RESEARCH ARTICLE

# Effects of hyperbaric oxygen on exercise-induced fatigue in rats

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**Exercise-induced fatigue (EIF) poses a significant challenge in competitive sports. Hyperbaric oxygen (HBO) therapy, due to its operational simplicity, has emerged as a potential treatment, though its underlying mechanisms remain unclear. This study investigated the efficacy and mechanism of HBO against EIF in a rat model. Twenty-four Sprague-Dawley rats were randomly assigned to a control group (CG), an EIF model group (EG), and an EIF model with HBO treatment group (HG). After a 10-day intervention, rats were euthanized for sample collection. The results showed that EIF significantly impaired growth status and altered key physiological markers compared to CG including decreased levels of myoglobin (Mb), hemoglobin (Hb), blood lactic acid (BLA), and blood urea nitrogen (BUN), as well as reduced activities of creatine kinase (CK) and lactate dehydrogenase (LDH) ($P < 0.01$). HBO treatment effectively reversed these alterations. The HG group showed significant improvements in Hb, Mb, BLA, BUN, blood glucose, muscle glycogen, liver glycogen, and CK activity compared to the EG group ($P < 0.05$). Histological examination further confirmed that HBO ameliorated EIF-induced tissue damage in the heart, liver, kidney, and skeletal muscle. The results confirmed that HBO alleviated EIF by modulating energy metabolism through maintaining glucose and glycogen, enhancing oxygen transport by increasing Hb, reducing metabolic waste by decreasing BLA and BUN, mitigating muscle damage through lowering Mb and CK activity, and protecting tissue against injury.**

**Keywords:** hyperbaric oxygen; exercise-induced fatigue; blood biochemical indexes; histomorphology.

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## Introduction

The relentless pursuit of higher performance in competitive sports has led to increasingly intense training regimens, often pushing athletes to the limits of their physiological capacity, which frequently results in exercise-induced fatigue (EIF), a state that not only impairs athletic performance and skill execution [1], but also, if not properly managed, leads to more severe consequences including endocrine dysfunction, immunosuppression, and even visceral pathology due to the cumulative effects of fatigue [2]. EIF poses a significant threat to the health and well-being of athletes, military personnel, and manual laborers. The underlying physiology of EIF is complex with energy depletion being a central factor. Carbohydrates serve as a critical energy substrate during exercise, fueling both aerobic and anaerobic metabolic pathways. Their depletion is a well-established contributor to the development of fatigue.

Hyperbaric oxygen (HBO) therapy has emerged

as a promising modality for accelerating recovery from EIF. By facilitating the dissolution of oxygen directly into plasma, HBO significantly increases oxygen delivery to tissues, thereby potentially mitigating the metabolic deficits that underpin fatigue [3]. This non-invasive approach enables a more rapid recovery compared to passive rest alone and is a currently approved clinical treatment in many regions [4]. Given its potential, HBO therapy is positioned to become a cornerstone of sports medicine and rehabilitation in the 21st century [5]. A growing body of research supports the efficacy of HBO for post-exercise recovery. Studies have demonstrated its benefits in alleviating delayed-onset muscle soreness [6], reducing exercise-induced inflammation and muscle damage [7], and even improving associated mood disturbances [8]. Furthermore, physiological investigations have shown that HBO can enhance cardiac parasympathetic reactivation in cyclists and is thought to modulate various metabolic and antioxidant defense pathways [9, 10].

Despite these encouraging findings, the precise molecular mechanisms through which HBO counteracts EIF remain incompletely understood [11]. To address this knowledge gap, this research established a standardized rodent model of EIF to systematically investigate the therapeutic effects of HBO and elucidate its impact on growth, key blood biochemical indices, organ morphology, and glucose metabolism, thereby providing deeper insights into the molecular basis of HBO action against EIF.

## Materials and methods

### Animals and grouping

Twenty-four (24) healthy, male Sprague-Dawley (SD) rats, aged 7 weeks, and weighing 188.7 ± 13.8 g were obtained from the Animal Center of Nanchang University (Nanchang, Jiangxi, China). The rats were housed in standard cages with 8 animals in each cage and under controlled conditions with room temperature of approximately 24°C, relative humidity of 40 – 55%, and a natural light cycle. All animals had free access to standard rodent food and water. All animals were randomly assigned to one of three experimental groups with 8 animals in each group including control group (CG) that rats did not undergo treadmill exercise or HBO exposure, exercise-induced fatigue group (EG) where rats were subjected to an EIF protocol *via* treadmill running, and HBO treatment group (HG) that followed the identical EIF protocol as the EG and received HBO treatment. All experimental procedures were approved by the Ethical Committee of Jiangxi Science and Technology Normal University (Nanchang, Jiangxi, China) with the approval number of 2006-398.

