

A review of the Research on the Mechanism and Recovery Strategy of Exercise-Induced Fatigue in the Hypoxic Environment of the Plateau

Xiaolu Ji¹, Xiangyun Geng¹, Rongyun Fan^{2,*}

¹*College of Physical Education, Qinghai Normal University, Xining, Qinghai, China*

²*Qinghai Institute of Sports Science, Xining, China*

**Corresponding Author*

Abstract: Hypoxic exposure at high altitude represents a unique and extreme exercise environment. Reduced oxygen partial pressure profoundly alters energy metabolism, oxidative stress, and neuromuscular regulation, resulting in substantial differences in the onset, accumulation, and recovery of exercise-induced fatigue compared with exercise performed at sea level. Unlike conventional exercise-induced fatigue under normoxic conditions, high-altitude exercise-induced fatigue is characterized by the combined effects of metabolic peripheral fatigue and central nervous system fatigue, representing a major constraint on the effectiveness of altitude training and a critical factor influencing athletic performance and physiological recovery. This review systematically examines recent domestic and international research on exercise-induced fatigue under high-altitude hypoxia. the underlying mechanisms of fatigue are discussed from four perspectives: energy metabolism disturbances, oxidative stress and associated damage, impaired neuromuscular regulation, and immune dysfunction. Furthermore, various recovery strategies, including physical interventions, nutritional supplementation, training regulation, and psychological adjustment, are summarized. Key controversies and emerging directions in the current research are identified, and future research trends are discussed. This review aims to provide a theoretical basis and practical insights for the scientific management of training in high-altitude endurance sports, precision-oriented interventions for exercise-induced fatigue, and the promotion of health and physical performance in high-altitude environments.

Keywords: high altitude; hypoxia; exercise-

induced fatigue; energy metabolism; recovery strategies

1. Introduction

The air in the plateau area is thin and the oxygen partial pressure is low. After the human body enters the plateau environment above 2500 m above sea level, the body is in a state of continuous hypoxic stress, which is also a typical special environment for sports training and public health exercise. Altitude training, as the core training method of endurance events, can effectively improve the cardiopulmonary function, oxygen utilization efficiency and anaerobic metabolism ability of the body. It is widely used in the pre-competition preparation of endurance athletes such as race walking, middle and long distance running and bicycle. However, a large number of studies have confirmed that the dual superposition of hypoxia and exercise stimulation will break the balance of oxygen supply and demand in the body, accelerate the accumulation of metabolic waste, cell oxidative damage and nerve function attenuation, and induce rapid, severe and persistent exercise-induced fatigue. the recovery cycle of fatigue is much longer than that of plain exercise fatigue. Exercise-induced fatigue is a protective inhibition reaction of the body to avoid excessive loss of function. the fatigue in the plain environment is mostly dominated by single metabolic fatigue, with relatively single mechanism and clear recovery rules. In the high altitude hypoxia environment, acute hypoxia exposure, chronic altitude acclimatization, and different exercise loads can cause differentiated fatigue responses, accompanied by secondary problems such as immune disorders and autonomic nerve imbalance. At present, the research on plateau exercise fatigue mostly focuses on a single mechanism or a single intervention method, and lacks a systematic review of the fatigue

occurrence system and recovery strategy. Based on this, this paper integrates the frontier achievements of plateau exercise physiology, sports nutrition and rehabilitation science in recent years, systematically analyzes the mechanism of hypoxic exercise-induced fatigue at high altitude, and summarizes the standardized and personalized recovery system, so as to provide theoretical support for solving the problem of fatigue accumulation in plateau training and optimizing the health intervention program of plateau exercise.

