EXPERIMENTAL STUDY

The effect of quercetin on ischemia-reperfusion injury in skeletal muscle in rats

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ABSTRACT

BACKGROUND: Ischemia-reperfusion (I/R) injury of the lower limbs is a significant clinical challenge that can arise due to surgical procedures, thrombotic events, embolism, or traumatic vascular damage. This study aimed to evaluate the antioxidative and histopathological protective effects of quercetin, a potent flavonoid antioxidant, on skeletal muscle subjected to I/R injury.

METHODS: Eighteen Wistar Albino rats were randomly assigned into three groups: Control (sham laparotomy), Ischemia-Reperfusion (IR) group (2 hours of ischemia followed by 2 hours of reperfusion), and Ischemia-Reperfusion plus quercetin treatment (IR-Q) group, receiving 20 mg/kg quercetin intraperitoneally 30 minutes before ischemia induction. After the experimental protocols, skeletal muscle samples were collected for biochemical assays measuring malondialdehyde (MDA) levels and superoxide dismutase (SOD) activity, as well as for histopathological examination.

RESULTS: The IR group demonstrated a significant increase in MDA concentration compared to controls ($p<0.0001$), whereas administration of quercetin in the IR-Q group significantly attenuated MDA levels relative to the untreated IR group ($p=0.012$). SOD activity was markedly diminished in the IR group ($p<0.0001$) but was significantly restored in the IR-Q group compared to IR alone ($p=0.012$). Histological analyses revealed pronounced muscle atrophy, degeneration, leukocyte infiltration, and fiber fragmentation/hyalinization in the IR group, which were significantly alleviated by quercetin treatment ($p<0.05$).

CONCLUSION: These findings indicate that quercetin exerts a protective effect against oxidative stress and structural damage induced by ischemia-reperfusion in skeletal muscle, potentially through enhancement of endogenous antioxidant defenses. Quercetin thus holds promise as a therapeutic agent in mitigating I/R injury; however, further studies are needed to elucidate its precise mechanisms and clinical applicability.

Keywords: Ischemia-reperfusion; quercetin; oxidative stress; superoxide dismutase; malondialdehyde; skeletal muscle.

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INTRODUCTION

Ischemia occurs when blood flow to tissues is insufficient, causing a lack of oxygen delivery that disrupts normal cellular activities and metabolism.[1] The subsequent restoration of blood flow, termed reperfusion, paradoxically may lead to further damage known as ischemia-reperfusion (I/R) injury. This injury involves complex biochemical and cellular events such as oxidative stress, inflammation, and tissue destruction. During ischemia, reduced ATP synthesis, accumulation of metabolic byproducts, and disruption of ionic balance impair cellular function. When oxygen supply is suddenly reinstated during reperfusion, excessive production of reactive oxygen species (ROS) is triggered, initiating oxidative damage and activating inflammatory pathways.[2,3] These processes cause oxidative modifications, including lipid membrane degradation, protein oxidation, and DNA strand breaks, which collectively contribute to cell death and exacerbate tissue injury.[4]

I/R injury of the lower extremities can be caused by a range of clinical conditions, including atherosclerosis, thrombotic or embolic arterial occlusions, traumatic vascular injuries, and surgical interventions. Thrombus or embolus formation leads to vascular blockage, while direct trauma to blood vessels may similarly result in ischemia. Clinically, I/R injury is often observed during procedures such as aortic aneurysm repair requiring cross-clamping, peripheral vascular surgeries, free flap tissue transfers, orthopedic operations, and shock states with compromised circulation.[5,6] Moreover, prolonged external pressure or the use of tourniquets can also precipitate ischemic conditions.

