International Journal of Sport Studies for Health

Journal Homepage

The Effect of Two Weeks of L-Carnitine Supplementation on Muscle Damage Markers Following Intense Aerobic Activity in Female Karate Players

Zohreh. Torab Beigi[1], Saeed. Ilbeigi[2*], Marziyeh. Saghebjoo[3], Mozhgan. Jafari[1]

[1] Department of Sport Sciences, Faculty of Sport Sciences, University of Birjand, Birjand, Iran
[2] Associate Professor of Sport Biomechanics, Department of Sport Sciences, Faculty of Sport Sciences, University of Birjand, Birjand, Iran.
[3] Professor of Exercise Physiology, Department of Sport Sciences, Faculty of Sport Sciences, University of Birjand, Birjand, Iran.

*** Corresponding author email address**: silbeigi@birjand.ac.ir

A r t i c l e I n f o

Article type:
Original Research

How to cite this article:
Torab Beigi, Z., Ilbeigi, S., Saghebjoo, M., & Jafari, M. (2025). The Effect of Two Weeks of L-Carnitine Supplementation on Muscle Damage Markers Following Intense Aerobic Activity in Female Karate Players. *International Journal of Sport Studies for Health*, *8*(3), 1-8.
http://dx.doi.org/10.61838/kman.intjssh.8.3.10

A B S T R A C T

Objective: This study investigated the effect of two weeks of L-carnitine Supplementation on muscle damage markers, specifically creatine kinase (CK) and lactate dehydrogenase (LDH), following intense aerobic activity in female karate athletes.

Methods and Materials: This experimental study employed a repeated-measures design with a control group. Twenty-one female karate athletes from Mashhad were selected based on inclusion criteria and randomly assigned to either the L-carnitine or placebo group. For 14 days, the L-carnitine group received 2 grams of L-carnitine daily in capsule form, while the placebo group received 2 grams of starch capsules. On day 15, participants underwent a fasting blood draw before performing an endurance running test. Subsequent blood samples were collected at 5 minutes, 2 hours, and 24 hours' post-endurance test. Data analysis was performed using repeated-measures ANOVA with SPSS version 24, with a significance level set at $p<0.05$.

Findings: The results indicated a significant increase in CK and LDH levels immediately after activity compared to immediately before activity within the L-carnitine group ($p<0.05$). No other significant differences were observed in subsequent pairwise comparisons within this group. Conversely, the placebo group showed significant increases in CK and LDH levels immediately, 2 hours, and 24 hours after activity compared to two weeks prior to the activity ($p<0.05$). Furthermore, a significant difference was observed between the L-carnitine and placebo groups for CK and LDH levels immediately, 2 hours, and 24 hours after activity, with significantly higher values in the placebo group.

Conclusion: These findings suggest that two weeks of L-carnitine Supplementation at a dose of 2 grams may potentially help prevent further muscle breakdown up to 24 hours following intense aerobic activity.

Keywords: *L-carnitine, Creatine kinase, Lactate dehydrogenase*

Article history:
Received 26 April 2025
Revised 01 June 2025
Accepted 08 June 2025
Published online 01 July 2025