2. The Core Mechanism of Exercise-Induced Fatigue under High Altitude Hypoxia Environment

2.1 Energy Metabolism Disorder: the Core Cause of Fatigue

The energy supply of body movement depends on aerobic oxidation and anaerobic glycolysis pathway. High altitude hypoxia will directly inhibit the function of mitochondrial oxidative respiratory chain and completely reshape the energy metabolism mode. Mitochondria are the core place for ATP production in the body. Oxidative phosphorylation is completed by the respiratory chain of mitochondrial inner membrane. the division of labor of multiple complexes catalyzes substrate dehydrogenation, electron transfer, and the coupling of oxidation and phosphorylation for energy supply. Any abnormality in the respiratory chain will break the balance of energy metabolism and induce exercise-induced fatigue. Acute exercise can down-regulate the activity of respiratory chain complex II in brain tissue, destroy the coupling efficiency of oxidative phosphorylation, block ATP synthesis, insufficient cell energy supply, imbalance of metabolic homeostasis, and drive exercise fatigue from the level of energy metabolism^[1].

At the same time, under the condition of high altitude hypoxia, the mitochondrial redox state shifted, the production of reactive oxygen species increased, and the respiratory chain complex and mitochondrial membrane structure were further damaged, forming a cascade amplification of hypoxia-mitochondrial function inhibition-ATP supply insufficiency-metabolic stress aggravation. the cascade causes the rapid consumption of ATP required for muscle fiber contraction and insufficient supplementation. the activity of functional proteins such as sarcoplasmic

reticulum calcium pump and sodium potassium pump is affected, the muscle excitation-contraction coupling efficiency is reduced, and the fatigue threshold appears in advance. Therefore, energy metabolism disorder is not only a concomitant phenomenon of altitude exercise, but also the core inducement and upstream driving link of exercise-induced fatigue. The transformation of this metabolic model directly leads to a large accumulation of lactic acid (LA), resulting in acid-base imbalance in muscle cells, inhibiting the activity of key enzymes in glycolysis, reducing the efficiency of myofilament gliding, and triggering skeletal muscle atony. At the same time, hypoxic stress will aggravate the catabolism of protein and fat, resulting in abnormal increase of muscle damage indexes such as blood urea nitrogen (BUN) and creatine kinase (CK), resulting in the accumulation of skeletal muscle micro-damage. Compared with the same exercise load in the plain, the body's metabolic waste removal rate after plateau exercise is slower, forming a vicious cycle of insufficient energy supply and accumulation of metabolic toxins, which is the fundamental mechanism for the rapid occurrence of peripheral exercise fatigue^[2].

2.2 Oxidative Stress Excessive Activation: Aggravate the Body's Cell Damage

Under normal physiological conditions, the body's oxidation and antioxidant systems are in dynamic balance. High altitude hypoxia combined with exercise stimulation will break this balance and induce a large amount of reactive oxygen species (ROS). Excessive ROS can attack skeletal muscle cell membrane, mitochondrial membrane and organelle structure, causing lipid peroxidation damage, resulting in increased cell membrane permeability and impaired cell function. Lipid peroxidation products malondialdehyde, protein carbonyl and DNA oxidative damage markers increased, further destroying mitochondrial membrane potential and calcium homeostasis, reducing muscle fiber contraction efficiency, thereby accelerating the fatigue process.

Studies have shown that the dual stimulation of high altitude hypoxia combined with exercise activates the sympathetic-adrenal medulla axis, cortisol and catecholamine are secreted in large quantities, disrupting the balance of oxidase and antioxidant enzymes, and excessive accumulation of ROS causes oxidative stress.

Excessive reactive oxygen species cause lipid and DNA damage in immune cells and inhibit immune function; at the same time, it destroys the normal metabolism of cells, aggravates cell damage, imbalance of internal environment homeostasis, induces immunosuppression, and eventually leads to early exercise-induced fatigue, which is an important oxidative stress pathway for fatigue in plateau environment^[3]. This pathway and energy metabolism disorders amplify each other: ROS damages the mitochondrial respiratory chain, and ATP synthesis is further limited; ATP deficiency weakens the ability of antioxidant defense and cell repair, so that oxidative damage continues to accumulate in skeletal muscle and immune cells, which constitutes an important mechanism for the occurrence and aggravation of exercise-induced fatigue at high altitude.