Ischemia-reperfusion (I/R) injury is not confined to local tissue damage but can also trigger systemic effects impacting vital organs such as the heart, lungs, kidneys, and brain.[7] The reperfusion phase initiates a cascade of harmful events, including the overproduction of reactive oxygen species (ROS), disruption of endothelial function, infiltration of inflammatory leukocytes, and secretion of proinflammatory cytokines, collectively intensifying tissue damage.[8] Central to these processes are key mediators such as tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β), as well as adhesion molecules including intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1). These molecules facilitate leukocyte adhesion and extravasation, elevate vascular permeability, promote edema development, and drive cellular apoptosis and necrosis in affected tissues.[9]

Antioxidants serve as crucial modulators in counteracting the harmful effects of I/R injury.[10] Among them, quercetin-a flavonoid compound naturally found in many plants-has garnered attention due to its strong antioxidant and anti-inflammatory effects.[11] It protects cells by reducing lipid peroxidation, boosting endogenous antioxidant enzyme activities, and suppressing the release of proinflammatory mediators such as TNF-α and IL-1β.[12,13] At the cellular level, quercetin supports mitochondrial function, sustains energy metabolism, and inhibits pathways leading to apoptosis. Additionally, it impedes neutrophil infiltration by modulating interactions between neutrophils and endothelial cells.[14] Other compounds such as melatonin, mannitol, allopurinol, and N-acetylcysteine have also shown efficacy in limiting oxidative damage in various I/R experimental setups.[15-20]

Quercetin is widely present in numerous fruits and vegetables, including onion (Allium cepa), apple (Malus domestica), grape (Vitis vinifera), cherry (Prunus avium), green tea (Camellia sinensis), and broccoli (Brassica oleracea).[21]

This study aims to investigate the protective role of quercetin against ischemia-reperfusion injury in rat skeletal muscle by evaluating biochemical oxidative stress parameters, inflammatory responses, and histopathological alterations.

MATERIALS AND METHODS

Ethical Approval and Experimental Animals

Approval for this research was granted by the Local Ethics Committee for Animal Experiments at Gazi University (Protocol No: GÜET-16.066; approved on July 13, 2016). All experimental protocols were carried out in accordance with the guidelines provided by the U.S. National Institutes of Health regarding the care and use of laboratory animals and conformed to the ethical standards outlined in the Declaration of Helsinki.

Eighteen adult male Wistar Albino rats weighing between 200 and 250 grams were housed under standardized laboratory conditions, maintained at 20-21°C with a 12-hour light/dark cycle. Animals had unrestricted access to standard chow and water, except for a fasting period of two hours prior to anesthesia. The rats were randomly divided into three groups of six animals each. Anesthesia was induced by intramuscular administration of ketamine (100 mg/kg) before surgical procedures.

Experimental Design

The study groups were organized as follows:

- **Group K (Control):** Rats underwent only midline laparotomy without any additional intervention. After a 4-hour observation period, skeletal muscle samples were collected, and the animals were euthanized.

- **Group IR (Ischemia-Reperfusion):** Following laparotomy, ischemia was induced by clamping the infrarenal abdominal aorta for two hours, followed by two hours of reperfusion. Skeletal muscle tissues were harvested after reperfusion, and the animals were sacrificed.

- **Group IR-Q (Ischemia-Reperfusion + Quercetin):** This group underwent the same ischemia-reperfusion protocol as Group IR. Additionally, quercetin (20mg/kg; Sigma–Aldrich, Q4951-10G) was administered intraperitoneally 30 minutes before ischemia onset. Tissue collection was performed after the reperfusion period.

At the conclusion of the experiments, intracardiac blood samples (up to 10mL) and skeletal muscle specimens were obtained for subsequent biochemical and histological analyses.[22]

Aortic Occlusion and Ischemia-Reperfusion Procedure

Anesthesia was induced with ketamine hydrochloride (50mg/kg, intramuscular; Ketalar, Parke-Davis Eczacıbaşı, Istanbul, Türkiye) combined with xylazine hydrochloride (2%, intramuscular; Alfazyne, Ege Vet, Izmir, Türkiye), with supplemental doses administered as needed.

Rats were placed in a supine position beneath a warming lamp. After shaving and aseptic preparation of the abdominal area, a midline laparotomy was performed. The abdominal aorta was carefully exposed and occluded using a non-traumatic microvascular clamp. Successful occlusion was verified by the absence of a palpable distal arterial pulse. To preserve body temperature and fluid homeostasis, the incision was covered with sterile plastic.