### Construction of chronic EIF animal model

A chronic EIF animal model was constructed over a 10-day period using a progressively intensified treadmill running protocol, adapted from a previously described method [12]. The experiments were performed using a ZH-PT animal treadmill (Huaibei Zhenghua Co., Ltd., Huaibei, Anhui, China). Prior to the formal experiment, all rats underwent a 3-day adaptation period on the treadmill, which consisted of one session per day for 30 – 60 minutes at a speed of 10 – 15 m/min and a slope of 0 – 10°. After a 2-day rest period, the formal 10-day exercise protocol was initiated, which was divided into two phases with days 1 to 5 as phase 1 and days 6 to 10 as phase 2. The exercise intensity for each phase was graded across four levels defined by slope (°), speed (m/min), duration (min). In phase 1, level I was defined as 0°, 18 m/min, 30 min, while level II was defined as 10°, 22 m/min, 30 min, level III was defined as 17°, 26.8 m/min, 60 min, and level IV was defined as 17°, 30 m/min, 60 min, respectively. The total daily exercise duration was 180 minutes. In phase 2, levels I - III were identical to Phase 1, while level IV was intensified to 17° slope, 33 m/min for 90 minutes, resulting in a total daily exercise duration of 210 minutes. To ensure compliance with the prescribed intensity, auditory, visual, and tactile (brush) stimuli were applied during running with mild electrical stimulation being used as a last resort. EIF was considered

successfully induced when rats exhibited significantly diminished exercise capacity accompanied by visible signs such as limb tremors and rapid breathing, which were later corroborated by post-experiment biochemical analyses.

**Hyperbaric oxygen treatment**

Rats in the HG group received hyperbaric oxygen (HBO) therapy once daily for 10 consecutive days. The treatment protocol was established based on the previous report and in consultation with the Hyperbaric Oxygen Treatment Center of Nanchang Hospital (Nanchang, Jiangxi, China) [13]. HBO treatments were conducted in a GYQ32 Hyperbaric Oxygen Chamber (Jiujiang Haitian Co., Ltd., Jiujiang, Jiangxi, China) with the treatment procedures as that rats were initially exposed to pure oxygen for 10 minutes before the chamber pressure being increased at a rate of 0.01 MPa/min to an absolute pressure of 0.25 MPa and being maintained for 60 minutes. During the treatment process, the chamber was ventilated with pure oxygen at 10-minute intervals to ensure an internal oxygen concentration > 98% and a $CO_2$ concentration < 0.05%. The chamber temperature was maintained at 22 – 24°C throughout the session. Upon completion of the exposure, decompression was carried out uniformly over 15 minutes to return to atmospheric pressure before removing the animals from the chamber.

**Determination of blood biochemical indexes**

Following the final exercise session on day 10, rats were anesthetized *via* intraperitoneal injection of 3% pentobarbital sodium at a dose of 0.25 mL/100 g body weight. Approximately 5 mL of blood was collected from the abdominal aorta using a blood collecting tube containing 1% heparin sodium as an anticoagulant for creatine kinase (CK), lactate dehydrogenase (LDH), myoglobin (Mb), blood urea nitrogen (BUN), and blood lactic acid (BLA) analysis. The sample was allowed to stand for 30 minutes and then centrifuged at 3,000 rpm for 4 minutes to separate the serum. The resulting serum was analyzed using an Au2700 automatic biochemical analyzer (Beckman Coulter, Inc., Brea, California, USA). A separate whole blood sample was collected into an EDTA-2K anticoagulant vacuum tube for hemoglobin (Hb) determination by using a Merry 3000 whole blood analyzer (Shenzhen Merry Co. Ltd., Shenzhen, Guangdong, China).

