

## Review Article / Derleme

# Sports injuries and hyperbaric oxygen therapy: physiological effects and previous findings

## *Spor yaralanmaları ve hiperbarik oksijen tedavisi: fizyolojik etkiler ve önceki bulgular*

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### ABSTRACT

Problems encountered in the management of sports injuries can lead to a prolonged treatment process and a long period of time away from sports that may cause significant economic loss, as well. To ensure the most effective return to sports, a multidisciplinary approach is often required.

Research has demonstrated that hyperbaric oxygen therapy (HBOT) shows promising results as a treatment modality for sports injuries. A search of the scientific literature was conducted using the keywords "hyperbaric oxygen therapy" and "sports injuries" across PubMed, Cochrane Library, and Web of Science databases. The positive effects of HBOT have been reported in the treatment of various sports injuries, including muscle and soft tissue injuries, as well as bone marrow edema.

This review examines the physiological effects of hyperbaric oxygen therapy, as well as the findings obtained from animal injury models and human clinical studies on its use in sports injuries. The effects of HBOT vary depending on the type of injury, and it is generally more beneficial as an adjunctive treatment rather than as a standalone therapy.

Although numerous studies have been published, a standardized treatment protocol has yet to be established. However, based on current protocols in the literature, it is believed that a 90- to 120-minute treatment session, administered at least five days per week at 2.0-2.5 ATA, will provide the greatest benefit. The total number of sessions should be determined by the physician following the treatment, based on the patient's specific condition and needs.

**Keywords:** Hyperbaric oxygen therapy, sports injuries

### ÖZ

Spor yaralanmalarının yönetiminde karşılaşılan sorunlar tedavi sürecinin uzamasına ve spordan uzun süre uzak kalınmasına yol açabilir. Bu durum önemli ölçüde ekonomik kayba neden olabilir. Spora dönüş sürecinin en iyi şekilde tamamlanabilmesi sıklıkla multidisipliner bir yaklaşım gerektirmektedir.

Yapılan araştırmalar, hiperbarik oksijen tedavisinin spor yaralanmalarında bir tedavi yöntemi olarak umut verici sonuçlar sunduğunu göstermektedir. Bilimsel literatür, PubMed, Cochrane Library ve Web of Science veritabanları aracılığıyla "hyperbaric oxygen therapy" ve "sports injuries" anahtar sözcükleriyle taranmıştır. Kas ve diğer yumuşak doku yaralanmaları ile kemik iliği ödemi gibi pek çok spor yaralanmasında hiperbarik oksijen tedavisinin olumlu etkileri raporlanmıştır.

Bu derlemede, hiperbarik oksijen tedavisinin fizyolojik etkileri ile spor yaralanmalarına ilişkin çalışmalarda kullanılan hayvan modelleri ve insan klinik çalışmalarının sonuçları incelenmiştir. HBO2'nin etkisi, yaralanma türüne göre değişmekte olup, tek başına bir tedavi yöntemi olarak değil, daha çok yardımcı bir tedavi olarak yarar sağlamaktadır. Literatürde birçok çalışma bulunmasına rağmen, standart bir tedavi protokolü henüz oluşturulmamıştır. Ancak, literatürdeki mevcut protokoller göz önünde bulundurulduğunda, 2,0-2,5 ATA'da haftada en az 5 gün uygulanan 90 veya 120 dakikalık bir tedavi protokolünün en iyi yararı sağlayacağı düşünülmektedir. Toplam seans sayısı, hastanın durumu göz önünde bulundurularak tedavi sürecini izleyen hekim tarafından belirlenmelidir.

**Anahtar Sözcükler:** Hiperbarik oksijen tedavisi, spor yaralanmaları

## INTRODUCTION

Competing in sports involves significant engagement of various body parts. The pressure to succeed often leads athletes to train without being adequately physically prepared for competitions. Insufficient recovery and improper training can result in decreased performance, fatigue, and an increased risk of injury (1). The approach of the sports physician to injury management plays a critical role in an athlete's ability to resume regular activity. Failure to promptly

diagnose and properly manage sports injuries can lead to extended absences from athletic activities. This can also result in substantial financial costs for both amateur and professional sports (2).

Fair and Champa estimate that eliminating contact in college sports could save up to \$18.4 billion annually (3). In the English Premier League, injury-related performance declines lead to losses of approximately £900 million GBP each

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season (4). In the Netherlands, sports injuries cost over €3 billion per year (5).

The management of sports injuries often requires a multi-disciplinary approach to ensure that athletes can return to training as quickly as possible. Research indicates that hyperbaric oxygen therapy shows considerable promise in the management of sports injuries (6,7). Many elite athletes from diverse sports, including tennis, swimming, basketball, and golf, have reported using hyperbaric oxygen therapy as part of their recovery (8).

