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Effects of lycopene supplementation on oxidative stress markers and inflammatory cytokines in a rat model of multiple sclerosis; an experimental study

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Abstract

Introduction: Multiple sclerosis (MS) is a chronic inflammatory and neurodegenerative disorder in which oxidative stress and immune dysregulation play important roles in disease progression. Lycopene, a potent antioxidant carotenoid, may help modulate these pathways, but its therapeutic potential in MS remains insufficiently explored.
Objectives: This experimental study aimed to evaluate the effects of lycopene on oxidative stress markers and inflammatory cytokines in a rat model of MS.
Materials and Methods: This experimental study was conducted at Jahrom University of Medical Sciences from July to September 2018. In a duration of 8 weeks, 40 male Sprague-Dawley rats were allocated to a healthy control group, an MS-induced group, a lycopene-treated group (200 mg/kg), and two lycopene noisome-treated groups (100 and 200 mg/kg). Serum samples were collected after the intervention to assess total antioxidant capacity (TAOC), total oxidant capacity (TOC), malondialdehyde (MDA), interleukin-10 (IL-10), and IL-17. The outcomes include the comparison of TAOC, TOC, MDA, IL-10, and IL-17 among experimental groups.
Results: The results demonstrated that compared with the healthy rats, the inducted-MS rats indicated a significant deterioration in oxidative stress and inflammatory profiles, with a reduction in TAOC and IL-10 and an increase in TOC, MDA, and IL-17. Lycopene treatment ameliorated these abnormalities across all intervention groups, as evidenced by increased TAOC and IL-10 levels alongside reductions in TOC, MDA, and IL-17 relative to the MS group. Among the treated groups, the lycopene + noisome at 200 mg/kg produced the most pronounced improvement.
Conclusion: Lycopene supplementation effectively mitigates oxidative stress and inflammation in MS, with high-dose, lycopene-loaded noisome providing a superior therapeutic strategy. These findings underscore the potential of nano-formulated delivery to enhance the efficacy of lycopene-based interventions.

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Introduction

Multiple sclerosis (MS) is a chronic autoimmune disease of the central nervous system characterized by inflammation, demyelination, and progressive neurodegeneration, leading to motor, sensory, and cognitive disability over time (1,2). Recent epidemiological data from the Atlas of MS indicate that approximately 2.8 million people worldwide are living with MS, with a global prevalence of about 35.9 per 100,000 and a higher burden in women (3). Beyond immune-mediated demyelination, accumulating evidence implicates oxidative stress and redox imbalance as major contributors to axonal injury and disease progression in both relapsing–remitting and progressive forms of MS (4). Experimental autoimmune encephalomyelitis (EAE) remains the most widely used animal model to explore MS pathomechanisms and to test antioxidant and immunomodulatory strategies (5,6).

Clinical and experimental studies consistently show increased oxidative damage and altered antioxidant defenses in MS and EAE, including elevated lipid peroxidation products such as malondialdehyde (MDA) and disturbed activities of superoxide dismutase (SOD), catalase, and glutathione systems (4,7). In EAE, enhanced MDA levels, decreased sulfhydryl groups, and glutathione depletion correlate with neurological deficits and tissue damage, supporting a causal link between oxidative stress and demyelination (6). Pro-inflammatory cytokines, particularly interleukin-17 (IL-17), tumor necrosis factor-α (TNF-α), and interleukin-6, are upregulated in MS lesions and EAE, whereas the anti-inflammatory cytokine interleukin-10 (IL-10) is often reduced, shifting the immune milieu toward a pathogenic Th1/Th17 response

Key point

The findings indicated that lycopene supplementation effectively mitigates oxidative stress and inflammation in a rat model of multiple sclerosis (MS), with lycopene-loaded noisome supplementation at the highest dosage providing a superior therapeutic strategy for restoring antioxidant status and curbing inflammatory pathways in experimental MS, highlighting the potential for nano-formulated delivery to enhance the efficacy of lycopene-based interventions.

(4,8). Therefore, interventions capable of restoring redox homeostasis while modulating cytokine networks may offer neuroprotective benefits in MS.