Following 120 minutes of ischemia, the clamp was removed, allowing reperfusion for an additional 120 minutes. Animals were euthanized under deep anesthesia at the end of reperfusion, and skeletal muscle samples were collected.

Biochemical Analyses

Skeletal muscle samples were rinsed with cold (4°C) deionized water to eliminate blood residues, then homogenized at 1000rpm for three minutes using a Heidolph Diax 900 homogenizer (Germany). Homogenates were centrifuged at 10,000×g for 60 minutes, and the resulting supernatant was used for assays.

- **Superoxide Dismutase (SOD) Activity:** Evaluated by monitoring the inhibition of nitroblue tetrazolium (NBT) reduction at an absorbance of 560nm, following the method described in.[23] One unit of SOD activity is defined as the amount of enzyme that inhibits 50% of the NBT reduction. Results are presented as units per milligram of protein (U/mg protein).

- **Malondialdehyde (MDA) Levels:** Quantified using the thiobarbituric acid reactive substances (TBARS) assay, according to Van Ye et al.[24] Tissue homogenates were precipitated with 20% trichloroacetic acid and centrifuged. The resulting supernatant was incubated with 0.6% thiobarbituric acid, boiled for 30 minutes, and then cooled. Absorbance was measured at 532nm using a Shimadzu UV/VIS-1601 spectrophotometer (Japan). MDA concentrations were determined in nmol/mg protein, calculated against a standard curve generated from 1,1,3,3-tetramethoxypropane. Protein content was measured via the Lowry method, employing bovine serum albumin as the standard.[25]

Histopathological Evaluation

Twenty-five skeletal muscle samples were fixed in 10% neutral-buffered formalin, subjected to dehydration and clearing using xylene, and subsequently embedded in paraffin blocks. Serial sections, each 4µm thick, were prepared and stained with hematoxylin and eosin (H&E).

The histological assessment targeted features including muscle fiber atrophy and hypertrophy, degeneration, vascular congestion, nuclear internalization, leukocyte infiltration, as well as fiber fragmentation and hyalinization. The standard H&E staining protocol involved hematoxylin application for 3 minutes, rinsing with tap water, differentiation in acid-alcohol solution, eosin staining for 10 minutes, followed by dehydration and mounting of the slides.

Statistical Analysis

Data analysis was conducted using SPSS software version 20.0 (SPSS Inc., Chicago, IL, USA). Statistical significance was set at $p<0.05$. Biochemical results are presented as mean±standard deviation (SD), whereas histopathological data are reported as median (25th–75th percentile). The normality of data distribution was evaluated using the Shapiro-Wilk test. Group comparisons were performed using the Kruskal-Wallis test, with post hoc pairwise comparisons conducted using Bonferroni-adjusted Mann–Whitney U tests.

RESULTS

Histopathological Evaluation

The histopathological parameters-including muscle atrophy/hypertrophy, muscle degeneration/congestion, nuclear internalization (oval or central nuclei), leukocyte infiltration, and fiber fragmentation/hyalinization-showed statistically significant differences among the groups ($p=0.012$, $p<0.0001$, $p=0.001$, $p=0.002$, and $p=0.020$, respectively) (Table 1, Figures 1-6).

Table 1. Histopathological findings of rat skeletal muscle tissue [Mean±SD]

Histopathological Parameters	Control Group (K) (n=6)	Ischemia-Reperfusion Group (IR) (n=6)	Ischemia-Reperfusion + Quercetin Group (IR-Q) (n=6)	p-value
Muscle Atrophy/Hypertrophy	0.00±0.00 *	1.33±0.42	0.50±0.22 *	0.012
Muscle Degeneration/Congestion	0.33±0.21 *	2.17±0.31	0.67±0.21 *	<0.0001
Nuclear Internalization (Oval/Central Nuclei)	0.00±0.00 *	1.50±0.22	0.83±0.31 *, &	0.001
Fragmentation/Hyalinization	0.33±0.21 *	1.83±0.31	0.67±0.21 *	0.002
Leukocyte Infiltration	0.17±0.17 *	1.17±0.31	0.33±0.21 *	0.020

p: Statistical significance was determined using the Kruskal-Wallis test ($p<0.05$). *$p<0.05$:Compared to the IR group. & $p<0.05$: Compared to the Control (K) group.