2.3 Neuromuscular Dysregulation: Central and Peripheral Fatigue Double Superposition

High altitude hypoxia can simultaneously induce central inhibition and peripheral muscle conduction abnormalities, forming compound fatigue. From the central level, hypoxia reduces cortical blood oxygen and disrupts neurotransmitter secretion, resulting in decreased cortical excitability and central protective inhibition; after superimposed exercise fatigue, the functional connectivity of α and γ bands in the primary motor cortex hemisphere is weakened, and the body can only maintain motor control by strengthening the interhemispheric β -band coupling and relying on bilateral cortical co-compensation^[4]. With the accumulation of fatigue, the functional connectivity of α and γ bands in the hemisphere decreased, and the efficiency of local cortical information integration decreased. Strengthening the interhemispheric β -band coupling becomes a key compensatory path to maintain motion control through bilateral cortical coordination. When the intensity or duration of exercise exceeds the compensatory threshold, the lack of central drive and the decrease of peripheral muscle strength are superimposed, and the performance of exercise decreases rapidly.

From the peripheral neuromuscular level, hypoxia will affect the release and transport of calcium ions in the sarcoplasmic reticulum, resulting in dysfunction of skeletal muscle excitation-contraction coupling, and a significant decrease in the maximum voluntary contraction

strength of the muscle. At the same time, the lack of KATP channels will lead to excessive membrane depolarization, increased calcium influx of L-type calcium channels, aggravated calcium transport disorders, and faster muscle strength attenuation^[5]; hypoxia can interfere with the release and transport of sarcoplasmic reticulum calcium, and aggravate the calcium-mediated systolic function damage. the neuromuscular junction transmission efficiency, motor unit synchronization and muscle fiber recruitment order are also affected by hypoxia, resulting in increased power output fluctuation and decreased anti-fatigue ability. Different from the characteristics of metabolic fatigue in the plain, plateau exercise fatigue is the dual result of central inhibition+peripheral function injury, and it is also the key neural mechanism for the sharp decline in plateau exercise performance.

2.4 Skeletal Muscle Inflammation Disorder: the Peripheral Mechanism of Exercise-induced Fatigue

High-intensity exercise stretches and tears muscle fibers, induces tissue damage and recruits neutrophils and macrophages to accumulate, releases a large number of pro-inflammatory factors such as TNF- α , IL-1 β , COX-2, activates the NF- κ B pathway, increases ROS, induces muscle cell apoptosis, aggravates muscle damage and inflammatory edema, and hinders muscle strength recovery^[6]. In the high altitude hypoxia environment, the decrease of oxygen partial pressure and the disorder of energy metabolism further amplify the inflammatory cascade: hypoxia can enhance the transcriptional activity of NF- κ B and promote the expression of pro-inflammatory factors; the increase of ROS activates NLRP3 inflammasome, promotes the mature release of IL-1 β , enhances the infiltration of neutrophils and M1 macrophages, and aggravates the damage of muscle fiber membrane and inflammatory edema. Inflammatory microenvironment also accelerates muscle protein degradation through ubiquitin-proteasome and autophagy-lysosomal pathways, inhibits mTOR signaling pathway, weakens muscle protein synthesis, hinders muscle strength recovery, and continues to accumulate peripheral fatigue.

At the same time, the synchronous compensatory release of myogenic IL-6 alleviates inflammation by inhibiting pro-inflammatory factors and secreting anti-inflammatory mediators; however,

if the injury continues, the pro-inflammatory factors are dominant, and the inflammation becomes chronic, which continues to aggravate the body's soreness and muscle strength decline, forming a vicious cycle of muscle injury-inflammation accumulation-exercise fatigue aggravation^[7]. At the same time, the synchronous compensatory release of myogenic IL-6 alleviates inflammation by inhibiting pro-inflammatory factors and secreting anti-inflammatory mediators; however, if the injury continues, the pro-inflammatory factors are dominant, and the inflammation becomes chronic, which continues to aggravate the body's soreness and muscle strength decline, forming a vicious cycle of muscle injury-inflammation accumulation-exercise fatigue aggravation. Therefore, skeletal muscle inflammation disorder bears the peripheral amplification mechanism in high altitude exercise-induced fatigue: acute inflammation initiates repair, and chronic inflammation promotes injury-fatigue cycle, which becomes an important peripheral pathway for the aggravation of exercise fatigue.

