in VO2 max and SaO2, which can be evaluated in either normoxia or hypoxia. As a result, those with the worst decline in VO2 max are probably the least able to sustain SaO2 (Chapman, 2013).

Mechanisms of Adaptation

At the cellular level, adaptation to hypoxia is achieved by reducing energy-consuming processes and, on the one hand, improving the efficiency of energy-producing pathways, primarily through increased anaerobic glycolysis activity (Michiels, 2004). Chronic exposure to hypoxia triggers adaptive reactions, including downregulating adrenergic receptors, altering the acid-base balance to increase the excretion of bicarbonates, stimulating erythropoiesis through erythropoietin, altering the secretion of different hormones, such as an increase in catecholamines and corticosteroids, and inhibiting the renin-angiotensin-aldosterone system.

Stabilized erythropoiesis typically aids in long-term adaptation to living in hypoxia after months, years, or lifetimes of exposure. However, in high-altitude residents, excessive erythropoiesis can result in pulmonary hypertension, heart failure, thrombosis, and increased blood viscosity in chronic mountain sickness (Marciante et al., 2021; Richalet et al., 2024). Altitude exposure stimulates erythropoiesis via hypoxia-inducible factor-1α (HIF-1α), increasing hemoglobin mass and oxygen transport capacity. This directly enhances VO_2 max and endurance performance.

Studies indicate repeated exposures (multi-year or seasonal) can yield cumulative gains in hemoglobin mass. Adaptations at the cellular and muscular level include enhanced muscle buffering capacity, aiding in lactate clearance and delaying fatigue, mitochondrial biogenesis, and improving energy production efficiency. These adaptations occur over shorter time frames than hematological changes and are less understood, requiring further research (Mujika et al., 2019; Ramos-Campo et al., 2015).

Methodologies in Altitude Training

Elite athletes often employ various training techniques, but the most popular one is the conventional extended exposure to artificial or natural altitude (Tomazin et al., 2021). Various methods of altitude training exist, such as live high-train high (LH-TH), live high-train low (LH-TL), and live low-train high (LL-TH). The LH-TH method involves athletes living and exercising at intermediate elevations (2,000–3,000 m) to promote erythropoiesis, which raises erythrocyte volume and improves sea-level endurance performance. Although this approach is still used today, a significant finding from scientific studies and athletic practice is that athletes cannot train at an intensity comparable to or near equal to that found at sea level.

In the LH-TL model of altitude training, athletes have intense training sessions at sea level. Then, they return to altitude at night to finish the acclimatization process. Under the LL-TH regimen, athletes train in a natural, hypobaric, or simulated normobaric hypoxic environment while living in normoxic conditions. The LL-TH approach can help activate non-hematological adaptive mechanisms. However, the exposure to acute hypoxia during the protocol is too brief and insufficient to alter hematological variables (Bonetti and Hopkins, 2009; Płoszczyca et al., 2018; Treff et al., 2022).

Training periodization

Athletes benefit most from structured altitude exposure over several years, integrating altitude camps into broader athletic development plans. Evidence suggests repeated exposures (e.g., 3-4 camps/year) maximize cumulative hematological and non-hematological adaptations. Studies have shown that athletes previously exposed to altitude adapt faster to subsequent exposures due to their physiological memory of hypoxia. Within a season, altitude training aligns with competition schedules to optimize performance peaks. The training schedule consists of two main phases.

The general preparation phase incorporates high-volume, low-intensity training at altitude to maximize hematological adaptations. The pre-competition phase combines altitude and sea-level training to maintain intensity and prepare for peak performance. Return from altitude is timed to align with competition, typically after 3–5 weeks, for optimal performance benefits (González-Ravé et al., 2023; Mujika et al., 2019).

Natural vs. Artificial Altitude Training

Combining terrestrial altitude training with normobaric hypoxia (e.g., altitude tents or chambers) can extend hypoxic exposure beyond the altitude camp period. Emerging evidence supports using normobaric hypoxia to pre-acclimatize before natural altitude or maintain benefits post-altitude. The most common way to induce artificial altitude conditions is a normobaric chamber with lowered oxygen concentration (normobaric hypoxia).