### Hyperbaric Oxygen Therapy

**Hyperbaric Oxygen Therapy (HBOT)** involves the intermittent administration of 100% oxygen at a pressure greater than one atmosphere absolute (ATA) in a closed chamber. For the treatment to be clinically effective, the pressure must be at least 1.4 ATA, though the typical range is between 2.0 and 3.0 ATA. According to the Undersea and Hyperbaric Medical Society (UHMS), for a treatment to be classified as hyperbaric oxygen therapy, the pressure must exceed 2.0 ATA. Treatment duration can be adjusted based on the

patient's condition, typically lasting between 90 and 120 minutes. Patients breathe oxygen through a mask, hood, or endotracheal tube, in either a single or multi-patient chamber (9). The indications approved by UHMS and recommended by the European Committee on Hyperbaric Medicine are listed in Tables 1 and 2.

**Table 1.** UHMS HBO<sub>2</sub> Indications

Air or Gas Embolism	Carbon Monoxide Poisoning
Central Retinal Artery Occlusion	Decompression Sickness
Clostridial Myonecrosis (Gas Gangrene)	Acute Traumatic Ischemia
Compromised Grafts and Flaps	Sudden Sensorineural Hearing Loss
Delayed Radiation Injuries	Thermal Burns
Intracranial Abscess	Avascular Necrosis*
Necrotizing Soft Tissue Infections	
Refractory Osteomyelitis	
Severe Anemia	

\* Recently added as an indication in the new version of the UHMS indication list

**Table 2.** Recommendations on the HBO<sub>2</sub> indications accepted for European Consensus (5)

Type 1	Type 2	Type 3
CO poisoning	Crush injury without fracture	Brain injury (acute and chronic TBI, chronic stroke, post anoxic encephalopathy) in highly selected patients
Open fractures with crush injury	Osteoradionecrosis (bones other than mandible)	Radio-induced lesions of larynx
Prevention of osteoradionecrosis after dental extraction	Radio-induced lesions of soft tissues (other than cystitis and proctitis)	Radio-induced lesions of the CNS
Osteoradionecrosis (mandible)	Surgery and implant in irradiated tissue (preventive treatment)	Post-vascular procedure reperfusion syndrome
Soft tissue radionecrosis (cystitis, proctitis)	Ischemic ulcers	Limb replantation
Decompression illness	Refractory chronic osteomyelitis	Selected non-healing wounds secondary to systemic processes
Gas embolism	Burns, 2nd degree more than 20% body surface area	Sickle cell disease
Anaerobic or mixed bacterial infections	Pneumatosis cystoides intestinalis	Interstitial cystitis
Sudden deafness	Neuroblastoma, stage IV	
Diabetic foot lesions		
Femoral head necrosis		
Compromised skin grafts and musculocutaneous flaps		
Central retinal artery occlusion		

Type 1 = strongly recommended, Type 2 = recommended, Type 3 = possible/optional

### Physiological Effects of HBO<sub>2</sub> on Sports Injuries

The plasma oxygen concentration at sea level is approximately 3 ml/l (10). Under standard perfusion conditions, tissues require about 60 ml/l of oxygen at rest to maintain normal cellular metabolism. Without the contribution of oxygen bound to hemoglobin, dissolved oxygen at a pressure of 3 ATA is nearly sufficient to meet the resting total oxygen requirement of many tissues (11). By increasing PaO<sub>2</sub>, hyperbaric oxygen therapy (HBOT) promotes the proliferation of endothelial progenitor cells, neoangiogenesis, and neovascularization (12). Additionally, HBOT reduces cellular

ischemia and edema and induces vasoconstriction by raising the concentration of extracellular oxygen (13).

HBOT also stimulates the mobilization of hypoxia-inducible factor 1- $\alpha$ , vascular endothelial growth factor (VEGF), stromal-derived factor 1, and bone marrow-derived stem cells (CD34), while reducing neutrophil adhesion through modification of integrin  $\beta$ -2, which helps attenuate ischemia-reperfusion injury (14-17). Furthermore, HBOT enhances cell growth and modulates the inflammatory response by increasing the production of reactive oxygen species (ROS) and reactive nitrogen species. This leads to improved vasculari-

zation and better tissue survival following ischemia. ROS play a crucial role in multiple pathways that facilitate neo-angiogenesis and vasculogenesis, both of which are mediated by hypoxia-inducible factors (18,19).

Following muscle damage, inflammation is marked by the generation and release of inflammatory cytokines, increased vascular permeability, neutrophil migration, and edema (20). HBOT helps reduce edema through improved homeostatic processes and vasoconstriction. Moreover, by reducing tissue damage and inhibiting leukocyte adhesion to the endothelium, HBOT enhances leukocyte motility and improves microcirculation (21). HBOT accelerates the transition from the inflammatory to the proliferative phase (7). Horie et al. found that HBOT treatment accelerated satellite cell proliferation and myofiber maturation (22). Similarly, Oyaizu et al. reported that HBOT increased the number of proliferating and differentiating satellite cells, as well as regenerating muscle fibers (23).