3. Diversified Recovery Strategies for Hypoxic Exercise-Induced Fatigue at High Altitude

Based on the complex mechanism of plateau exercise fatigue, it is difficult to achieve efficient repair by a single recovery method. At present, the academic community has formed a comprehensive recovery system of training regulation, nutritional intervention, physical rehabilitation and psychological adjustment.

3.1 Scientific Training Regulation: Reduce Fatigue Accumulation from the Source

A reasonable training program is the basis for preventing and alleviating plateau fatigue. the first is to implement gradient altitude adaptation to avoid rapid high altitude exposure. Through low altitude preconditioning and stepwise elevation of training altitude, the body's hypoxia tolerance can be activated and acute hypoxia fatigue damage can be reduced. Gradient adaptation can gradually improve ventilation efficiency, erythropoiesis, capillary density and mitochondrial oxidation ability, so that the body can establish metabolism and neuromuscular compensation before hypoxia exposure, and reduce the risk of acute altitude sickness and fatigue. the second is to optimize the training load ratio, follow the principle of 'low intensity, multiple groups, step by step', reduce the

proportion of high-intensity interval training, increase aerobic adaptive training, and reduce the accumulation of anaerobic metabolic waste. By controlling the intensity, capacity and interval, the aerobic oxidation energy supply is maintained, the lactic acid accumulation and muscle injury are reduced, the effective training time is prolonged, and the training stimulation and recovery ability are kept in dynamic balance.

At the same time, the classic altitude training modes such as 'living high and training low, living low and training high' are used to balance hypoxia adaptation and exercise recovery, and avoid fatigue overload caused by continuous high-intensity hypoxia stimulation. High living and low training use resting hypoxia exposure to promote adaptation, low training to maintain plain training intensity and recovery quality; low living and high training improves special adaptation by training low oxygen stimulation, and living in plains ensures recovery. the reasonable combination of the two can achieve the optimal balance between hypoxic adaptation and exercise performance, so that the body can maintain the training quality and recovery efficiency while obtaining hypoxic adaptation. In addition, the training load can be dynamically adjusted through real-time physiological monitoring such as heart rate variability (HRV) and blood oxygen saturation (SpO₂) to achieve early warning and accurate regulation of fatigue^[8].

3.2 Targeted Nutrition Intervention: Repair Metabolism and Oxidative Damage

Nutritional supplementation is the core means of rapid recovery of plateau fatigue, which has the advantages of strong targeting and quick effect. First, antioxidant nutrition supplement, by supplementing polyphenols, vitamins, and peptides, enhances the body's antioxidant capacity, removes excessive ROS, and reduces oxidative damage to muscle cells. Among them, the plateau characteristic active substances (Nitraria extract, yak collagen peptide, seabuckthorn polysaccharide) can significantly increase the levels of GSH-Px and TAOC, reduce the content of MDA, improve the oxidative stress injury of plateau exercise, and adapt to the special metabolic characteristics of plateau^[9].

The second is energy and repair nutrition supplement, timely supplement of high-quality protein, branched-chain amino acids and compound carbon water, repair of damaged

skeletal muscle, inhibition of excessive protein decomposition, acceleration of PCr resynthesis, and improvement of energy metabolism disorder^[10]. High-quality proteins and branched-chain amino acids can activate mTOR signaling pathway, promote muscle protein synthesis, inhibit ubiquitin-proteasome degradation pathway, and accelerate muscle fiber microdamage repair; compound carbohydrate quickly replenishes muscle glycogen and liver glycogen, maintains blood glucose stability, provides substrates for ATP resynthesis, and shortens recovery time. the combined application of the three can synergistically improve the fatigue state after altitude exercise at the three levels of energy supply, muscle protein repair and metabolic homeostasis.