**Determination of glycogen in muscle and liver**

Tissue samples of skeletal muscles and liver were collected and rinsed with ice-cold saline, dried, and mechanically homogenized. An alkaline hydrolysis was then performed by adding 10% sodium hydroxide (NaOH) at a ratio of 3 µL per mg of tissue wet weight. The mixture was boiled for 20 minutes and subsequently cooled down. The resulting glycogen hydrolysate was diluted with distilled water to prepare the sample solution. After thorough mixing, the solution was boiled again for 5 minutes, cooled down, and the optical density (OD) was measured using a 721 spectrophotometer (Shandong Rainbow Co. Ltd., Gaomi, Shandong, China).

**Observation on histomorphology**

Tissue samples (each approximately 1 $cm^3$) of the heart, liver, kidney, and skeletal muscle were promptly excised and rinsed in ice-cold 0.9% saline, gently blotted dry, and fixed in 10% neutral buffered formalin for 24 – 48 hours. The fixed tissues were then processed through a standard histological protocol including gradient ethanol dehydration (70% to 100% ethanol), clearing in xylene, infiltration with molten paraffin, and final embedding in paraffin blocks. Sections at 5 µm thickness were prepared and mounted on polylysine-coated slides for storage at 4°C prior to staining. The sample slice sections were deparaffinized in xylene and rehydrated through a descending ethanol series to distilled water. Hematoxylin and eosin (H&E) staining was performed by staining with Harris hematoxylin for 10 min, differentiation in acid alcohol, bluing in 1% ammonia water, and counterstaining with 0.25% eosin for 10 min. The sample sections were dehydrated through an ascending ethanol series, cleared in xylene, and coverslipped with neutral balsam for microscopic examination.

**Table 1.** Body weight of rats in different periods (mean ± standard deviation).

| | CG (g) | EG (g) | HG (g) |
|---|---|---|---|
| **Day 0** | 197.96 ± 14.44 | 198.84 ± 9.35 | 198.28 ± 11.29 |
| **Day 1** | 209.96 ± 14.93 | 210.64 ± 10.04 | 210.79 ± 10.30 |
| **Day 4** | 225.28 ± 12.86 | 225.25 ± 9.82 | 222.56 ± 11.71 |
| **Day 7** | 244.10 ± 12.59 | 232.94 ± 8.16 | 238.33 ± 13.96 |
| **Day10** | 264.15 ± 12.38 | 235.18 ± 8.76** | 251.38 ± 13.75*▼ |

**Notes:** *: $P < 0.05$ compared to CG. **: $P < 0.01$ compared to CG. ▼: $P < 0.05$ compared to EG.

**Statistical analysis**

All experimental data were processed and analyzed using SPSS 21.0 (IBM, Armonk, New York, USA). Data was presented as the mean ± standard deviation. Differences among the three groups were assessed by one-way analysis of variance (ANOVA) followed by post-hoc tests for multiple comparisons after confirming the homogeneity of variances.

## Results and discussion

**Animal growth status**

Prior to the experimental intervention, all rats exhibited normal physiological conditions, characterized by alertness, normal activity, responsiveness to stimuli, healthy appetite, and well-groomed coats. Throughout the experimental period, rats in the CG group maintained this normal status. In contrast, rats in the EG group displayed pronounced fatigue symptoms including lethargy, reduced appetite, weight loss, piloerection, a hunched posture, and delayed responsiveness. Rats in the HG group demonstrated better tolerance to the exercise regimen. The results showed that the initial body weights did not differ significantly among the groups. During the study, body weight increased steadily in the CG group but progressed only marginally in the EG group. By day 10, the final body weight in the EG group was significantly lower than that in the CG group ($P < 0.01$). Notably, HBO treatment significantly ameliorated this effect as evidenced by a higher body weight in the HG group compared to the EG group ($P < 0.05$) (Table 1). These findings indicated that chronic exercise-induced fatigue adversely affected growth metrics, and HBO therapy could effectively mitigate these negative consequences, thereby improving overall physiological status.