### Complications

Complications of HBO<sub>2</sub> are typically oxygen- or pressure-related and most often include barotrauma or oxygen toxicity affecting the central nervous system or pulmonary systems. Rarely, symptoms such as weakness, dizziness, hypoglycemia, and vision problems (e.g., reversible myopia) may occur. The most common complication is **middle ear barotrauma**. In a study involving 62,614 HBO<sub>2</sub> sessions with 2,334 patients, middle ear barotrauma occurred in 9.2% of patients and 0.04% of sessions. Other complications, such as hypoglycemia, oxygen toxicity, dizziness, anxiety reactions, shortness of breath, and chest pain, were reported in 0.5%-1.5% of patients (24).

Evaluating patients for potential risks before initiating HBO<sub>2</sub>, taking precautions to prevent complications, and closely monitoring patients during treatment are critical for enhancing safety. Most adverse events can be effectively prevented or managed with simple measures. From this perspective, HBO<sub>2</sub> is considered one of the safest medical treatments. It is also important to note that patients undergoing HBO<sub>2</sub> often have multiple comorbidities, which may increase their risk of complications. Conversely, healthy individuals have a significantly lower likelihood of experiencing adverse effects.

### WADA's Stance and Potential Risks for Athletes

In 2006, the World Anti-Doping Agency (WADA) banned the use of HBO<sub>2</sub> under the category of oxygen transfer enhancement. However, due to insufficient evidence supporting its potential to enhance performance, WADA reclassified supplemental oxygen as permitted in 2009. In 2016, it was

further clarified that supplemental oxygen was allowed only via inhalation (25).

It is important to note that WADA may revise its stance or impose additional restrictions-such as limits on pressure or duration-if future evidence reveals new effects of HBO<sub>2</sub>.

Moreover, although rare in healthy individuals, there is always a possibility of complications arising from HBO<sub>2</sub> use. Experiencing any severe complications could delay an athlete's return to the field.

### HBO<sub>2</sub> Application in Different Types of Sports Injuries

#### Muscle Injuries

Muscle injuries encompass a wide range of conditions, including muscle strains, muscular contusions, delayed-onset muscle soreness, and muscle cramps (26). According to a study conducted between 2009 and 2015 on collegiate athletes, muscle strains were the second most common cause of sports suspension lasting more than 21 days (27).

Research on animals by Best et al. demonstrated that HBO<sub>2</sub> therapy promotes both morphological and functional recovery after acute muscular stretch injuries (28). Another animal study found that rats treated with HBO<sub>2</sub> following a muscular contusion exhibited lower levels of creatine kinase-an indicator of muscle injury-and an increase in muscle weight (29).

HBO<sub>2</sub> is beneficial not only for treating sports injuries but also for alleviating sports-related fatigue. During the Nagano Winter Olympics, Ishii and colleagues reported using HBO<sub>2</sub> as a recovery method for muscular fatigue. Their findings revealed that athletes who underwent HBO<sub>2</sub> therapy at a pressure as low as 1.3 ATA (approximately 11 feet below sea level) for short sessions of 30-40 minutes (averaging two sessions, with a maximum of six) experienced faster recovery times (30).

Additionally, other studies have shown a significant reduction in blood lactate concentration and notable improvements in maximal oxygen consumption and oxygen consumption at the anaerobic threshold following HBO<sub>2</sub> therapy (31, 32).

As previously reported in the literature, HBO<sub>2</sub> may also be effective in treating delayed-onset muscle soreness (DOMS). DOMS is characterized by soreness in skeletal muscles following intense exercise, typically resolving within 5-7 days (7). However, DOMS can lead to a temporary decline in physical performance and an increased risk of injury (33, 34). HBO<sub>2</sub> has been shown to enhance muscle strength, reduce pain, and accelerate recovery in individuals with DOMS.

Webster et al. investigated the effects of HBO<sub>2</sub> on recovery from exercise-induced muscle damage in a study involving

12 healthy males (35). Muscle damage was induced through calf raises performed to failure. Participants were divided into two groups: one received HBO<sub>2</sub> therapy (2.5 ATA with 100% oxygen for 60 minutes), while the other received a placebo (1.3 ATA with air). Treatments were administered at 3-4 hours, 24 hours, and 48 hours post-exercise. In the placebo group, isometric peak torque declined from baseline on days 1 and 2, whereas no such decline was observed in the HBO<sub>2</sub> group. By day 5, the HBO<sub>2</sub> group reported significantly lower levels of pain and soreness compared to the placebo group.

Staples et al. conducted the largest study on HBO<sub>2</sub> and DOMS, involving 66 untrained males, and demonstrated that HBO<sub>2</sub> significantly accelerated recovery in athletes (36). In this study, DOMS was induced in the non-dominant quadriceps, and pain and muscle torque were measured before, during, and after HBO<sub>2</sub> therapy. Participants were divided into four groups: HBO<sub>2</sub>, delayed HBO<sub>2</sub>, sham treatment, and control.

The HBO<sub>2</sub> group received 100% oxygen at 2.0 ATA for one hour at 0-, 24-, and 48-hours post-exercise, followed by sham treatments at 72 and 96 hours. The sham treatment group received 21% oxygen at 1.2 ATA for the same duration. The delayed HBO<sub>2</sub> group underwent sham treatments at 0- and 24-hours post-exercise, followed by HBO<sub>2</sub> therapy at 48, 72, and 96 hours. Results showed that the HBO<sub>2</sub> group exhibited significantly improved muscle torque compared to the other groups.