The third is the supplementation of trace elements. the supplementation of iron, zinc and vitamins can improve the oxygen carrying capacity of plateau blood, alleviate the hypoxia state of the body, and reduce the degree of fatigue from the root^[11]. Iron is the core component of hemoglobin and myoglobin, which is directly involved in oxygen transport and storage. Zinc is involved in the composition of a variety of antioxidant enzymes and energy metabolism enzymes; b vitamins, vitamin C and E assist mitochondrial energy metabolism and antioxidant defense. Synergistic supplementation of trace elements can improve oxygen utilization efficiency under hypoxic conditions, reduce oxidative damage, and improve neuromuscular function. Compared with ordinary nutritional supplements, plateau compound special diet nutrition is more in line with the law of hypoxia metabolism. Through the synergistic effect of multi-component, multi-target and multi-pathway, the comprehensive effect of anti-oxidation, promoting repair, increasing oxygen and stabilizing energy is realized, which is a hot and innovative direction of current research.

3.3 Physical Factors Rehabilitation Intervention: Accelerate the Body Function Repair

Physical rehabilitation methods can directly act on muscles, nerves and circulatory systems to quickly relieve physical fatigue. In the high altitude hypoxic environment, blood flow redistribution, delayed removal of metabolic waste, accumulation of muscle micro-damage and neuromuscular tension are more prominent. Physical factor intervention has become a key

link between training and recovery by improving local circulation, regulating autonomic nerves, promoting metabolite transport and muscle fiber repair. At present, the commonly used methods include active recovery and passive physiotherapy:

Active recovery is mainly based on low-intensity jogging, stretching, and respiratory training, which can promote systemic blood circulation, accelerate the removal of metabolic wastes such as lactic acid and urea nitrogen, and relieve muscle stiffness and soreness. Low-intensity aerobic activity can maintain the muscle pump and respiratory pump effect, enhance venous return and lymphatic drainage, and accelerate the transport of metabolites such as lactic acid and urea nitrogen; stretching can improve the extensibility of myofascial, reduce muscle tension and restore the range of motion of joints. Breathing training can improve diaphragmatic efficiency and blood oxygen saturation, assist the autonomic nerve to shift from sympathetic excitation to parasympathetic recovery, and create conditions for subsequent repair.

Passive physiotherapy covers massage relaxation, hot compress, negative pressure recovery, physical factor therapy, etc., which can effectively improve skeletal muscle microcirculation, repair micro-damage, and relieve neuromuscular tension. Among them, massage after training can increase local tissue temperature, accelerate local circulation, promote congestion metabolism and muscle injury repair, release soft tissue adhesions, and increase joint mobility. Standardized operation can effectively relieve muscle acid stiffness and accelerate exercise fatigue recovery^[12]. Hot compress expands blood vessels through heat effect, improves tissue metabolism and oxygen supply; negative pressure recovery utilizes pressure changes to promote blood and lymphatic return and accelerate the removal of metabolic waste; physical factor therapy, such as neuromuscular electrical stimulation, low-intensity laser, and compression therapy, can regulate neuromuscular excitability, reduce inflammatory edema, and promote muscle fiber repair and pain relief. the combination of various means can form a continuous recovery chain from circulation improvement, metabolic clearance to neuromuscular relaxation and tissue repair.

Physical intervention has the advantages of non-invasive and wide adaptability, and is an important means of normalized recovery of

altitude training. In the high altitude hypoxic environment, physical rehabilitation can be coordinated with training regulation, nutrition intervention and psychological adjustment to form a three-dimensional recovery system, which is suitable for multiple scenarios such as immediate recovery after training, conditioning during fatigue accumulation period and pre-competition state adjustment. It provides a stable, repeatable and easy-to-promote practical path for the rapid relief and functional repair of high altitude exercise fatigue.