1. Introduction

For many years, exercise scientists have investigated the potential of antioxidants to counteract the effects of reactive oxygen species (ROS) during physical activity. These investigations have spanned areas such as muscle damage, muscle fatigue, lipid peroxidation, and protein damage. However, despite extensive research, there remains considerable debate among researchers regarding the efficacy of the body's endogenous antioxidant system in combating activity-induced free radicals, and the effectiveness of antioxidant dietary supplementations in mitigating exercise-induced oxidative stress and subsequent muscle damage(1). Some researchers contend that dietary antioxidant Supplementation has no impact on oxidative stress; indeed, a few studies have even reported that such supplementations can exacerbate oxidative stress. Conversely, other researchers hypothesize that the body's antioxidant system is overwhelmed during intense and prolonged activities, suggesting that antioxidant Supplementation could bolster the antioxidant defense system, thereby reducing oxidative stress and free radical production, and consequently delaying or even halting the cellular damage process (2). The majority of research, however, indicates a beneficial effect of antioxidant consumption on oxidative stress. In recent years, various nutritional supplementations have been introduced to enhance athletic performance, with L-carnitine emerging as a prominent ergogenic aid (3). L-carnitine is a potent antioxidant, structurally composed of carnitine and short-chain acylcarnitines (4). It is endogenously synthesized in the human body from the amino acids lysine and methionine. Its potential antioxidant properties include protecting cardiac endothelial cells from oxidative stress and myocardial injury (5), reducing doxorubicin-induced liver damage, treating neurological disorders, male infertility, Alzheimer's disease, and improving fatty liver disease (6). Furthermore, L-carnitine may help prevent and reduce ischemia-reperfusion injury (7). Regarding lipid profiles, Lee et al. (2016) demonstrated that daily consumption of 1000 mg of L-carnitine significantly increased high-density lipoprotein (HDL) and slightly decreased triglycerides in patients with coronary artery disease (8). Elevated HDL levels are indicative of a healthy circulatory system, while increased plasma triglycerides are associated with cardiovascular diseases (9). L-carnitine exerts its antioxidant effects by reducing the activity of superoxide dismutase (SOD) and catalase (CAT), enzymes crucial for detoxifying hydrogen peroxide (H2O2), thereby protecting body cells from oxidative stress. This supplementation also contributes to improved athletic performance by lowering malondialdehyde (MDA) levels, a recognized marker of oxidative stress. In the context of inflammation, inflammatory responses are vital for host defense and tissue homeostasis. L-carnitine aids in reducing inflammation by decreasing the production of reactive oxygen species (ROS) and inhibiting inflammatory pathways such as nuclear factor-kappa B (NF-κB) (10). A study by Asjadi et al. (2023) found that L-carnitine Supplementation prior to exercise enhanced fat oxidation more than carbohydrate oxidation in young obese men compared to normal-weight young men (11). Chow et al. (2001) reported a significant increase in plasma free fatty acid (FFA) concentrations following L-carnitine intake, which was accompanied by a considerable improvement in fat utilization during rest (12). Furthermore, Assunção et al. (2007) noted a significant increase in total antioxidant capacity (TAC) from pre-competition to immediately and one hour post-competition (13). Given the limited number of studies, there appears to be a scarcity of research specifically investigating how L-carnitine consumption influences oxidative stress induced by intense aerobic activity. Moreover, consistent and precise findings regarding the impact of L-carnitine on athletic performance in aerobic activities are lacking. Most existing studies in this area have primarily examined the effects of L-carnitine on submaximal aerobic activities. Therefore, considering these points, there is a clear need for further research to explore the antioxidant effects of L-carnitine on exercise-induced oxidative stress.

Karate is a high-intensity martial art characterized by rapid accelerations, explosive kicks, and frequent eccentric muscle contractions, all of which contribute to significant muscular fatigue and damage. Studies have demonstrated that both male and female karate athletes experience elevated levels of creatine kinase (CK) and lactate dehydrogenase (LDH) following competitive matches or intensive training sessions, reflecting increased membrane permeability and muscle fiber disruption(14, 15). In particular, female karate athletes may be more prone to post-exercise muscle damage due to physiological and hormonal differences, making them an important demographic for research into recovery strategies (15). Given the physical demands of karate and the potential protective effects of antioxidants such as L-carnitine, this study aimed to evaluate the impact of two weeks of L-carnitine supplementation on

selected muscle damage markers following intense aerobic activity in female karate practitioners.

2. Methods and Materials

2.1 Study Design and Participants

This study employed an experimental research design with repeated measures and a control group, conducted as a randomized, single-blind study. It involved two distinct groups: a placebo group and a Supplementation group. The target population for this research comprised female karate athletes residing in Mashhad. An open invitation was extended to prospective participants, requesting their presence at the Exercise Physiology Laboratory on a specified date. From the volunteers, 21 individuals who met the inclusion criteria were meticulously selected and randomly assigned to either the supplementation group (n=10) or the placebo group (n=11).

Participants were selected based on specific inclusion and exclusion criteria to ensure homogeneity within the sample and enhance the validity of the results. Female karate athletes aged between 18 and 30 years were recruited from local karate clubs in Mashhad, Iran. All participants were required to complete a written informed consent form and a Physical Activity Readiness Questionnaire (PAR-Q) to assess their eligibility. The inclusion criteria consisted of: being within the age range of 18–30 years, having at least three years of regular karate training experience, maintaining a normal body mass index (BMI) between 19 and 25 kg/m², achieving a maximal oxygen uptake (VO_2max) greater than 40 ml/kg/min as determined by the Balke treadmill test, having no history of chronic diseases or musculoskeletal injuries in the past month, not using antioxidant supplements or anti-inflammatory medications in the four weeks prior to the study, and not being pregnant or breastfeeding, which was confirmed via blood testing. Exclusion criteria included the presence of cardiovascular, metabolic, or neuromuscular disorders, use of corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), or anticoagulant medications, self-reported muscle injury or infection during the experimental period, failure to comply with the supplementation protocol or dietary instructions, and any changes to the habitual training regimen throughout the course of the study.