Lycopene, a non-provitamin A carotenoid abundant in tomatoes and other red fruits, is one of the most potent dietary antioxidants and exhibits broad anti-inflammatory activity in vitro and in vivo (9,10). Mechanistic and experimental studies show that lycopene scavenges reactive oxygen species, enhances endogenous antioxidant enzymes, and attenuates lipid peroxidation in diverse tissues (9-11). In hypercholesterolemia and other rodent models, lycopene supplementation downregulates NF-κB (nuclear factor-κB) signalling and reduces levels of pro-inflammatory cytokines such as TNF-α and IL-6, while restoring glutathione, SOD, catalase, and related antioxidant defenses (11). These properties position lycopene as a promising candidate for targeting the intertwined pathways of oxidative stress and inflammation that underlie neuroinflammatory diseases.

Natural products with antioxidant properties have shown efficacy in ameliorating clinical scores, reducing oxidative damage, and limiting demyelination in MS animal models, including EAE, and are increasingly explored as adjunctive strategies (12). A recent study reported that a Mediterranean diet supplemented with lycopene delayed EAE onset, lowered clinical scores, reduced IL-17 production, and improved spinal cord myelination in mice, suggesting a disease-modifying potential for lycopene in central nervous system autoimmunity (5). However, data directly addressing how lycopene affects specific oxidative stress markers and cytokine profiles, such as IL-10 and IL-17, in MS-like conditions remain limited.

Objectives

The present experimental study, entitled the effects of lycopene supplementation on oxidative stress markers and inflammatory cytokines in a rat model of MS, was therefore designed to evaluate whether lycopene supplementation can modulate oxidative stress indices and key inflammatory cytokines in an EAE-like rat model, thereby providing mechanistic insight into its potential neuroprotective role.

Materials Methods

Study design and samples

This experimental animal study was conducted at Jahrom University of Medical Sciences from July to September 2018. Forty Sprague-Dawley male rats with an average weight of 200 to 220 g were randomly assigned to five groups of 8 animals each; a healthy control group, an MS-induced group, a lycopene-treated group receiving 200 mg/kg, and two lycopene noisome-treated groups receiving 100 mg/kg and 200 mg/kg body weight.

Animal preparation

Forty male Sprague-Dawley rats were used in the study and were acclimatized in the animal facility of Jahrom University of Medical Sciences for one week before the experiment. The animals were maintained under a 12-hour light/12-hour dark cycle at a temperature of 23±2 °C and a relative humidity of 50–55%.

Preparation of lycopene

Five kilograms of ripe tomatoes were stored at 4°C and used within 48 hours for lycopene isolation. After blanching by immersing the tomatoes in boiling water for 1–2 minutes, followed by rapid cooling, the tomato skins were manually removed. The peels were dried at room temperature (24–25 °C) for 2–3 hours and then packed in sealed polyethylene zip-lock bags and stored at 4°C. The dried peels were mixed with a small volume of solvent system (n-hexane:acetone:ethanol, 2:1:1, v/v) in the presence of CO2, and then processed for 4 hours under an inert CO2 atmosphere in the dark using an ultrasonic crusher and magnetic shaker. The resulting extract was collected in a colored glass container and stored under CO2 at 4 °C. Finally, the extract was dried in the dark at 60 °C and 50 rpm using a rotary flash evaporator. The yield of purified lycopene extracted from tomato peel was 571 micrograms per milliliter of the solvent system (13,14).

Lycopene niosomes were prepared according to the method described by Sharma et al. In this procedure, the purified extract was dispersed in a small volume of solvent system (n-hexane:acetone:ethanol, 2:1:1, v/v). Approximately 22 mL of Span 60 and cholesterol at a 1:1 molar ratio was added to 10 mL of the solvent as the organic phase containing the wall material. This phase was adsorbed onto an inert support mixed at constant temperature at 60 °C, as previously described by Sharma et al. The aqueous phase (10 mL mannitol solution, pH 7.4) was then slowly added to the lycopene-loaded support and stirred at 60°C, followed by sonication using a probe sonicator for 60 seconds per cycle in three cycles to prevent overheating. The suspension was then allowed to stand for 2–3 hours to stabilize the niosomes. The lycopene-loaded noisome suspension was filtered through Whatman No. 1 filter paper to remove inert materials, and the filtrate was then passed through a Sephadex G-50 column to separate unentrapped lycopene. Finally, the niosomal vesicles were collected and lyophilized, and the freeze-dried formulation was stored in a sealed plastic container under compressed air (15).

MS induction method

To induce MS, mice were fed a diet containing cuprizone, which causes oligodendrocyte damage and subsequent demyelination. The disease course was monitored daily using a clinical scoring system, and animals with a score of 1 were considered to have developed MS (16).