3.4 Psychological Adjustment Intervention: Improve the Central Fatigue State

High altitude hypoxia can easily induce negative emotions such as irritability and burnout, and aggravate central fatigue. Psychological intervention can regulate nerve function and relieve mental fatigue. In the hypoxic environment, the decrease of cerebral oxygenation, the activation of HPA axis and the excessive excitation of sympathetic nerve jointly weaken the executive function and emotional regulation ability of the prefrontal lobe, so that the subjective fatigue appears in advance and the recovery speed slows down. Psychological adjustment directly acts on the key link of central fatigue by stabilizing autonomic nerves, improving sleep structure and enhancing psychological resilience. Adequate sleep is the core recovery method, which ensures 7-9 hours of sleep every day and cooperates with lunch break to accelerate the removal of brain metabolic waste and repair central function. During sleep, the clearance efficiency of the lymphoid system was improved, the transport of metabolic wastes such as β -amyloid protein was accelerated, the cortisol rhythm and growth hormone secretion were restored, the prefrontal lobe and hippocampus functions were repaired, and central fatigue was alleviated. With meditation, mindfulness breathing, progressive relaxation training, inhibit sympathetic overexcitation, relieve anxiety. Mindfulness and relaxation training can improve heart rate variability, enhance parasympathetic tension, reduce cortisol levels, improve attention distribution and emotional regulation, and shift the body from stress state to recovery state.

With the help of imagery training to consolidate sports technology, while enriching amateur life, balance training and daily life, to achieve psychological withdrawal, to avoid long-term

psychological burnout. Psychological means can cooperate with physical and nutritional recovery to comprehensively improve the physical and mental fatigue caused by altitude exercise^[13]. Under the combined application of sleep support, mindfulness training and imagery training, psychological adjustment has the most direct effect on the improvement of central fatigue, and forms a central-peripheral collaborative recovery chain with physical and nutritional intervention, which provides stable support for altitude training adaptation and exercise performance improvement.

4. Current Research Deficiencies and Prospects

4.1 Insufficient Research

Throughout the existing achievements at home and abroad, there are still obvious research limitations in the field of high altitude hypoxic exercise fatigue.

4.1.1 The research system is relatively fragmented.

The existing research is limited to a single altitude condition, a single biochemical index or a single recovery method, and lacks the overall comparative analysis of altitude gradient-exercise load-fatigue mechanism-intervention benefit, and has not yet formed a unified and standardized plateau exercise fatigue evaluation system and standardized recovery plan. Different studies have great differences in altitude simulation methods, exercise load setting, index collection time points and intervention cycles, resulting in horizontal difficulties in the results, and it is difficult to reveal the continuous process of plateau exercise fatigue occurrence, development and recovery as a whole.

4.1.2 The coverage of the subjects was relatively single.

The current research mostly takes professional endurance athletes as the core object, and the research on the fatigue characteristics of plateau sojourners, mass rehabilitation sports groups and young athletes is relatively scarce. the hypoxic fatigue response law, tolerance characteristics and recovery differences of different groups are still unclear. There are significant differences between professional athletes and the general population in terms of hypoxic adaptability, training basis, metabolic reserve and recovery needs. It is difficult to directly extrapolate the existing conclusions to a wider population.

4.1.3 Intervention innovation is weak

The existing recovery methods are mainly based on routine training regulation, basic physiotherapy and single nutrition supplement, and the homogenization problem is prominent. the research on targeted nutrition intervention and precise rehabilitation technology for high altitude hypoxia-specific metabolic disorders is insufficient, and the synergistic mechanism of compound intervention programs still needs to be explained. Current intervention studies pay more attention to the short-term effects of a single method, and there is a lack of systematic verification of the time window, dose effect, interaction and individual adaptability of the combined application of multiple methods.

4.1.4 The depth of mechanism research needs to be deepened.

Most studies still remain in the representation analysis of macroscopic physiological indicators, and the systematic exploration of microscopic molecular mechanisms such as HIF hypoxia signaling pathway, mitochondrial energy metabolism, and central neurotransmitter regulation is insufficient, and the molecular regulation network of high altitude exercise-induced fatigue has not been fully revealed. the existing research has limited understanding of the linkage relationship between multiple mechanisms, time series changes and key regulatory nodes, and there is still a lack of effective connection between macro indicators and micro mechanisms.

4.2 Research prospects

In the future, this field can promote in-depth research from four aspects, focusing on mechanism analysis, population stratification, intervention innovation and intelligent monitoring, and build a precise prevention and control and recovery system for plateau exercise fatigue.