Harrison et al. investigated the use of HBO<sub>2</sub> for treating exercise-induced muscle injuries in 18 untrained, college-aged males (37). Participants were divided into three groups: immediate HBO<sub>2</sub>, delayed HBO<sub>2</sub>, and control. Muscle injury was induced in the non-dominant arm using preacher curls. The immediate HBO<sub>2</sub> group received oxygen therapy shortly after exercise and for the next four days, while the delayed group initially received sham treatments and began HBO<sub>2</sub> therapy later. Measurements included creatine kinase levels, muscle strength, soreness, and MRI scans. The study found no significant differences between the groups in any measure, suggesting that HBO<sub>2</sub> therapy did not facilitate soft tissue injury recovery. However, the authors cautioned that their findings might not fully apply to soft tissue athletic injuries due to the study's limitations. They suggested that HBO<sub>2</sub> could be more effective for injuries involving more extensive soft tissue damage or where oxygen availability is significantly compromised by the injury's location or severity of edema.

Babul et al. also explored the effects of intermittent HBO<sub>2</sub> on exercise-induced muscle damage in 16 females (38). Participants were divided into HBO<sub>2</sub> and control groups and

subjected to 300 maximal eccentric contractions on the non-dominant leg, performed in 30 sets of 10 repetitions with 15-second rests between sets. The HBO<sub>2</sub> group received 100% oxygen at 2 ATA for 60 minutes, while the control group was exposed to 1.2 ATA with air. Four treatment sessions were conducted over four days. Measurements included muscle soreness, eccentric strength, quadriceps circumference, creatine kinase, malondialdehyde, and muscle signal intensity via MRI. No significant differences were observed between the groups. Presti et al. later noted that the small sample size limits the generalizability of these findings (39).

In contrast, Chen et al. studied 41 baseball players already experiencing exercise-induced muscle soreness or grade I muscle strain in their extremities (40). Participants were divided into an HBO<sub>2</sub> group and a placebo group while continuing their training routines. The HBO<sub>2</sub> group was exposed to normal air for 15 minutes while the chamber was pressurized to 2.5 ATA, followed by three 25-minute sessions of 100% oxygen with five-minute air breaks between sessions. The placebo group was pressurized to 1.3 ATA and received 100% oxygen only during depressurization. Treatments were conducted twice a week for five weeks. Blood samples were taken before therapy began, after the fifth and tenth sessions, and two weeks post-treatment to assess creatine phosphokinase, myoglobin, lactate, blood urea nitrogen, and glutamic oxaloacetic transaminase.

Results revealed significant reductions in glutamic oxaloacetic transaminase, myoglobin, creatine phosphokinase, and pain in the HBO<sub>2</sub> group after the fifth and tenth sessions. The HBO<sub>2</sub> group also reported significant improvements in pain intensity and interference compared to the control group. These findings suggest that HBO<sub>2</sub> is effective in reducing markers of muscle injury and alleviating pain, thereby supporting recovery.

Although multiple studies have concluded that HBO<sub>2</sub> has no significant effect on DOMS (6), methodological issues such as small sample sizes, lack of randomization and blinding, and contradictory study designs limit their reliability (41). Further large-scale, randomized controlled trials are needed to obtain more definitive conclusions.

### ***Ligament Injuries***

Ishii et al. investigated the effects of HBO<sub>2</sub> on ligament healing in the right hind limbs of 44 male Wistar rats with experimentally induced injuries (42). The rats were divided into four groups post-injury: (a) a control group that breathed room air in a hyperbaric chamber for 60 minutes; (b) HBO<sub>2</sub> at 1.5 ATA for 30 minutes once a day; (c) HBO<sub>2</sub> at 2 ATA for 30 minutes once a day; and (d) HBO<sub>2</sub> at 2 ATA for

60 minutes once a day. Fourteen days after the injury, the researchers compared gross appearance, histology, and pro-alpha (I) mRNA expression across groups. HBO<sub>2</sub> significantly enhanced ligament healing compared to the control group, with the most effective treatment being HBO<sub>2</sub> at 2 ATA for 60 minutes daily, as evidenced by increased extracellular matrix deposition and collagen synthesis.

Horn et al. evaluated HBO<sub>2</sub>'s effects on healing lacerated medial collateral ligaments (MCL) in 48 Sprague-Dawley rats (43). The right MCL was surgically lacerated, while the left remained intact as a control. Half of the rats underwent daily HBO<sub>2</sub> at 2.8 ATA for 1.5 hours over five days. Stiffness and force to failure were assessed at 2, 4, 6, and 8 weeks post-surgery. At 4 weeks, the HBO<sub>2</sub> group showed significantly increased force to failure in the injured ligaments compared to the control group, though no additional improvements were observed by 6 weeks.