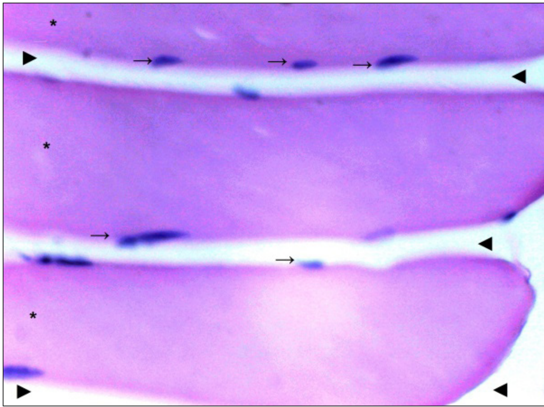

Figure 1. Longitudinal section of skeletal muscle in the Control group showing normal morphology (H&E: hematoxylin and eosin, magnification ×100). (→ peripheral flat nuclei, * muscle fibers: myofibrils, ► intercellular space).

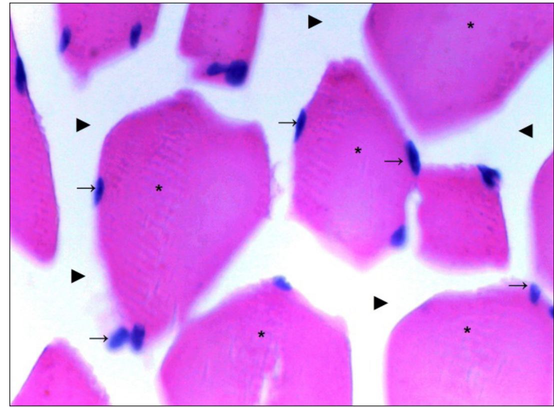

Figure 2. Cross-sectional view of skeletal muscle in the Control group demonstrating normal morphology (H&E: hematoxylin and eosin, magnification ×100). (→ peripheral flat nuclei, *muscle fibers: myofibrils, ► intercellular space).

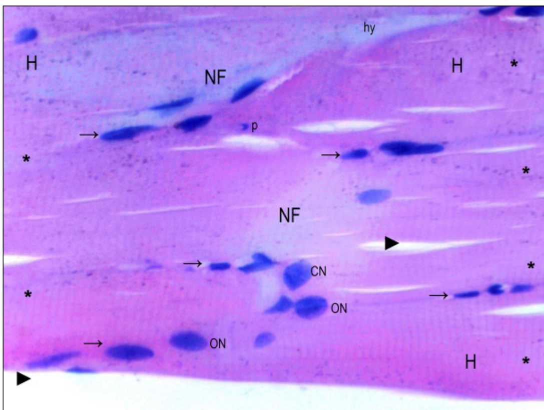

Figure 3. Longitudinal light microscopic view of skeletal muscle in the IR group (H&E: hematoxylin and eosin, magnification ×100).(→ peripheral flat nuclei, * muscle fibers: myofibrils, H: hypertrophy in muscle fibers, NF: necrotic fiber area, CN: central nucleus, ON: oval nucleus, f: fragmentation, hy: hyalinization areas, p: pyknotic nucleus).