Despite variations in factors like ventilatory response, fluid balance, or the risk of acute altitude illness, training adaptations are often similar due to the similarity in acclimatization to natural and artificial hypoxia. A normobaric chamber is preferred in the LH-TL protocol as it is less difficult to implement. Artificial hypoxic protocols, especially brief intermittent LHTL, effectively improve sub-elite athletes' performance; however, studies have shown mixed results for elite athletes (Bonetti and Hopkins, 2009; Mujika et al., 2019; Treff et al., 2022).

Benefits of Altitude Training

Performance Enhancement

It is commonly known that performance in endurance exercises is closely related to the capacity to consume oxygen at a high rate. Prescreening SaO2 during intense or maximal exercise may assist in identifying individuals who would be more adversely affected at altitude than a typical response, according to data (Table 1) (Chapman, 2013).

Endurance benefits for athletes

Three types of stimuli cause endurance athletes to experience the training effect: mechanical, neuromuscular, and chemical. The muscles' ability to take in and use oxygen is more significant in endurance athletes than the cardiovascular system's ability to carry it. The primary goal of altitude training is to raise hemoglobin mass and red blood cell volume overall to strengthen the limiting link by raising arterial blood oxygen-carrying capacity. This will raise VO2 max and enhance performance at sea level and above.

In addition to energy availability and depletion, it has been demonstrated that neuromuscular system function affects endurance performance under normoxia and hypoxia. Both maximal cardiac output and muscular electrical activity are reduced during maximal exercise in hypoxia compared to normoxia and hyperoxia, indicating that the central nervous system may be involved in restricting exercise capacity (Flaherty et al., 2016; Rusko et al., 2004).

The "Live High, Train Low" method

Live High-Train Low protocol is recognized as one of the most effective hypoxic training methods. Studies have shown that it enhances endurance performance in both elite and sub-elite athletes. The combination of hypoxia exposure and training at low altitudes provides benefits of low-oxygen conditions while mitigating the performance limitations typically associated with training at high altitudes (Table 1). As with other protocols, LHTL promotes hematological adaptations, such as increased erythropoietin production, red blood cell count, and hemoglobin mass, which improve oxygen transport and aerobic capacity.

It is also beneficial to VO2 max, enabling athletes to sustain higher intensities of training sessions. Additionally, LHTL enhances muscle efficiency through mitochondrial adaptations. Those benefits come without the cost of lower training intensity. Well-organized high-altitude training camps provide an environment conducive to focused and intensive preparation, eliminating distractions, enhancing motivation among athletes, and contributing to performance improvements (Bonetti and Hopkins, 2009; Tomazin et al., 2021).

Medical and Non-Sporting Applications

The effects of IH are based on severity, frequency, duration, and cumulative time of hypoxia exposure. Outcomes of IH are dependent on those variables. Low-dose IH is based on exposure to mild hypoxia (oxygen levels between 9–16%) and fewer daily cycles, often around 3–15 episodes, each lasting from minutes to a few hours. Numerous studies have shown that this protocol confers benefits such as lower inflammation, improved immunity, and cardiovascular improvements, among many others. Firstly, low-dose IH increases nitric oxide production, enhancing endothelial function, leading to vasodilation, and decreased peripheral vascular resistance.

Due to their effects on blood pressure, these agents have been used for their beneficial actions in patients with hypertension. They provide protective myocardial effects against ischemia-reperfusion injury by reducing myocardial infarct size, decreasing oxidative stress, and enhancing coronary blood flow. It also improves physical performance in elderly patients. Low-dose IH has been shown to increase ventilatory responses, which is already beneficial in chronic obstructive pulmonary disease. It boosts forced vital capacity and forced expiratory volume, helping patients do things physically. It is additionally beneficial to patients with obstructive sleep apnea since it stimulates the respiratory motor output, stabilizes the breathing pattern, and augments the activity of the upper airway muscles.

Thirdly, low-dose IH can benefit spinal cord injury or amyotrophic lateral sclerosis, as it promotes neuroplasticity, enhancing synaptic connections and motor function recovery. Similarly to coronary heart disease, it also reduces the size of brain infarctions and mitigates post-stroke cognitive impairments, as it promotes angiogenesis and neuronal survival signaling. There is also evidence of anti-depressant effects of low-dose IH in both human and animal models. Last but not least, studies have shown that low-dose IH benefits the metabolism and the immune system. IH reduces body weight by increasing leptin levels, suppressing appetite, enhancing serotonin activity, and modulating food intake.