2.2 Executive Protocol

After providing informed consent and receiving a comprehensive briefing on the study's objectives and procedures, participants' Body Mass Index (BMI) and maximal oxygen uptake (VO_2max) were assessed using the Balke test. Prior to the commencement of supplementation and the intense aerobic activity, participants attended the laboratory for their initial blood collection, where approximately seven cubic centimeters of blood were drawn from the antecubital vein. This initial collection served as a baseline. For 14 consecutive days, participants in the supplementation group consumed 2 grams of L-carnitine daily in capsule form, while the placebo group received 2 grams of starch capsules. On day 15, a fasting blood sample was collected from all participants just before the endurance test. Following this, all participants consumed an identical, standardized breakfast to ensure nutritional consistency. Two and a half hours later, they proceeded to perform the main aerobic test. Immediately upon completing this strenuous endurance test, a third blood sample was collected. Subsequent blood collections were performed 2 hours and 24 hours (fasting) after the main protocol. Plasma separation was efficiently conducted at the Exercise Physiology Laboratory, and all collected serum and plasma samples from each participant were diligently stored at -70°C until required for creatine kinase (CK) and lactate dehydrogenase (LDH) measurements , preserving their integrity for accurate analysis.

2.3 Blood Sampling and Evaluation Analysis

Blood samples were systematically collected at critical junctures throughout the study to track physiological changes. An initial baseline blood collection was performed prior to the commencement of supplementation and the strenuous aerobic activity. On day 15, a fasting blood sample was drawn just before the endurance test. Immediately upon completing the demanding endurance test, a third blood sample was collected to assess immediate post-exercise responses. Further blood collections were precisely performed 2 hours and 24 hours (fasting) after the main protocol. Approximately seven cubic centimeters of blood were drawn from the antecubital vein for each sampling point. Following collection, plasma separation was efficiently conducted at the Exercise Physiology Laboratory. All collected serum and plasma samples from each participant were then meticulously stored at -70°C to preserve their integrity until required for analysis. Creatine

kinase (CK) and lactate dehydrogenase (LDH) levels were subsequently measured using a spectrophotometer with a photometric method, operating at a wavelength of 340 nm. For both enzymes, non-hemolyzed serum or heparinized plasma samples were specifically utilized to ensure the accuracy and reliability of the measurements

2.4 Supplementation

To ensure consistency and maximize the reliability of the findings, participants were instructed to consume the L-carnitine or placebo capsules daily at approximately the same time in the morning, within 30 minutes after breakfast, under supervision at the laboratory. Furthermore, to control for potential dietary influences on muscle damage markers, all participants were asked to maintain their habitual dietary patterns throughout the 14-day supplementation period. A standardized breakfast was provided to all participants on each testing day, including the days of blood sampling and the endurance test. Additionally, participants completed a 3-day dietary recall questionnaire before and during the supplementation period to monitor caloric intake, macronutrient distribution, and antioxidant consumption. No significant differences were observed between groups regarding total caloric intake or macronutrient composition. (16).

2.5 Exercise Interventions

The main aerobic test, central to this study, involved participants running two laps of a 7-kilometer course, totaling a demanding 14 kilometers. This intense activity was performed at each participant's maximum heart rate, which was meticulously monitored throughout the test using a heart rate monitor to ensure consistent effort levels. This strenuous endurance test was conducted two and a half hours after all participants had consumed an identical, standardized breakfast on day 15 of the study, following the 14-day Supplementation period(17). The warm-up consisted of 5 minutes of light jogging at a self-selected pace followed by dynamic stretching exercises targeting major lower limb muscle groups, including leg swings, walking lunges, high knees, and hamstring pulls. This was followed by 5 minutes of low-intensity running at approximately 40–50% of maximum heart rate to gradually elevate core temperature and prepare the musculoskeletal system for the upcoming strenuous activity. After completion of the 14-kilometer endurance run, participants engaged in a 10-minute cool-down , which included light jogging (at approximately 30–40% of maximum heart rate) for 5 minutes, followed by static stretching of the major lower body muscles (hamstrings, quadriceps, calves, and hip flexors).