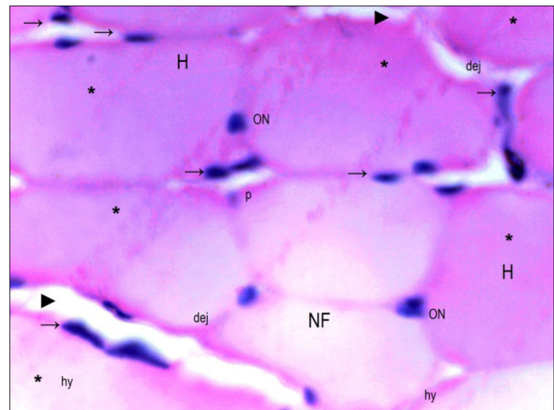

Figure 4. Transverse light microscopic view of skeletal muscle in the IR group (H&E: hematoxylin and eosin, magnification ×100). (→ peripheral flat nuclei, * muscle fibers: myofibrils, ► intercellular space; H: hypertrophy in muscle fibers, NF: necrotic fiber area, CN: central nucleus, ON: oval nucleus, f: fragmentation, hy: hyalinization areas, p: pyknotic nucleus, dej: degeneration).

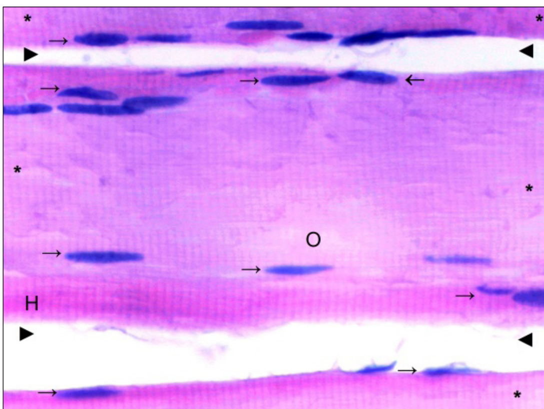

Figure 5. Longitudinal light microscopic view of skeletal muscle in the IR-Quercetin group (H&E: hematoxylin and eosin, magnification ×100). (→ peripheral flat nuclei, * muscle fibers: myofibrils, ► intercellular space; H: hypertrophy in muscle fibers, hy: hyalinization, O: edema).

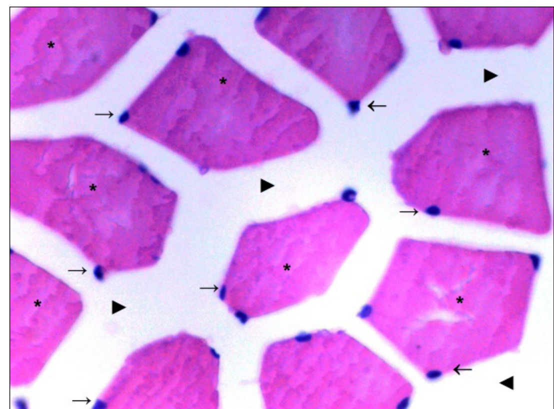

Figure 6. Transverse light microscopic view of skeletal muscle in the IR-Quercetin group (H&E: hematoxylin and eosin, magnification ×100). (→ peripheral flat nuclei, * muscle fibers: myofibrils, ► intercellular space).

Table 2. Biochemical data of skeletal muscle tissue [Mean±Standard Deviation]

Parameter	Group K (n=6)	Group IR (n=6)	Group IR-Q (n=6)	p-value **
MDA (nmol/mg protein)	21.13± 3.00	53.33±7.66*	24.11±3.19&	0.001
SOD (U/mg protein)	11.47±1.10	6.07±1.11*	10.28±0.93&	0.006

K: Control, I/R: Ischemia/Reperfusion, IR-Q: Ischemia-Reperfusion + Quercetin, MDA: Malondialdehyde, SOD: Superoxide Dismutase.

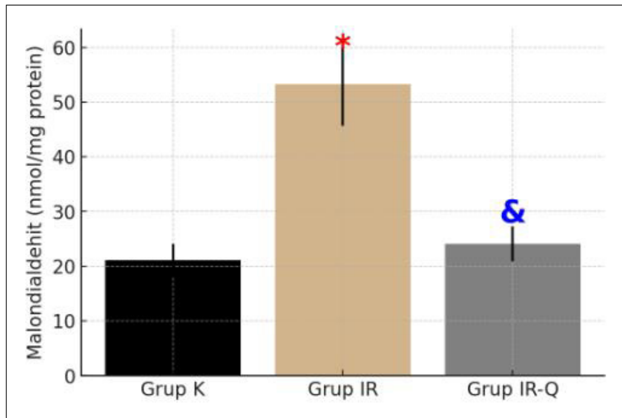

Figure 7. Malondialdehyde (MDA) levels in skeletal muscle tissue.