Mashitori et al. studied 76 Sprague-Dawley rats with a 2-mm segment of the MCL removed (44). Thirty-eight rats received HBO<sub>2</sub> at 2.5 ATA for 2 hours, five days a week, while the remaining rats were exposed to room air. The HBO<sub>2</sub> group exhibited more scar tissue, significantly higher Type I procollagen gene expression at 7 and 14 days, and greater tensile strength and stiffness at 14 days. These findings indicate that HBO<sub>2</sub> enhances scar tissue formation and improves ligament tensile properties.

In a double-blind, randomized, controlled study, Soolsma examined HBO<sub>2</sub>'s effect on grade II MCL recovery in humans (45). The protocol included clinical assessments, MRI scans, and two weeks of either HBO<sub>2</sub> (2 ATA, 100% oxygen, one hour daily) or sham treatment (1.2 ATA). Results showed that HBO<sub>2</sub> reduced edema and muscle wasting, improved range of motion, and enhanced maximum knee flexion.

Takeyama et al. investigated the effects of HBO<sub>2</sub> on medial collateral ligament (MCL) and anterior cruciate ligament (ACL) injuries in 64 Sprague-Dawley rats (46). HBO<sub>2</sub>-treated groups (2.5 ATA) displayed significantly higher Type I procollagen and TIMPs gene expression compared to controls. While HBO<sub>2</sub> improved structural protein synthesis and inhibited degradation in ACL injuries, it was insufficient for complete healing. The authors suggested that HBO<sub>2</sub> could enhance ACL surgery outcomes when used adjunctively.

Yeh et al. studied HBO<sub>2</sub>'s effects on ACL reconstruction in rabbits (47). Forty rabbits were divided into an HBO<sub>2</sub> group (2.5 ATA, 100% oxygen, 2 hours daily) and a control group. Postoperative analyses revealed that the HBO<sub>2</sub> group exhibited increased Sharpey's fibers, enhanced neovascularization, and better tendon-bone integration. Biomechanical testing showed higher pullout strength, while electron mic-

roscopy revealed more compact collagen fibers in the HBO<sub>2</sub> group.

HBO<sub>2</sub> generally promotes ligament and tendon healing by enhancing scar tissue formation, collagen synthesis, and tensile strength. Its effectiveness is dose-dependent, with higher-pressure and longer-duration treatments yielding better results. However, some studies suggest benefits may plateau over time, highlighting the need for further research into optimal treatment protocols.

### **Bone Marrow Edema**

Bone marrow edema is a common sports injury, particularly following acute trauma (48). If untreated, it can lead to complications such as osteonecrosis (49-51). Treatment options include pharmaceuticals like non-steroidal anti-inflammatory drugs (NSAIDs) and bisphosphonates, weight-bearing restrictions, and core decompression. However, these interventions may be less effective in cases of poor circulation (49,51,52).

HBO<sub>2</sub> therapy offers additional benefits, including circulation improvement and anti-edema effects. It also promotes bone regeneration by enhancing osteosynthesis, neoangiogenesis, and vasculogenesis (18). Studies show that HBO<sub>2</sub> stimulates the expression of osteogenic markers and increases mineral deposition in mesenchymal stem cells (53).

Clinical studies support the beneficial effects of HBO<sub>2</sub> on osteonecrosis and bone marrow edema. Numerous reports document reduced pain scores, improved range of motion, enhanced quality of life, and radiographic improvements in patients with avascular necrosis of the femoral head following HBO<sub>2</sub> therapy (54-60).

Moghamis et al. identified HBO<sub>2</sub> as a promising, non-invasive alternative to core decompression for treating non-traumatic pre-collapse avascular necrosis of the femoral head (61). Additionally, Ververidis et al. reviewed eight studies (2004-2018) and found that HBO<sub>2</sub> effectively resolved edema, relieved pain, and improved range of motion in patients unresponsive to conservative treatments. Although the time required for remission (15-90 sessions) was noted as a drawback, HBO<sub>2</sub> was highlighted as a potential alternative for these cases (51).

### **CONCLUSION**

This systematic review examines the potential benefits of HBO<sub>2</sub> for various sports injuries. The literature is extensive, but standardization is challenging due to the diversity of injury types, the use of different animal models in experiments, the limited number of human studies, and the lack of randomization and blinding. Narrowing the scope of fu-

ture reviews to specific injury types could improve the practical value of such studies.

HBO<sub>2</sub> appears more effective as an adjunctive treatment than a standalone therapy, with its impact varying by injury type. While no standardized treatment protocol has been established, evidence from existing studies suggests that a protocol involving 90 to 120 minutes of HBO<sub>2</sub> at 2.0-2.5 ATA for at least five days a week could be optimal. The total number of sessions should be tailored to the patient's condition.

Future research should focus on standardizing treatment protocols, evaluating long-term effects, and conducting randomized, controlled, double-blind clinical trials with larger human sample sizes, particularly among athletes. This would enable more precise conclusions about the efficacy of HBO<sub>2</sub> in sports injury management.

Despite some limitations, the literature provides encouraging evidence of HBO<sub>2</sub>'s therapeutic potential for sports injuries. Continued research will help solidify its role as a valuable treatment modality.

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Both authors contributed to all stages of the manuscript writing process. All authors contributed to the final version of the manuscript and discussed the results and contributed to the final manuscript.