2.6 Data Analysis

For the data analysis, individual participant characteristics were comprehensively analyzed using descriptive statistics, providing an initial overview of the sample. To rigorously confirm the normality of the data distribution, the Kolmogorov-Smirnov test was meticulously applied. Independent t-tests were conducted to examine baseline differences between the two groups (supplementation vs. placebo), ensuring comparability prior to the intervention. The primary method for assessing the effects of the intervention on the dependent variables was repeated-measures analysis of variance (ANOVA), which is particularly suited for designs involving multiple measurements from the same subjects over time. All statistical analyses were diligently performed using SPSS software, version 24, a widely recognized and robust statistical package. The level of significance for all tests was strictly set at $p<0.05$, indicating that a result was considered statistically significant if the probability of obtaining it by chance was less than 5%.

3. Results

Table 1 presents the descriptive statistics for age, height, weight, body mass index (BMI), and VO2max for participants in both groups.

Table 1. The descriptive statistics for age, height, weight, body mass index (BMI), and VO2max

Variable	Group	Mean ± SD
Height (cm)	Supplementation	165.2±4.1
	Placebo	164.8±3.9
Weight (kg)	Supplementation	58.5±3.2
	Placebo	59.1±3.5
Body Mass Index (kg/m^2)	Supplementation	21.4±1.2
	Placebo	21.8±1.3
Maximal Oxygen Uptake (ml/kg/min)	Supplementation	42.5±1.8
	Placebo	42.1±1.9

Table 2 shows the CK Concentration in Two Groups at Each Measurement Time

Table 2. CK and LDH Concentration (International Units/Liter) in Two Groups at Each Measurement Time (Mean±SD)

Measurement Time	CK Units/Liter		LDH Units/Liter	
	Placebo	Supplementation	Placebo	Supplementation
First Measurement (Base)	92.72±19.58	92.39±23.03	0.83±0.20	0.71±0.37
Second Measurement (PreTest)	82.71±24.13	75.95±24.69	0.82±0.10	0.79±0.25
Third Measurement (PostTest)	422.73±166.06	399.38±20.76	1.50±0.23	1.06±0.29
Fourth Measurement (2 hours after activity)	227.41±41.28	181.51±41.91	1.26±0.22	0.98±0.24
Fifth Measurement (24 hours after activity)	169.31±70.36	72.79±14.72	1.42±0.26	0.96±0.25

As shown in figure1, the Bonferroni post-hoc test revealed significant increases in creatine kinase (CK) levels in the L-carnitine group at immediate post-activity, 2 hours, and 24 hours' post-activity compared to both two weeks and immediately pre-activity measurements ($p<0.05$). Similarly, in the placebo group, CK levels significantly increased at immediate post-activity, 2 hours, and 24 hours' post-activity relative to both two weeks and immediately pre-activity measurements ($p<0.05$). Furthermore, within the placebo group, a significant increase in CK levels was observed at 24 hours' post-activity compared to immediately post-activity. Also the independent t-test comparing CK levels between the L-carnitine and placebo groups showed a significant difference in blood CK levels 24 hours after intense aerobic activity. Specifically, CK levels were significantly higher in the placebo group.