It also increases fatty acid oxidation and reduces cholesterol synthesis. In type 2 diabetes mellitus patients, IH increases insulin sensitivity, lowers blood glucose levels, and enhances glucose transporter-4 expression in muscle tissues. Low-dose IH stimulates osteoblast activity, promoting bone formation and increasing bone mineral density. IH enhances the phagocytic and bactericidal activity of neutrophils, improving immunity. Additionally, unlike high-dose IH, which can promote inflammation, low-dose IH suppresses pro-inflammatory mediators like tumor necrosis factor-alpha and interleukin-6 (Burtscher et al., 2021; Burtscher et al., 2024; Dudnik et al., 2018; Navarrete-Opazo and Mitchell, 2014; Richalet et al., 2024).

Risks and Dangers

Acute mountain sickness, high-altitude cerebral edema, or high-altitude pulmonary edema are among the clinical issues that can arise during altitude training. The degree of individual vulnerability, altitude, and climb pace affect how frequently and severely these issues are (Table 1) (Rusko et al., 2004).

Acute Mountain Sickness (AMS)

Acute Mountain Sickness (AMS) commonly occurs when individuals ascend to high altitudes too quickly. Sudden exposure to hypoxia often results in symptoms like headaches, insomnia, dizziness, nausea, fatigue, and loss of appetite. If untreated, AMS can progress to more severe and potentially fatal conditions, including high-altitude cerebral edema (HACE) and high-altitude pulmonary edema (HAPE). The underlying cause of AMS and HACE is the brain's response to hypoxia, where increased blood flow and capillary permeability lead to fluid accumulation and swelling.

These adjustments — induced by elevated sympathetic nervous system activity — lead to fluid retention and hormone imbalance. This biological adaptive phase, including adequate ventilation, increased red blood cell mass, and alteration of fluid handling, counteracts the adverse effects of hypoxia, the most famous of which is acute mountain sickness (AMS). Experts advise limiting ascents to 300 meters or less per day over 3,000 meters to mitigate some of the risk. Medications like acetazolamide, which enhances ventilation, and dexamethasone, which reduces brain swelling, can support acclimatization and symptom relief.

For severe AMS or HACE, immediate descent by at least 300 meters is crucial, along with oxygen therapy or the use of portable hyperbaric chambers. While most AMS cases resolve with rest and symptomatic care, understanding the molecular mechanisms—such as the role of hypoxia-inducible factors and cytokines—could pave the way for better preventive and therapeutic options. Proper preparation and early intervention make safe and enjoyable travel to high altitudes achievable (Bärtsch, 1999; Imray et al., 2010; Pichler et al., 2023; Turner et al., 2021).

Cardiovascular and Pulmonary Concerns

Traveling to regions of high altitude is not advised in many cases, such as less than 6 months after a cardiac event, electrocardiographic abnormalities were present during the stress test, heart failure (maximum advised altitude above sea level is dependent on NYHA class), uncontrolled or severe hypertension, pulmonary hypertension, serious ventricular arrhythmias, cyanotic heart disease or right-to-left shunt before surgical intervention (Richalet et al., 2024).

Other Risks

High-altitude conditions can tax the immune system, raising the risk of upper respiratory infections. Also, invalid training loads can prevent performance improvements. For athletes participating in high-intensity and/or technical sports, decreased oxygen availability and lower exertion levels can be detrimental. However, protocols such as "Live High, Train Low" help mitigate this risk by having high-intensity training sessions at lower altitudes. Key tools for tracking athlete adaptation include hemoglobin mass via carbon monoxide rebreathing tests, measurement of blood oxygen saturation, and perceptual wellness questionnaires to assess fatigue, sleep quality, and recovery (Mujika et al., 2019).