## REFERENCES

1. Reilly T, Ekblom B. The use of recovery methods post-exercise. *J Sports Sci.* 2005 Jun;23(6):619-27.
2. Cisyk J, Courty P. An Economic Approach to Sports Injury Policies. *J Sports Econ.* 2024 Apr;25(3):388-419.
3. Fair RC, Champa C. Estimated Costs of Contact in College and High School Male Sports. *J Sports Econ.* 2019 Jun;20(5):690-717.
4. Eliakim E, Morgulev E, Lidor R, Meckel Y. Estimation of injury costs: financial damage of English Premier League teams' underachievement due to injuries. *BMJ Open Sport Exerc Med.* 2020;6(1):e000675.
5. Verhagen EALM, van Stralen MM, van Mechelen W. Behaviour, the key factor for sports injury prevention. *Sports Med Auckl NZ.* 2010 Nov 1;40(11):899-906.
6. Barata P, Cervaens M, Resende R, Camacho Ó, Marques F. Hyperbaric Oxygen Effects on Sports Injuries. *Ther Adv Musculoskelet Dis.* 2011 Apr;3(2):111-21.
7. Moghadam N, Hieda M, Ramey L, Levine BD, Guillod R. Hyperbaric Oxygen Therapy in Sports Musculoskeletal Injuries. *Med Sci Sports Exerc.* 2020 Jun;52(6):1420-6.
8. Slater G. Hyperbaric Oxygen Therapy as an Accelerator in Regenerative Medicine. *J Regen Biol Med [Internet].* 2023 Dec 1 [cited 2024 May 3]; Available from: <https://maplespub.com/article/hyperbaric-oxygen-therapy-as-an-accelerator-in-regenerative-medicine>
9. Huang ET, editor. UHMS Hyperbaric Medicine Indications Manual. 15th ed. North Palm Beach: Best Publishing Company; 2023.
10. Pittman RN. The Circulatory System and Oxygen Transport. In: Regulation of Tissue Oxygenation [Internet]. Morgan & Claypool Life Sciences; 2011 [cited 2024 May 3]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK54112/>
11. Leach RM, Rees PJ, Wilmshurst P. ABC of oxygen: Hyperbaric oxygen therapy. *BMJ.* 1998 Oct 24;317(7166):1140-3.
12. Vezzani G, Iezzi M, Rizzato A, Quartesan S, Mangar D, Camporesi EM, et al. Effects of Hyperbaric Oxygen Exposure on Mobilization of Endothelial Progenitor Cells in Healthy Volunteers. *Acta Medica Mediterr.* 2017 Jul 3;(5):801-5.
13. Bosco G, Yang Z jin, Nandi J, Wang J, Chen C, Camporesi EM. Effects of hyperbaric oxygen on glucose, lactate, glycerol and anti-oxidant enzymes in the skeletal muscle of rats during ischemia and reperfusion. *Clin Exp Pharmacol Physiol.* 2007;34(1-2):70-6.
14. Benincasa JC, de Freitas Filho LH, Carneiro GD, Sielski MS, Giorgio S, Werneck CC, et al. Hyperbaric oxygen affects endothelial progenitor cells proliferation in vitro. *Cell Biol Int.* 2019 Feb;43(2):136-46.
15. Shaw FL, Winyard PG, Smerdon GR, Bryson PJ, Moody AJ, Eggleton P. Hyperbaric oxygen treatment induces platelet aggregation and protein release, without altering expression of activation molecules. *Clin Biochem.* 2009 Apr;42(6):467-76.
16. Sunkari VG, Lind F, Botusan IR, Kashif A, Liu ZJ, Ylä-Herttuala S, et al. Hyperbaric oxygen therapy activates hypoxia-inducible factor 1 (HIF-1), which contributes to improved wound healing in diabetic mice. *Wound Repair Regen Off Publ Wound Heal Soc Eur Tissue Repair Soc.* 2015;23(1):98-103.
17. Yang Y, Wei H, Zhou X, Zhang F, Wang C. Hyperbaric oxygen promotes neural stem cell proliferation by activating vascular endothelial growth factor/extracellular signal-regulated kinase signaling after traumatic brain injury. *Neuroreport.* 2017 Dec 13;28(18):1232-8.
18. Camporesi EM, Bosco G. Mechanisms of action of hyperbaric oxygen therapy. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc.* 2014;41(3):247-52.
19. Thom SR. Hyperbaric oxygen: its mechanisms and efficacy. *Plast Reconstr Surg.* 2011 Jan;127 Suppl 1(Suppl 1):1315-1415.