Experimental groups

The control group was maintained under standard conditions without any treatment, while the experimental groups were subjected to MS induction and then received either olive oil as the lycopene solvent by gavage, lycopene at 200 mg/kg body weight, lycopene noisome at 100 mg/kg body weight by gavage, or lycopene noisome at 200 mg/kg body weight by gavage for eight consecutive weeks after induction.

Blood sample collection

One day after the final lycopene injection on day 57, motor performance was assessed, and all mice were anesthetized with diethyl ether before blood collection using a 5 mL syringe. The blood samples were centrifuged at 3000 rpm for 15 minutes, and the separated serum was stored at -20 °C for subsequent measurement of total antioxidant capacity (TAOC), total oxidant capacity (TOC), MDA, IL-10, and IL-17 using rat-specific enzyme-linked immunosorbent assay (ELISA) kits.

Measurement outcomes

The primary outcomes include comparison of TAOC, TOC, MDA, IL-10, and IL-17 between the healthy control group, the MS-induced group, the lycopene-treated group receiving 200 mg/kg, and two lycopene noisome-treated groups receiving 100 mg/kg and 200 mg/kg body weight.

Statistical analysis

Data were analyzed using IBM SPSS version 27 (IBM Corp., USA) and GraphPad Prism 8 (California, USA). Group comparisons were carried out by one-way analysis of variance (ANOVA), followed by Duncan's post hoc test for pairwise comparisons. Statistical significance was set at $P<0.05$.

Results

In this experimental study, 40 male rats (8 per group), each weighing 220 ± 10 g, were included in the analysis. The comparative analysis indicated that the distribution of oxidative stress markers and inflammatory cytokines across experimental groups differed significantly. Compared with the control group, the MS group showed a marked deterioration in oxidative stress and inflammatory profiles, with reduced TAOC and IL-10 and increased TOC, MDA, and IL-17, while all between-group differences were statistically significant. Treatment with lycopene improved these abnormalities in all intervention groups, with TAOC rising and TOC, MDA, and IL-17 declining relative to MS, alongside an increase in IL-10; overall, the MS + lycopene noisome (LN) 200 mg/kg group demonstrated the most favorable pattern, showing the strongest restoration of antioxidant status and the greatest reduction in oxidative and inflammatory markers (Table 1).

The comparison of TAOC between groups indicated that the TAOC was reduced in the MS rats compared with the healthy control group, while all treatment groups showed significant improvement, with the highest recovery observed in the higher-dose LN group. In contrast, TOC increased in the MS rats versus the healthy control group and then declined after treatment, with the greatest reduction again seen in the higher-dose LN group (Figure 1).

Following the induction of MS in rats, the IL-10 was

Table 1. Comparison of oxidative stress markers and inflammatory cytokines across experimental groups

Group		Mean	*P* value*
Oxidative stress markers	TAOC (U/mL)		
	Control	9.8	
	MS	5.7	
	MS + L 200 mg/kg	8.7	<0.001
	MS + LN 100 mg/k	8.2	
	MS + LN 200 mg/k	9.3	
	TOC (U/mL)		
	Control	12.8	
	MS	36.5	
	MS + L 200 mg/kg	15.9	<0.001
	MS + LN 100 mg/k	15.1	
	MS + LN 200 mg/k	13.4	
Inflammatory cytokines	MDA (nmol/mL)		
	Control	1.5	
	MS	7.3	
	MS + L 200 mg/kg	2.6	<0.001
	MS + LN 100 mg/k	2.7	
	MS + LN 200 mg/k	2.1	
	IL-10 (pg/mL)		
	Control	248	
	MS	106	
	MS + L 200 mg/kg	164	<0.001
	MS + LN 100 mg/k	171	
	MS + LN 200 mg/k	197	
	IL-17 (pg/mL)		
	Control	234	
	MS	417	
	MS + L 200 mg/kg	327	<0.001
	MS + LN 100 mg/k	312	
	MS + LN 200 mg/k	276	

MDA: Malondialdehyde, TOC: Total oxidant capacity, TAOC: Total antioxidant capacity, MS: Multiple Sclerosis, L: Lycopene, N: Noisome , IL: Interleukin, SD: Standard deviation. *ANOVA.