**Blood biochemical indexes**

The concentrations of blood BUN and BLA were key indicators of exercise-induced metabolic stress. After the 10-day intensive exercise regimen, the EG group exhibited substantially elevated levels of both BUN and BLA compared to the CG group ($P < 0.01$). Conversely, HBO treatment significantly reduced the concentrations of these metabolites in the HG group relative to the EG group ($P < 0.05$) (Table 2), which suggested that HBO accelerated the clearance of BLA and BUN, thereby alleviating EIF. The therapeutic mechanism was likely multi-faceted, which might include that HBO enhanced aerobic metabolism and suppressed anaerobic glycolysis by increasing blood oxygen content, thereby reducing BLA production. Concurrently, by elevating blood glucose and favoring carbohydrate utilization, HBO reduced reliance on protein catabolism, leading to decreased BUN generation. Further, HBO promoted lactic acid oxidation in skeletal and cardiac muscles and enhanced hepatic gluconeogenesis from lactate, further facilitating BLA clearance. In addition, HBO might improve renal function and urine output, potentially reduce tubular reabsorption of urea and accelerate BUN elimination [14]. The activities of CK and LDH served as sensitive markers for skeletal muscle micro-injury and EIF. The results confirmed a significant increase in CK and LDH activities in the EG group compared to the CG group ($P < 0.01$). HBO treatment effectively mitigated this increase, resulting in a

**Table 2.** Content of blood biochemical indexes in rats (mean ± standard deviation).

| | CG | EG | HG |
|---|---|---|---|
| **BLA (mmol/L)** | 2.41 ± 0.70 | 5.40± 1.10** | 4.06 ± 1.13**▼ |
| **BUN (mmol/l)** | 5.29 ± 0.55 | 10.04 ± 1.86** | 7.05 ± 1.80▼ |
| **CK (U/L)** | 202.24 ± 89.61 | 550.13 ± 98.66** | 311.21 ± 35.26**▼▼ |
| **LDH (U/L)** | 513.32 ± 112.55 | 983.41 ± 253.11** | 706.34 ± 155.94* |
| **Mb (μg/L)** | 0.95 ± 0.34 | 2.60 ± 1.14** | 1.34 ± 0.28▼ |
| **Hb (g/L)** | 174.13 ± 6.45 | 104.26 ± 5.04** | 159.38 ± 11.02*▼▼ |

**Notes:** *: $P < 0.05$ compared to CG. **: $P < 0.01$ compared to CG. ▼: $P < 0.05$ compared to EG. ▼▼: $P < 0.01$ compared to EG.

markedly lower CK activity in the HG group than in the EG group ($P < 0.01$). Although LDH activity in the HG group was also reduced compared to the EG group, the difference did not reach statistical significance ($P > 0.05$) (Table 2). The trend of reduced enzyme activities indicated that HBO aided in the repair of exercise-induced muscle micro-damage. The underlying mechanism was attributed to improved tissue oxygenation under HBO. The increased plasma oxygen partial pressure enhanced oxygen diffusion and perfusion in microvasculature, alleviating local hypoxia. This improved oxygen status helped stabilize cell membrane integrity, reducing permeability and the subsequent leakage of intracellular enzymes like CK and LDH into circulation [15]. Hb and Mb levels were indicative of oxygen-carrying capacity and muscle fiber integrity, respectively. The EG group showed a significant increase in Mb and a decrease in Hb compared to the CG group ($P < 0.01$). HBO treatment effectively reversed these alterations with the HG group exhibiting a lower Mb content than the EG group ($P < 0.05$) and a significantly higher Hb concentration ($P < 0.01$), restoring levels comparable to the CG. The rapid normalization of Mb suggested that HBO mitigated sarcolemma damage, preventing its release from muscle cells, which was likely a consequence of improved cellular oxygenation and the subsequent correction of the acidic metabolic environment to stabilize membrane permeability and facilitate cellular repair. The significant increase in Hb could be explained by HBO's documented effects on improving erythrocyte deformability and reducing plasma viscosity, which enhanced red blood cell survival and circulation. Furthermore, by correcting acidosis, HBO promoted the oxygen-binding affinity of hemoglobin, collectively improving oxygen delivery and accelerating recovery from fatigue.