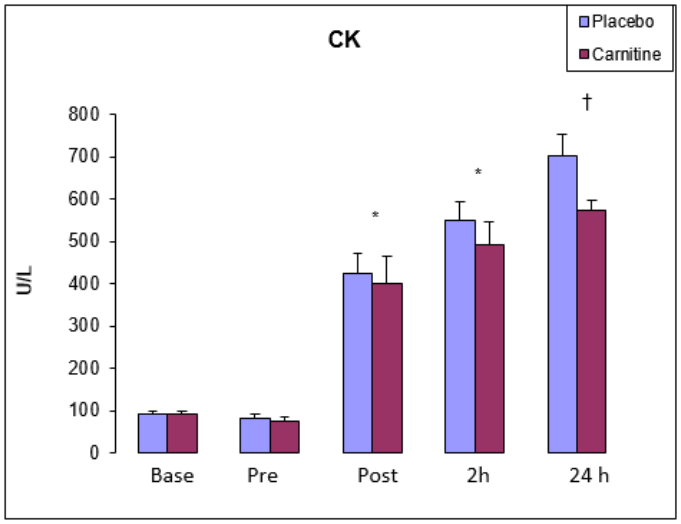

Figure 1. CK concentrations in both groups at each measurement time point

Figure 1 shows the CK concentrations in both groups at each measurement time point ($p<0.05$). The symbol ★ indicates a significant increase in CK in both groups compared to immediately pre-activity. The symbol ✝ denotes a significant increase in CK in the placebo group compared to the L-carnitine group at 24 hours' post-activity, relative to 2 hours post-activity. The time points are defined as follows: Base: 2 weeks before activity, Pre: Immediately before activity, Post: Immediately after activity, h2: 2 hours after activity, and 24 h: 24 hours after activity.

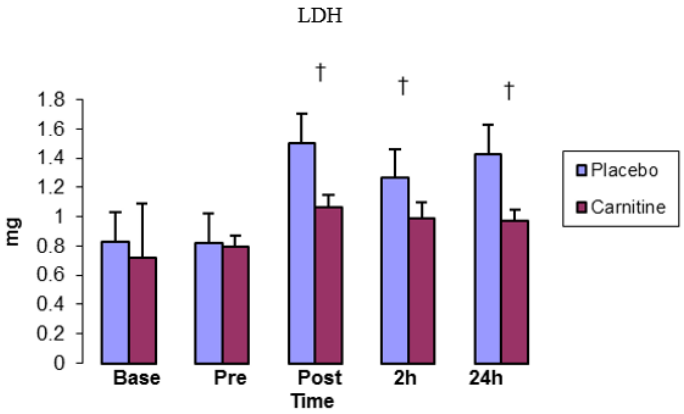

Figure 2. Bonferroni post-hoc test for LDH

Figure 2 shows the Bonferroni post-hoc test for LDH that in the L-carnitine group, lactate dehydrogenase (LDH) levels significantly increased immediately after activity compared to immediately before activity ($p<0.05$). No other significant differences were observed in subsequent pairwise comparisons within this group.

Conversely, in the placebo group, LDH levels significantly increased immediately, 2 hours, and 24 hours after activity compared to two weeks prior to the activity ($p<0.05$). Furthermore, significant increases in LDH levels were also observed in the placebo group immediately, 2 hours, and 24 hours after activity when compared to immediately before activity.

An independent t-test comparing LDH levels between the L-carnitine and placebo groups revealed significant differences in blood LDH levels immediately, 2 hours, and 24 hours after intense aerobic activity. LDH levels were significantly higher in the placebo group at these time points.

4. Discussion and Conclusion

The primary objective of the present study was to investigate the effect of two weeks of L-carnitine supplementation on creatine kinase (CK) and lactate dehydrogenase (LDH) levels following intense aerobic activity in female karate athletes. Regarding CK concentration, the findings revealed no significant difference in CK levels between the two groups before the initiation of L-carnitine or placebo consumption. Consistent with almost all prior research, blood CK levels typically increase after intense or prolonged exercise. In the current study, a significant elevation in blood CK levels was observed in both groups immediately post-activity, indicating that the exercise protocol utilized induced muscle damage and oxidative stress. Notably, 24 hours' post-activity, blood CK levels in the supplement group exhibited a reduction, whereas the placebo group still showed a significant increase compared to pre-activity levels. This finding aligns with the research by Petersen (2001) and Tsakiris et al. (2006). The observed effect of L-carnitine supplementation on blood CK levels may suggest an influence on lipid peroxidation (18, 19). Cell membrane disruption due to lipid peroxidation can lead to increased efflux of CK from cells into the bloodstream. L-carnitine has been shown to prevent the increase of malondialdehyde (MDA), a key indicator of lipid peroxidation. In essence, L-carnitine, through its antioxidant properties, mitigates exercise-induced lipid peroxidation, thereby protecting cell membranes. This protective effect consequently reduces the leakage of CK from cell membranes into the blood (20). Therefore, the observed reduction in CK levels in the supplement group 24 hours post-activity appears biologically plausible. This suggests that two weeks of L-carnitine supplementation at a dose of 2 grams may potentially prevent further muscle degradation 24 hours after activity. Concerning lactate dehydrogenase (LDH) enzyme activity, our findings indicated no significant difference in LDH activity between the two groups before L-carnitine or placebo administration. However, in the placebo group, LDH levels significantly increased immediately, 2 hours, and 24 hours after activity compared to two weeks prior to the activity. Furthermore, significant increases in LDH were observed immediately, 2 hours, and 24 hours post-activity compared to immediately pre-activity in the placebo group. Walshojerdt et al. investigated the effect of L-carnitine on reducing platelet contamination over a 5-day period. They found a significant reduction in dehydrogenase enzyme activity in the platelet environment after 5 days in L-carnitine-treated groups compared to the control group (21). Similarly, Klaus.et al (2010) observed in their research