20. Brancaccio P, Lippi G, Maffulli N. Biochemical markers of muscular damage. *Clin Chem Lab Med.* 2010 Jun;48(6):757-67.
21. Mortensen CR. Hyperbaric oxygen therapy. *Curr Anaesth Crit Care.* 2008 Oct;19(5-6):333-7.
22. Horie M, Enomoto M, Shimoda M, Okawa A, Miyakawa S, Yagishita K. Enhancement of satellite cell differentiation and functional recovery in injured skeletal muscle by hyperbaric oxygen treatment. *J Appl Physiol Bethesda Md* 1985. 2014 Jan 15;116(2):149-55.
23. Oyaizu T, Enomoto M, Yamamoto N, Tsuji K, Horie M, Muneta T, et al. Hyperbaric oxygen reduces inflammation, oxygenates injured muscle, and regenerates skeletal muscle via macrophage and satellite cell activation. *Sci Rep.* 2018 Jan 22;8(1):1288.
24. Hadanny A, Meir O, Bechor Y, Fishlev G, Bergan J, Efrati S. The safety of hyperbaric oxygen treatment-retrospective analysis in 2,334 patients. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc.* 2016;43(2):113-22.
25. World Anti Doping Agency [Internet]. [cited 2024 Aug 20]. Health, Medical & Research Committee - Meeting Minutes. Available from: <https://www.wada-ama.org/en/resources/health-medical-research-committee-meeting-minutes>
26. Petersen J, Hölmich P. Evidence based prevention of hamstring injuries in sport. *Br J Sports Med.* 2005 Jun;39(6):319-23.
27. Kay MC, Register-Mihalik JK, Gray AD, Djoko A, Dompier TP, Kerr ZY. The Epidemiology of Severe Injuries Sustained by National Collegiate Athletic Association Student-Athletes, 2009-2010 Through 2014-2015. *J Athl Train.* 2017 Feb;52(2):117-28.
28. Best TM, Loitz-Ramage B, Corr DT, Vanderby R. Hyperbaric oxygen in the treatment of acute muscle stretch injuries. Results in an animal model. *Am J Sports Med.* 1998;26(3):367-72.
29. Cervaens Costa Maia M, Camacho OF, Pinto Marques AF, Barata de Silva Coelho PM. Hyperbaric oxygen therapy treatment for the recovery of muscle injury induced in rats. *Diving Hyperb Med.* 2013 Dec;43(4):222-5.
30. Ishii Y, Deie M, Adachi N, Yasunaga Y, Sharman P, Miyanaga Y, et al. Hyperbaric oxygen as an adjuvant for athletes. *Sports Med Auckl NZ.* 2005;35(9):739-46.
31. Hadanny A, Hachmo Y, Rozali D, Catalogna M, Yaakobi E, Sova M, et al. Effects of Hyperbaric Oxygen Therapy on Mitochondrial Respiration and Physical Performance in Middle-Aged Athletes: A Blinded, Randomized Controlled Trial. *Sports Med - Open.* 2022 Feb 8;8(1):22.
32. Sueblinvong T, Egtasaeng N, Sanguangrangsirikul S. Hyperbaric oxygenation and blood lactate clearance: study in sixty male naval cadets. *J Med Assoc Thai Chotmailhet Thangphaet.* 2004 Sep;87 Suppl 2:S218-222.
33. Burt DG, Twist C. The effects of exercise-induced muscle damage on cycling time-trial performance. *J Strength Cond Res.* 2011 Aug;25(8):2185-92.
34. Cheung K, Hume P, Maxwell L. Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Med Auckl NZ.* 2003;33(2):145-64.
35. Webster AL, Syrotaik DG, Bell GJ, Jones RL, Hanstock CC. Effects of hyperbaric oxygen on recovery from exercise-induced muscle damage in humans. *Clin J Sport Med Off J Can Acad Sport Med.* 2002 May;12(3):139-50.
36. Staples JR, Clement DB, Taunton JE, McKenzie DC. Effects of hyperbaric oxygen on a human model of injury. *Am J Sports Med.* 1999;27(5):600-5.
37. Harrison BC, Robinson D, Davison BJ, Foley B, Seda E, Byrnes WC. Treatment of exercise-induced muscle injury via hyperbaric oxygen therapy. *Med Sci Sports Exerc.* 2001 Jan;33(1):36-42.
38. Babul S, Rhodes EC, Taunton JE, Lepawsky M. Effects of intermittent exposure to hyperbaric oxygen for the treatment of an acute soft tissue injury. *Clin J Sport Med Off J Can Acad Sport Med.* 2003 May;13(3):138-47.