It has been proven in studies that hypoxia affects cognitive processes such as attention, memory, and executive functions, resulting in high-risk decisions that can harm health and safety (Pighin et al., 2022; Ramírez-delaCruz et al., 2024). Studies have also shown that hypoxia may induce cognitive functions, psychological fatigue, and mood changes. It is also related to heightened negative emotional responses and fatigue, suggesting a link between reduced oxygen levels and psychological fatigue (Stavrou et al., 2018). Hypoxia has also been associated with mood disorders. Damage to the brain caused by hypoxia, the researchers concluded, perhaps explains the depression, anxiety, and neuropsychological deficits. Patients with chronic obstructive pulmonary disease (COPD) with hypoxia and patients with heart disease, for example, are more likely to suffer from depression and anxiety (Zhao et al., 2017).

Protocols and Optimization

Adequate iron levels are critical for erythropoiesis. Athletes should ensure optimal iron stores pre-altitude and maintain supplementation during exposure. Studies show higher doses (100–200 mg/day) significantly enhance hemoglobin mass gains. Energy balance must also be maintained, as reduced appetite at altitude and increased metabolic demands can lead to weight loss and hinder adaptations (Mujika et al., 2019). Studies suggest that athletes should include plenty of antioxidant-rich foods in their daily diet while training at high altitudes to reduce the adverse effects of oxidative stress caused by hypoxia. Additionally, to optimize altitude training, it is recommended to decrease external training load and prioritize training volume rather than intensity (Treff et al., 2022).

Gaps in current research

Awareness of this decay timeline is important for planning subsequent exposures and competitions. Further research is warranted into mitochondrial, muscle buffering, and neural adaptations to hypoxia in elite populations (Mujika et al., 2019). However, honing those exact altitude thresholds for living and training is essential. To increase the generalizability, the duration of the LHTL protocol at its best and the effects of LHTL on other sports disciplines remain to be fully explored (Tomazin et al., 2021).

Future Directions

Altitude training can be combined with additional stressors, such as heat acclimation, or training modalities, such as sprint intervals in hypoxia, to potentiate the synergistic performance benefits. It is important to research the physiological capacity of athletes to perform under altitude conditions in various sports. Exploration of the long-term adaptation strategies and hypoxic memory phenomenon is still pending. Further studies are required to optimize altitude training protocols for maximal effectiveness, especially for high-level athletes looking for marginal gains (Bonetti and Hopkins, 2009; Treff et al., 2022).

Table 1 Summary of Benefits and Risks of Altitude Training

Key Point	Benefits	Risks
Athletic Performance	- Increased red blood cell production and oxygen transport - Improved VO_2 max and endurance - Enhanced muscle efficiency and buffering capacity	- Risk of acute mountain sickness (AMS) - Decreased training intensity at high altitude - Potential immune suppression due to training stress
Physiological Adaptations	- Higher hemoglobin concentration and erythropoiesis - Improved capillary density and mitochondrial function	- Increased cardiovascular strain and blood pressure - Risk of high-altitude pulmonary/cerebral edema (HAPE/HACE)
Medical Applications	- Cardiovascular benefits, including blood pressure regulation - Neuroprotection and cognitive benefits - Improved metabolic function and weight management	- Not suitable for individuals with cardiac or respiratory conditions - Possible cognitive impairments at extreme altitudes - Psychological effects (mood changes, fatigue)
Training Methodologies	- "Live High, Train Low" optimizes performance - Structured altitude exposure maximizes long-term gains	- Poor adaptation can hinder performance gains - Insufficient iron levels can impair erythropoiesis

4. CONCLUSION

In conclusion, altitude training is a great tool to improve athletic performance; there are better ways to utilize it in medicine. This stimulation can significantly improve athletes' endurance through hypoxia adaptations- the alveoli's surface area, hemoglobin mass, vo2 max - and working muscle efficiency. However, hypoxia exposure may also lead to adverse phenomena such as acute mountain sickness, as well as cardiovascular, neurological, or psychological disorders.

The conditions discussed above should be avoided strictly by utilizing an individualized training plan with intensive health monitoring. Passively acquired genetic adaptations and the complex relationships between iron metabolism, erythropoiesis, and systemic oxygen homeostasis are key to optimal altitude training regimens. More studies are needed to investigate the hypoxic components that are useful in revealing the findings in various sports and clinical indications.

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Conflict of interest

The authors declare that there is no conflict of interests.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

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