that when cell membranes are damaged, dehydrogenase enzyme activity increases from the cytoplasm to the plasma, and the addition of L-carnitine to platelets under storage conditions leads to an improvement in platelet membrane damage (22). In the present study, the effect of L-carnitine on dehydrogenase enzyme activity was statistically significant. The results pertaining to the antioxidant protective effect of L-carnitine against the inhibition of dehydrogenase enzyme activity in platelets were consistent with findings from other studies in this field(23)

The findings of this study indicate that two weeks of L-carnitine supplementation at a dose of 2 grams per day may exert protective effects against exercise-induced muscle damage, as evidenced by significantly lower increases in creatine kinase (CK) and lactate dehydrogenase (LDH) levels following intense aerobic activity in female karate athletes compared to the placebo group. These results support the hypothesis that L-carnitine, through its antioxidant properties, can mitigate oxidative stress and subsequent cellular membrane disruption, thereby reducing the efflux of intracellular enzymes such as CK and LDH into the bloodstream

Authors' Contributions

All authors equally contributed to this study.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

Acknowledgments

We would like to express our gratitude to all individuals helped us to do the project.

Declaration of Interest

The authors report no conflict of interest.

Funding

According to the authors, this article has no financial support.

Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

References

1. Zhang D-M, Guo Z-X, Zhao Y-L, Wang Q-J, Gao Y-S, Yu T, et al. L-carnitine regulated Nrf2/Keap1 activation in vitro and in vivo and protected oxidized fish oil-induced inflammation response by inhibiting the NF-κB signaling pathway in Rhynchocypris lagowski Dybowski. Fish & shellfish immunology. 2019;93:1100-10. [DOI]
2. Pashayi P, Abbassi Daloii A, Barari AR, Mir Javadi SR. The Effect of Endurance Training and L-Carnitine Supplementation on Gene Expression of Hepatic Enzymes (AST, ALT, ALP) in Wistar Male Rats Toxicated by Boldenone. Journal of Advanced Biomedical Sciences. 2019;9(4):1710-8.
3. Khushboo M, Murthy MK, Devi MS, Sanjeev S, Ibrahim KS, Kumar NS, et al. Testicular toxicity and sperm quality following copper exposure in Wistar albino rats: ameliorative potentials of L-carnitine. Environmental Science and Pollution Research. 2018;25(2):1837-62. [PMID: 29103113] [DOI]
4. Bordoni L, Sawicka AK, Szarmach A, Winklewski PJ, Olek RA, Gabbianelli R. A pilot study on the effects of l-Carnitine and Trimethylamine-N-oxide on platelet mitochondrial DNA methylation and CVD biomarkers in aged women. International journal of molecular sciences. 2020;21(3):1047. [PMID: 32033285] [PMCID: PMC7037757] [DOI]
5. Tousson E, Keshta AT, Hussein Y, Fekry RM, Abo-Ghaneima WK. Renal protective effect of ginkgo biloba and L-carnitine extracts against pentylenetetrazol induced toxicity, oxidative stress, injury and proliferation alternation in epileptic rats. 2019. [DOI]
6. Fakourian A, Azarbayjani MA, Farzanegi P. The Effect of Aerobic Training and L-Carnitine on BCL2 and Mitochondrial Enzymes of Rat's Kidney. Iranian journal of diabetes and obesity. 2020. [DOI]
7. Gnoni A, Longo S, Gnoni GV, Giudetti AM. Carnitine in human muscle bioenergetics: can carnitine supplementation improve physical exercise? Molecules. 2020;25(1):182. [PMID: 31906370] [PMCID: PMC6982879] [DOI]
8. Lee B-J, Lin J-S, Lin Y-C, Lin P-T. Effects of L-carnitine supplementation on lipid profiles in patients with coronary artery disease. Lipids in health and disease. 2016;15:1-8. [PMID: 27317162] [PMCID: PMC4912751] [DOI]
9. Luz PLd, Favarato D, Faria-Neto Junior JR, Lemos P, Chagas ACP. High ratio of triglycerides to HDL-cholesterol predicts extensive coronary disease. Clinics. 2008;63:427-32. [PMID: 18719750] [PMCID: PMC2664115] [DOI]
10. Allen J, Sun Y, Woods JA. Exercise and the regulation of inflammatory responses. Progress in molecular biology and translational science. 2015;135:337-54. [PMID: 26477921] [DOI]
11. Asjodi F, Heidari N, Saydi A, Kordi N. Effects of Acute L-carnitine Supplementation with Incremental Training on Fat and Carbohydrate Metabolism in Overweight/Obese and Normal-Weight Young Men. Journal of Sport Physiology Special Groups. 2024;1(4):1-15.
12. Cha Y-S, Choi S-K, Suh H, Lee S-N, Cho D, Lim K. Effects of carnitine coingested caffeine on carnitine metabolism and endurance capacity in athletes. Journal of nutritional science and vitaminology. 2001;47(6):378-84. [PMID: 11922111] [DOI]