**Contents of blood glucose, muscle glycogen and liver glycogen**

The results demonstrated that the 10-day high-intensity treadmill exercise protocol significantly depleted energy reserves. The EG group showed substantially lower concentrations of blood glucose, muscle glycogen, and liver glycogen compared to the CG group ($P < 0.01$), which confirmed that the protracted exercise consumed key energetic substrates, contributing to the state of fatigue. HBO treatment effectively counteracted this depletion. The concentrations of blood glucose, muscle glycogen, and liver glycogen in the HG group were markedly higher than those in the EG group ($P < 0.01$) (Table 3), which indicated that HBO enhanced the preservation of glycogen storage in the liver and muscle, reduced glucose loss, and helped maintain blood glucose homeostasis. The mechanisms underlying this protective effect might be twofold, which included that HBO might protect the structural integrity of hepatic and muscular cell membranes, thereby ensuring a stable energy supply to motor muscles, the central nervous system, and red blood cells. This safeguarding of cellular function and energy delivery was a key anti-fatigue mechanism. In addition, under normal conditions, insufficient oxygen supply forced a greater reliance on anaerobic glycolysis, which accelerated glycogen consumption and increased lactic acid

**Table 3.** Concentrations of blood glucose and glycogen (mean ± standard deviation).

| | CG | EG | HG |
|---|---|---|---|
| **Blood glucose (mmol/L)** | 8.44 ± 0.90 | 5.82 ± 1.10** | 8.02 ± 1.98▼▼ |
| **Muscle glycogen (mg/g)** | 4.53 ± 1.02 | 2.70 ± 0.43** | 3.77 ± 0.49*▼▼ |
| **Liver glycogen (mg/g)** | 22.66 ± 6.96 | 8.55 ± 5.30** | 16.29 ± 3.89*▼ |

**Notes:** *: $P < 0.05$ compared to CG. **: $P < 0.01$ compared to CG. ▼: $P < 0.05$ compared to EG. ▼▼: $P < 0.01$ compared to EG.

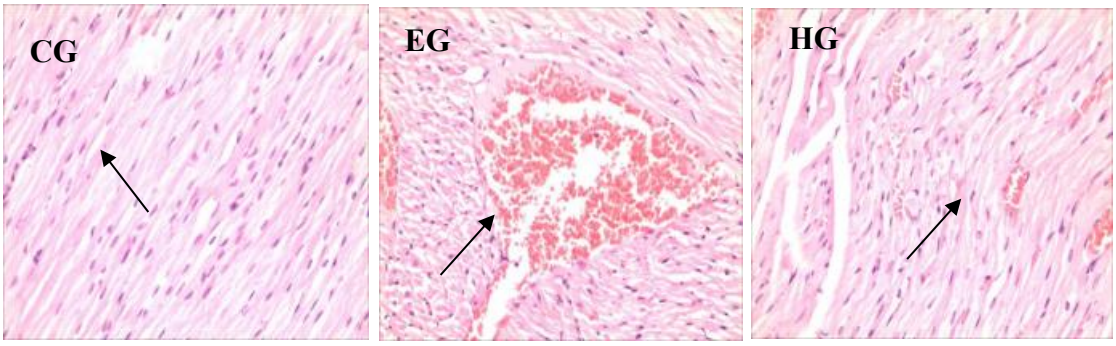


**Figure 1.** HE staining of rat myocardial tissue (×400).

production. HBO therapy increased oxygen availability and enhanced the efficiency of carbohydrate utilization, which shifted the energy supply during exercise towards the more efficient aerobic metabolism of sugars and might further promote the recycling of lactate as a substrate for glycogenesis.

**Morphological changes of cardiac tissue**

Histological examination revealed distinct morphological differences among the experimental groups. Cardiac tissue from the CG group exhibited a normal architecture, characterized by well-aligned cardiomyocytes with clear striations and an absence of vascular congestion. In contrast, EG group showed significant exercise-induced damage. The myocardial fibers were disorganized, and the interstitial spaces displayed marked vascular dilation and hyperemia. Notably, these pathological changes were ameliorated in the HG group. The myocardial fibers in the HG group were more uniform in thickness and better aligned with a pronounced reduction in interstitial vascular congestion (Figure 1). The cardiac damage observed in the EG group was likely attributable to the cumulative stress of repeated, high-intensity exercise without adequate recovery. Although rats in the HG group underwent the same exercise protocol, the application of post-exercise HBO therapy appeared to have facilitated recovery. The improved tissue morphology in the HG group suggested that HBO treatment mitigated exercise-induced cardiac damage, potentially by alleviating myocardial ischemia and hypoxia and supporting structural repair.