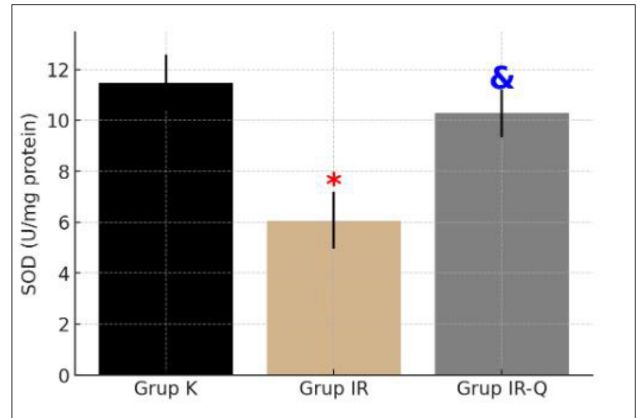

Figure 8. Superoxide dismutase (SOD) levels in skeletal muscle tissue.

Muscle atrophy/hypertrophy was significantly higher in the IR group compared to both the Control (K) and IR-Q groups (p=0.004 and p=0.049, respectively). Muscle degeneration/congestion was also significantly elevated in the IR group compared to the K and IR-Q groups (p<0.0001 and p=0.001, respectively). Nuclear internalization (oval or central nuclei) was significantly greater in the IR group compared to the K and IR-Q groups (p<0.0001 and p=0.048, respectively). Furthermore, the IR-Q group showed a significant increase in nuclear internalization compared to the K group (p=0.017). Leukocyte infiltration was significantly higher in the IR group than in the K and IR-Q groups (p=0.009 and p=0.025, respectively). Fiber fragmentation/hyalinization was significantly more pronounced in the IR group compared to the K and IR-Q groups (p=0.002 and p=0.014, respectively) (Table 1, Figures 1-6).

Biochemical Evaluation

Table 2 presents the MDA levels and SOD enzyme activities in skeletal muscle tissue across the groups. The results demonstrated that the MDA level in the IR group was significantly higher compared to the control group (p<0.0001). In the IR-Q group, the MDA level was significantly lower than in the IR group (p=0.012) (Figure 7).

SOD enzyme activity was significantly decreased in the IR group compared to the control group (p<0.0001 and p<0.001, respectively). In the IR-Q group, SOD enzyme activity was markedly higher compared to the IR group (p<0.001 and p=0.012, respectively) (Figure 8).

Malondialdehyde; SOD: Superoxide Dismutase. *p<0.05 compared to Group K; &p<0.05 compared to Group IR.)

DISCUSSION

Lower extremity ischemia may develop as a consequence of various clinical conditions, such as peripheral arterial disease, thromboembolism, traumatic vascular injury, and surgical interventions. Although the restoration of blood flow (revascularization) is vital, the reperfusion phase may exacerbate tissue injury, as it triggers a series of complex biochemical events that cause complications both in local muscle tissue and at the systemic level.[26,27] One of the main contributors to this damage is the generation of reactive oxygen species (ROS) and the subsequent inflammatory response, which can progress to multi-organ dysfunction.[28]

ROS are highly reactive molecules that initiate lipid peroxidation, disrupt cell membranes, and enhance neutrophil activation, thereby intensifying inflammation.[29] Thus, controlling oxidative stress is critical for reducing ischemia-reperfusion (I/R) injury. Enzymatic antioxidants such as superoxide dismutase (SOD) and glutathione peroxidase (GPx) constitute the first line of defense against oxidative damage in the body. Various pharmacological and immunological agents have been tested in experimental models to mitigate I/R injury. In this

study, the protective effect of quercetin-a natural flavonoid compound-was investigated in a rat skeletal muscle I/R model induced by abdominal aortic clamping.