13. Ascensão A, Ferreira R, Magalhães J. Exercise-induced cardioprotection—biochemical, morphological and functional evidence in whole tissue and isolated mitochondria. International journal of cardiology. 2007;117(1):16-30. [PMID: 16860886] [DOI]
14. Chaabène H, Mkaouer B, Franchini E, Souissi N, Selmi MA, Nagra Y, et al. Physiological responses and performance analysis difference between official and simulated karate combat conditions. Asian journal of sports medicine. 2013;5(1):21. [PMID: 24868428] [PMCID: PMC4009084] [DOI]
15. Clarkson PM, Sayers SP. Gender differences in exercise-induced muscle damage. Gender differences in metabolism: CRC Press; 2020. p. 283-99
16. Wall BT, Stephens FB, Constantin-Teodosiu D, Marimuthu K, Macdonald IA, Greenhaff PL. Chronic oral ingestion of L-carnitine and carbohydrate increases muscle carnitine content and alters muscle fuel metabolism during exercise in humans. The Journal of physiology. 2011;589(4):963-73. [PMID: 21224234] [PMCID: PMC3060373] [DOI]
17. Bassett DR, Howley ET. Limiting factors for maximum oxygen uptake and determinants of endurance performance. Medicine and science in sports and exercise. 2000;32(1):70-84. [PMID: 10647532] [DOI]
18. Petersen EW, Ostrowski K, Ibfelt T, Richelle M, Offord E, Halkjær-Kristensen J, et al. Effect of vitamin supplementation on cytokine response and on muscle damage after strenuous exercise. American Journal of Physiology-Cell Physiology. 2001;280(6):C1570-C5. [PMID: 11350752] [DOI]
19. Tsakiris S, Parthimos T, Parthimos N, Tsakiris T, Schulpis KH. The beneficial effect of L-cysteine supplementation on DNA oxidation induced by forced training. Pharmacological research. 2006;53(4):386-90. [PMID: 16517176] [DOI]
20. Cunningham P, Geary M, Harper R, Pendleton A, Stover S. HIGH INTENSITY SPRINT TRAINING REDUCES LIPID PEROXIDATION IN FAST-TWITCH SKELETAL MUSCLE. Journal of Exercise Physiology Online. 2005;8(6).
21. Ghasemzadeh M, Hosseini E, Roudsari ZO, Zadkhak P. Intraplatelet reactive oxygen species (ROS) correlate with the shedding of adhesive receptors, microvesiculation and platelet adhesion to collagen during storage: does endogenous ROS generation downregulate platelet adhesive function? Thrombosis Research. 2018;163:153-61. [PMID: 29407628] [DOI]
22. Klaus V, Hartmann T, Gambini J, Graf P, Stahl W, Hartwig A, et al. 1, 4-Naphthoquinones as inducers of oxidative damage and stress signaling in HaCaT human keratinocytes. Archives of biochemistry and biophysics. 2010;496(2):93-100. [PMID: 20153715] [DOI]
23. Vavaev A, Buryachkovskaya L, Uchitel I, Tishchenko E, Maksimenko A. Effect of hydrogen peroxide and catalase derivatives on functional activity of platelets. Bulletin of experimental biology and medicine. 2012;152:307-12. [PMID: 22803073] [DOI]