**Morphological changes in liver tissue**

Microscopic examination of liver tissue revealed distinct morphological alterations across the experimental groups. The CG group exhibited normal hepatic architecture, characterized by intact hepatic lobules, neatly arranged hepatocyte cords, and well-defined cellular structures with no evidence of congestion or necrosis. The EG group displayed significant pathological changes including hepatocyte swelling with enlarged nuclei, blurred stromal boundaries, dilation of the central veins, and hypertrophy of Kupffer cells within the hepatic sinusoids. These exercise-induced injuries were

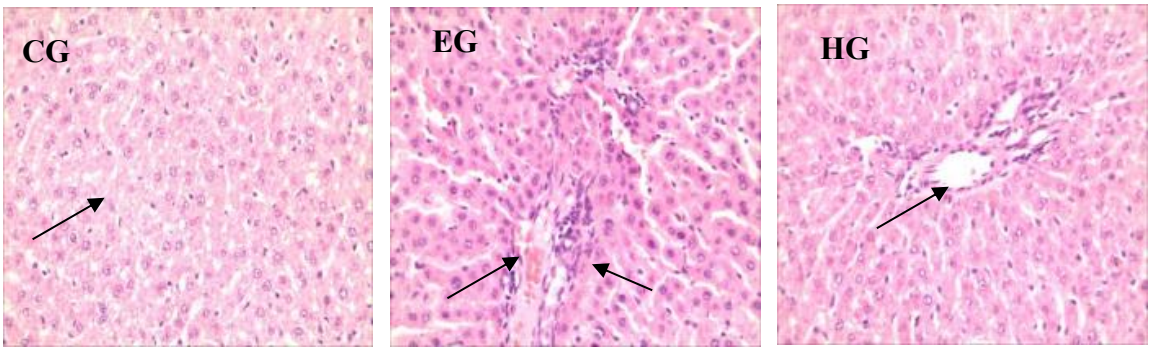


**Figure 2.** HE staining of rat liver tissue (×400).

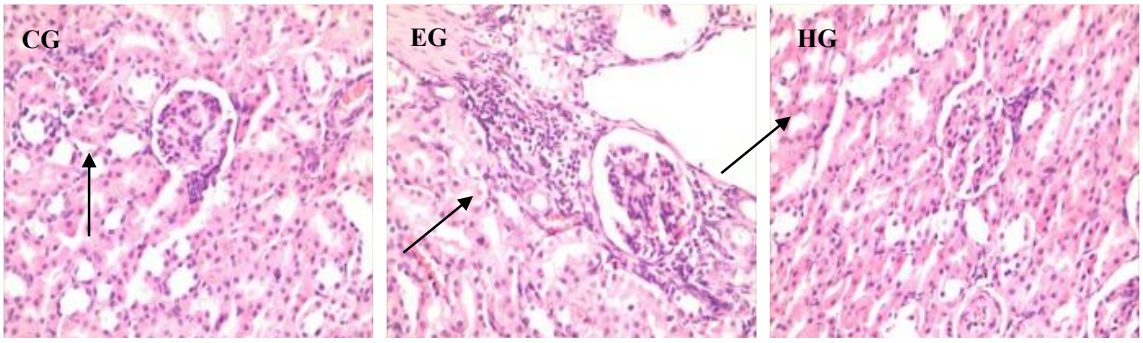


**Figure 3.** HE staining of rat renal tissue (×400).

markedly attenuated in the HG group. The liver tissues on the HG group showed considerable reduction in cellular swelling with clearer hepatocyte boundaries and restored definition of the interstitial texture (Figure 2). The observed histopathology in the EG group suggested that chronic high-intensity exercise induced substantial hepatic stress and injury. The significant amelioration of these features in the HG group indicated that post-exercise HBO therapy was effective in mitigating liver damage and promoting tissue repair, likely by improving localized oxygen supply and supporting metabolic recovery.

**Morphological changes in kidney**

Microscopic evaluation of renal tissue revealed a clear preservation of structure in the CG group, which displayed intact glomeruli and tubules without interstitial congestion. In contrast, the EG group exhibited marked pathological alterations including swelling of renal tubular epithelial cells and dilation of both glomerular capillaries and tubular lumens. HBO treatment effectively mitigated these morphological injuries. The renal tissue from the HG group showed improved architecture of glomeruli and tubules along with alleviated capillary congestion (Figure 3). These findings suggested that HBO therapy conferred a protective effect against exercise-induced renal injury, likely by counteracting ischemia and hypoxia. The underlying mechanism might involve the attenuation of vasoconstrictive hormones, thereby improving renal blood flow and oxygen delivery. Additionally, HBO was known to modulate the release of inflammatory mediators, which might further contribute to the observed reduction in tissue damage [16].