Following ischemia, reperfusion leads to an abrupt increase in ROS production. In particular, superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (OH^-) target cellular structures and trigger cell death through both apoptosis and necrosis.[30] Lipid peroxidation disrupts phospholipid integrity, increases membrane permeability, and disturbs ion homeostasis. Protein oxidation weakens enzymatic functionality, while DNA damage elevates mutation risk and activates cellular stress responses.[31-33]

Oxidative stress not only initiates tissue damage but also amplifies inflammation. Lipid peroxidation products act as danger signals, promoting the release of pro-inflammatory cytokines and accelerating neutrophil migration, thereby exacerbating tissue injury. Additionally, ROS-induced mitochondrial dysfunction impairs ATP production and increases cellular vulnerability.[34-37]

Among the organism's antioxidant defense mechanisms, SOD, catalase (CAT), and GPx play major roles. SOD converts superoxide into hydrogen peroxide, thereby reducing oxidative burden. However, under I/R conditions, SOD activity decreases due to the inhibitory effects of ROS and disruptions in intracellular homeostasis.[38-39] Malondialdehyde (MDA), a reliable biochemical marker of lipid peroxidation, was used in this study to assess the degree of oxidative damage.[38,40-42]

Currently, there is no clinically effective method capable of completely preventing I/R-induced muscle injury.[43] While pharmacological treatments, hyperbaric oxygen therapy, hypothermia, and pre/post-ischemic conditioning offer partial benefits,[32,33,44] experimental studies have demonstrated protective effects of compounds such as crocin, melatonin, lycopene, and ticlopidine against lower extremity I/R injury.[45-48]

Quercetin is a polyphenolic flavonoid abundantly found in fruits and vegetables and is recognized for its potent antioxidant, anti-inflammatory, and cytoprotective properties.[49] It exerts its effects through free radical scavenging, modulation of enzymatic activities, and suppression of pro-inflammatory cytokine release.[50-52] For example, Chen et al.[53] reported that quercetin protects cardiomyocytes against I/R injury by regulating kinases such as Src, FAK, p38, and STAT3. Similarly, Sul and colleagues demonstrated that quercetin decreases ROS levels and NOX2 expression in LPS-exposed pulmonary epithelial cells, thereby reducing oxidative stress and inflammation.[54] Its antiviral, anticancer, anti-inflammatory, and metabolic regulatory properties are also well documented.[55-57] Furthermore, Lin et al.[58] showed that quercetin attenuates hepatic I/R injury by inhibiting GSDMD-mediated macrophage pyroptosis in an experimental model.

Although the protective effects of quercetin on various organ systems have been reported, its specific effects on lower extremity skeletal muscle have not been sufficiently investigated. In the present study, a significant improvement was observed in oxidative stress markers following I/R. The reduced SOD activity in the IR group increased significantly in the quercetin-treated IR-Q group, indicating enhanced antioxidant defense. Moreover, MDA levels, which were highest in the IR group, were significantly reduced in the IR-Q group, reflecting preserved membrane integrity. These findings highlight the crucial role of quercetin in ROS neutralization and the maintenance of redox balance.

Histopathological evaluations supported these biochemical findings. In the IR group, pronounced signs of muscle structure deterioration-including atrophy, hypertrophy, degeneration, congestion, and nuclear internalization-were observed, whereas quercetin treatment markedly reduced these alterations. In particular, the reduction in leukocyte infiltration, as well as decreased fiber fragmentation and hyalinization, demonstrates quercetin's tissue-protective effects through the suppression of inflammation. Considering that nuclear internalization is regarded as a marker of muscle regeneration and cellular stress, its significant reduction in the IR-Q group indicates the restorative potential of quercetin.

CONCLUSION

One of the key strengths of this study is the use of a well-established ischemia-reperfusion model under strictly controlled laboratory settings. Additionally, the combination of biochemical and histopathological analyses allowed for a comprehensive assessment of quercetin's protective effects. However, some limitations must be noted. This experimental setup focused solely on the acute phase of ischemia-reperfusion injury without exploring potential long-term outcomes. Furthermore, only a single dose of quercetin was tested, leaving the dose–response relationship unexamined.