4.2.1 Deepening the research on precision mechanism

Combined with molecular biology and omics technology, the differential regulation pathways of exercise fatigue under acute and chronic hypoxia exposure were systematically analyzed, and the basic theoretical system of plateau fatigue was improved. Focus on HIF hypoxia signaling pathway, mitochondrial energy metabolism and quality control, oxidative stress-inflammation cascade, neurotransmitter regulation and muscle factor network, clarify key regulatory nodes and

spatiotemporal changes, and establish a mechanism mapping from molecular targets to physiological phenotypes. Through multi-omics integration and dynamic tracking, the molecular regulatory network of the occurrence, development and recovery of high altitude exercise fatigue is revealed, which provides a theoretical target for precise intervention.

4.2.2 Conducting population differentiation research

By comparing the fatigue response characteristics of different age, exercise basis and living population, an individualized plateau exercise fatigue assessment and recovery program was constructed to broaden the application scenarios of plateau exercise health promotion. We can compare the differences of hypoxia tolerance, fatigue accumulation and recovery rate among professional endurance athletes, plateau sojourners, public health exercise groups and young athletes, and identify the sensitive indicators and key influencing factors of different populations. On this basis, a hierarchical evaluation standard and individualized recovery strategy are established to improve the pertinence and adaptability of altitude exercise health guidance.

4.2.3 Innovative compound intervention technology

Focus on the combined intervention model of active nutrition supplement, physical rehabilitation and scientific training with plateau characteristics, clarify the multi-means collaborative recovery mechanism, and develop accurate recovery technology adapted to the plateau environment. Focusing on hypoxia-specific metabolic disorders, oxidative stress injury, abnormal neuromuscular regulation and immune-endocrine imbalance, the nutrition-training-physical-psychological multimodal combination was optimized to clarify the intervention timing, dose effect, synergistic target and applicable conditions. Through the systematic verification of the compound intervention program, the efficiency of plateau sports fatigue recovery is improved, and the competitive performance is improved and the public health is guaranteed.

4.2.4 Construction of intelligent monitoring system

Based on wearable devices and big data technology, the dynamic early warning model of altitude exercise fatigue is established by integrating physiological, psychological and

biochemical multi-dimensional indicators, so as to promote the development of altitude training and health intervention in the direction of precision and intelligence. Through continuous monitoring of heart rate variability, blood oxygen saturation, sleep quality, subjective fatigue degree and key biochemical markers, combined with machine learning and individualized modeling, a decision support system for early identification of fatigue risk, dynamic adjustment of intervention plan and real-time evaluation of recovery effect was constructed to provide intelligent support for scientific training and health management at high altitude.

5. Conclusion

Exercise-induced fatigue in high altitude hypoxic environment is a complex physiological problem caused by energy metabolism disorder, oxidative stress injury, neuromuscular abnormality and immune endocrine imbalance. the mechanism of exercise-induced fatigue is complex and the recovery is more difficult. the four types of mechanisms are not isolated, but promote the rapid occurrence and continuous accumulation of fatigue through the chain coupling of insufficient energy supply-oxidative damage amplification-neuromus regulation decline-immune endocrine imbalance, so that the plateau exercise fatigue presents the typical characteristics of peripheral and central double superposition, acute reaction and chronic accumulation. the current comprehensive recovery system of training regulation, nutritional intervention, physical rehabilitation and psychological adjustment can effectively improve the fatigue state of plateau sports, and form multi-path collaborative support in improving sports performance and promoting functional recovery. In the future, it is necessary to further promote the upgrading of research from single mechanism and single intervention to multi-mechanism coupling, individualized stratification and precise compound strategy, and to construct quantifiable, traceable and optimized recovery schemes around different altitudes, different projects and different individual response characteristics. Continuously deepening the research on plateau fatigue mechanism and innovating personalized compound recovery strategy can not only optimize the scientific training system of plateau and improve the competitive performance of athletes, but also provide important theoretical support and practical guidance for the health care of plateau

mass sports and the health protection of extreme environment.

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