**Morphological changes in skeletal muscle**

Histological analysis of skeletal muscle revealed well-organized fibers with uniform thickness, clear cross-striations, and regularly aligned nuclei

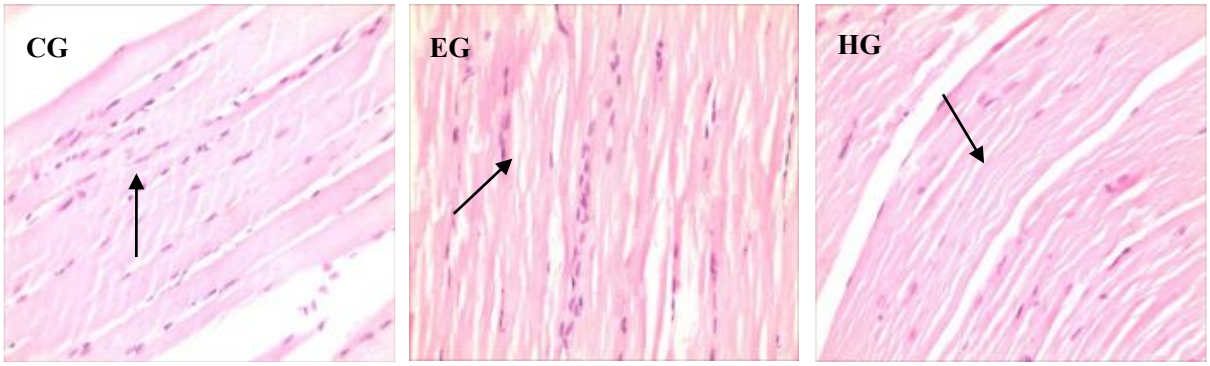


**Figure 4.** HE staining of rat skeletal muscle tissue (×400).

in the CG group. However, the EG group exhibited significant structural damage including fiber atrophy, swelling, fragmentation, uneven thickness, and disorganization, with nuclei displaying a beaded appearance. HBO treatment notably ameliorated these alterations by restoring tissue architecture, characterized by clear cross-striations, uniform fiber thickness, normal intercellular spacing, and evenly distributed nuclei (Figure 4). These results suggested that HBO exerted a protective effect against EIF-induced skeletal muscle injury with the possible mechanism of increased oxygen dissolution in the blood under hyperbaric conditions, which enhanced oxygen delivery to skeletal muscle tissue. Such improved oxygenation might facilitate the repair of exercise-induced micro-damage and support the functional recovery from fatigue [17].

**The anti-fatigue mechanism of HBO**

The mechanism by which HBO counteracted EIF was multifaceted. Previous research found that HBO dramatically increased oxygen dissolution in the blood, thereby enhancing oxygen delivery to hypoxic tissues and promoting recovery [3]. Additional studies suggested that HBO might mitigate oxidative stress by reducing reactive oxygen species and improving mitochondrial function [18]. Furthermore, HBO was recognized for accelerating the repair of exercise-induced muscle damage, thereby eliminating fatigue and restoring endurance [19], and for enhancing the body's capacity to resist exercise-induced oxidative stress [20]. The results of this study aligned with and extended this existing knowledge and confirmed that a standardized high-load exercise protocol successfully induced a state of chronic EIF in rats, which manifested as impaired growth and physiological status. Crucially, post-exercise HBO treatment effectively reversed these deficits. This research proposed an integrated mechanism for HBO against EIF, which was that HBO alleviated fatigue by orchestrating a coordinated response including preserving energy homeostasis by maintaining blood glucose and glycogen stores, accelerating the clearance of metabolic byproducts like BLA and BUN, mitigating muscle damage as evidenced by the normalization of CK, LDH, and Mb levels, enhancing oxygen-carrying capacity *via* increased Hb, and protecting against exercise-induced histopathological damage in vital organs. This multi-systemic restoration of physiological function underpinned the potent anti-fatigue effects of HBO therapy. This research provided a physiological foundation for the application of HBO therapy in sports medicine and offered new insights for developing strategies to combat exercise-induced fatigue.

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