39. Presti N, Huang E, Pryor JL, Hostler D. Effects of hyperbaric oxygen therapy on exercise-induced muscle damage. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc.* 2022;49(3):315-27.
40. Chen CY, Chou WY, Ko JY, Lee MS, Wu RW. Early Recovery of Exercise-Related Muscular Injury by HBOT. *BioMed Res Int.* 2019;2019:6289380.
41. Bennett M, Best TM, Babul S, Taunton J, Lepawsky M. Hyperbaric oxygen therapy for delayed onset muscle soreness and closed soft tissue injury. *Cochrane Database Syst Rev.* 2005 Oct 19;2005(4):CD004713.
42. Ishii Y, Ushida T, Tateishi T, Shimojo H, Miyanaga Y. Effects of different exposures of hyperbaric oxygen on ligament healing in rats. *J Orthop Res Off Publ Orthop Res Soc.* 2002 Mar;20(2):353-6.
43. Horn PC, Webster DA, Amin HM, Mascia MF, Werner FW, Fortino MD. The effect of hyperbaric oxygen on medial collateral ligament healing in a rat model. *Clin Orthop.* 1999 Mar;(360):238-42.
44. Mashitori H, Sakai H, Koibuchi N, Ohtake H, Tashiro T, Tamai K, et al. Effect of hyperbaric oxygen on the ligament healing process in rats. *Clin Orthop.* 2004 Jun;(423):268-74.
45. Soolsma SJ. The effect of intermittent hyperbaric oxygen on short term recovery from grade II medial collateral ligament injuries. 1996 [cited 2024 May 6]; Available from: <https://doi.library.ubc.ca/10.14288/1.0077081>
46. Takeyama N, Sakai H, Ohtake H, Mashitori H, Tamai K, Saotome K. Effects of hyperbaric oxygen on gene expressions of procollagen, matrix metalloproteinase and tissue inhibitor of metalloproteinase in injured medial collateral ligament and anterior cruciate ligament. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA.* 2007 Apr;15(4):443-52.
47. Yeh WL, Lin SS, Yuan LJ, Lee KF, Lee MY, Ueng SWN. Effects of hyperbaric oxygen treatment on tendon graft and tendon-bone integration in bone tunnel: biochemical and histological analysis in rabbits. *J Orthop Res Off Publ Orthop Res Soc.* 2007 May;25(5):636-45.
48. Vanhoenacker FM, Snoeckx A. Bone marrow edema in sports: general concepts. *Eur J Radiol.* 2007 Apr;62(1):6-15.
49. Hofmann S, Engel A, Neuhold A, Leder K, Kramer J, Plenk H. Bone-marrow oedema syndrome and transient osteoporosis of the hip. An MRI-controlled study of treatment by core decompression. *J Bone Joint Surg Br.* 1993 Mar;75(2):210-6.
50. Malizos KN, Karantanas AH, Varitimidis SE, Dailiana ZH, Bargiotas K, Maris T. Osteonecrosis of the femoral head: etiology, imaging and treatment. *Eur J Radiol.* 2007 Jul;63(1):16-28.
51. Ververidis AN, Paraskevopoulos K, Keskinis A, Ververidis NA, Molla Moustafa R, Tilkeridis K. Bone marrow edema syndrome/transient osteoporosis of the hip joint and management with the utilization of hyperbaric oxygen therapy. *J Orthop.* 2020 Nov;22:29-32.
52. Aigner N, Petje G, Schneider W, Meizer R, Wlk M, Kotsaris S, et al. Bone marrow edema syndrome of the femoral head: treatment with the prostacyclin analogue iloprost vs. core decompression: an MRI-controlled study. *Wien Klin Wochenschr.* 2005 Feb;117(4):130-5.
53. Gardin C, Bosco G, Ferroni L, Quartesan S, Rizzato A, Tatullo M, et al. Hyperbaric Oxygen Therapy Improves the Osteogenic and Vasculogenic Properties of Mesenchymal Stem Cells in the Presence of Inflammation In Vitro. *Int J Mol Sci.* 2020 Feb 20;21(4):1452.
54. Bennett M. Hyperbaric oxygen therapy improved both pain scores and range of motion in patients with early idiopathic femoral head necrosis (Ficat stage II). *Diving Hyperb Med.* 2011 Jun;41(2):105.
55. Camporesi EM, Vezzani G, Zanon V, Manelli D, Enten G, Quartesan S, et al. Review on hyperbaric oxygen treatment in femoral head necrosis. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc.* 2017;44(6):497-508.
56. Camporesi EM, Vezzani G, Bosco G, Mangar D, Bernasek TL. Hyperbaric Oxygen Therapy in Femoral Head Necrosis. *J Arthroplasty.* 2010 Sep;25(6):118-23.
57. Hsu SL, Wang CJ, Lee MSS, Chan YS, Huang CC, Yang KD. Cocktail therapy for femoral head necrosis of the hip. *Arch Orthop Trauma Surg.* 2010 Jan;130(1):23-9.
58. Li W, Ye Z, Wang W, Wang K, Li L, Zhao D. Clinical effect of hyperbaric oxygen therapy in the treatment of femoral head necrosis: A systematic review and meta-analysis. *Orthopade.* 2017 May;46(5):440-6.
59. Reis ND, Schwartz O, Militianu D, Ramon Y, Levin D, Norman D, et al. Hyperbaric oxygen therapy as a treatment for stage-I avascular necrosis of the femoral head. *J Bone Joint Surg Br.* 2003 Apr;85(3):371-5.
60. Salameh M, Moghamis IS, Kokash O, Ahmed GO. Hyperbaric oxygen therapy for the treatment of Steinberg I and II avascular necrosis of the femoral head: a report of fifteen cases and literature review. *Int Orthop.* 2021 Oct 1;45(10):2519-23.
61. Moghamis I, Alhammoud AA, Kokash O, Alhaneedi GA. The outcome of hyperbaric oxygen therapy versus core decompression in the non-traumatic avascular necrosis of the femoral head: Retrospective Cohort Study. *Ann Med Surg.* 2021 Feb 1;62:450-4.