In summary, our results demonstrate that quercetin provides significant protection against ischemia-reperfusion-induced skeletal muscle damage. Its antioxidant capabilities played an important role in reducing oxidative stress and preserving tissue structure. These findings suggest that quercetin holds promise as a therapeutic agent for preventing ischemic muscle injury. Nevertheless, further investigations involving various dosing regimens and long-term evaluations in preclinical and clinical trials are necessary before clinical application can be considered.

Ethics Committee Approval: This study was approved by the Gazi University Ethics Committee (Date: 13.07.2016, Decision No: GÜET-16.066).

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept: M.K., M.A.; Design: M.K., A.Ö.; Supervision: M.A., A.K.; Resource: M.K., Y.K.; Materials: Ö.E., M.Ka, A.C.B.; Data collection and/or processing: M.K., A.Ö.; Analysis and/or interpretation: A.C.B., M.Ka., Ö.E.; Literature review: L.O., G.K.; Writing: L.O., G.K.; Criti-

cal review: M.A., L.O., M.K.

Conflict of Interest: None declared.

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DENEYSEL ÇALIŞMA - *ÖZ*

Sıçanlarda iskelet kasında iskemi-reperfüzyon hasarında kuersetinin koruyucu rolü

AMAÇ: Alt ekstremite iskemi-reperfüzyon (İ/R) hasarı, cerrahi müdahaleler, tromboembolik olaylar veya travmatik vasküler lezyonlar sonrası ortaya çıkan ciddi bir patofizyolojik durumdur. Bu çalışmanın amacı, güçlü antioksidan özelliklere sahip flavonoid kuersetinin, İ/R sürecinde iskelet kasında oluşan oksidatif stres ve histopatolojik değişiklikler üzerindeki koruyucu etkilerini değerlendirmektir.

GEREÇ VE YÖNTEM: On sekiz Wistar Albino sıçan, kontrol (sahte laparotomi), iskemi-reperfüzyon (İR; 2 saat iskemi + 2 saat reperfüzyon) ve İR + kuersetin (İR-K; iskemi öncesi 30 dakika 20 mg/kg intraperitoneal kuersetin) olmak üzere üç gruba randomize edildi. Deneysel uygulamalar sonrası iskelet kası dokuları, malondialdehit (MDA) düzeyleri ve süperoksit dismutaz (SOD) aktivitesi açısından biyokimyasal olarak analiz edildi; ayrıca histopatolojik incelemeler gerçekleştirildi.

BULGULAR: İR grubunda MDA seviyeleri kontrol grubuna kıyasla anlamlı derecede artarken ($p<0.0001$), kuersetin uygulanan İR-K grubunda bu artış anlamlı ölçüde azaldı ($p=0.012$). SOD aktivitesi İR grubunda belirgin şekilde düşerken ($p<0.0001$), İR-K grubunda anlamlı bir restorasyon gözlendi ($p=0.012$). Histopatolojik değerlendirmelerde İR grubunda kas liflerinde atrofi, dejenerasyon, lökosit infiltrasyonu ve lif parçalanması/hiyalinizasyonun belirgin olduğu; kuersetin tedavisi ile bu patolojik değişikliklerin anlamlı ölçüde azaldığı tespit edildi ($p<0.05$).

SONUÇ: Kuersetin, iskemi-reperfüzyon hasarına bağlı oksidatif stres ve doku hasarını azaltarak iskelet kasında endojen antioksidan savunma mekanizmalarını güçlendirmektedir. Bu bulgular, kuersetinin İ/R kaynaklı doku hasarını önlemede potansiyel bir terapötik ajan olduğunu göstermektedir. Mekanizmalarının ayrıntılı incelenmesi ve klinik uygulama olanaklarının değerlendirilmesi için ileri preklinik araştırmalara ihtiyaç duyulmaktadır.

Anahtar sözcükler: İskemi-reperfüzyon; quercetin; oksidatif stres; süperoksit dismutaz; malondialdehit; iskelet kası.

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