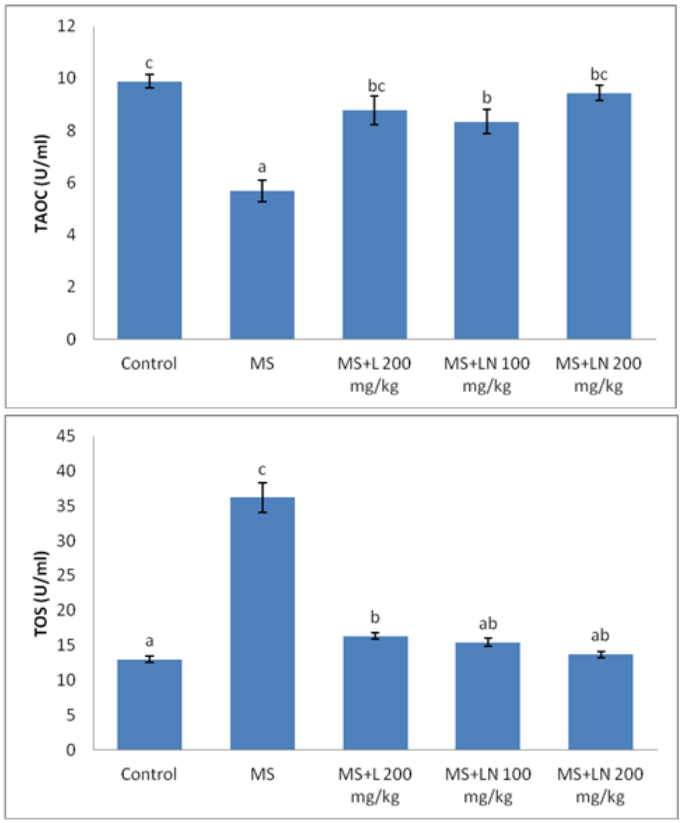

Figure 1. Comparison of oxidative stress markers among treatment groups. TOC: Total oxidant capacity, TAOC: Total antioxidant capacity, MS: Multiple Sclerosis, L: Lycopene, N: Noisome. The means within each column that share at least one common letter do not differ significantly according to Duncan's test, whereas comparisons with no letters in common are statistically significant.

decreased compared to the healthy control group, whereas treatment with lycopene or lycopene-loaded noisome improved TAOC, with the higher-dose noisome group showing the greatest restoration toward control levels. In contrast, both MDA and IL-17 were elevated in the MS group compared with the control and were reduced by both treatments, with the strongest reduction observed in the higher-dose noisome group (Figure 2).

Discussion

Our experimental MS model confirmed that disease induction was associated with a marked shift toward oxidative and inflammatory imbalance, and that lycopene, particularly when delivered as a high-dose noisome formulation, partially normalized this profile by increasing TAOC and IL-10 and decreasing TOC, MDA, and IL-17 compared with untreated MS rats. These data support a protective role for lycopene against MS-related oxidative stress and inflammation in vivo. Our findings are consistent with previous experimental studies showing that lycopene can reduce oxidative injury and inflammatory responses. In an EAE mouse model, a Mediterranean diet supplemented with lycopene delayed disease onset, reduced clinical scores, lowered T-cell counts, decreased IL-17A production, and improved spinal cord myelination, providing MS-model–specific support for the IL-17-modulating and neuroprotective potential of lycopene (5). In an experimental rat model, lycopene attenuated liver injury, suggesting a partial hepatoprotective effect against endotoxemia-induced damage. The observed benefits were associated with reduced oxidative stress, dampened nuclear factor kappa B–mediated inflammatory signaling, and inhibition of apoptosis, indicating that lycopene may exert its protective effects through multiple interconnected pathways (17). Fioretto et al reported that lycopene supplementation reduced inflammatory responses and histopathological lung injury, improved oxygenation, and decreased DNA damage (18). In an experimental rat study by Erten et al, lycopene was found to alleviate propionic acid–induced autism-like symptoms by suppressing inflammation and oxidative stress (19). Gao et al reported that lycopene may exert renoprotective effects in calcium oxalate nephrolithiasis by reducing oxidative stress, apoptosis, pyroptosis, fibrosis, and inflammatory injury (20). In a human study by Petyaev et al involving patients with coronary vascular disease, lycopene supplementation improved cardiovascular outcomes by lowering inflammatory and oxidative stress markers (21). In a study by Zhao et al, the results found that lycopene exerts strong protective effects against chronic prostatitis/chronic pelvic pain syndrome by reducing inflammation and oxidative

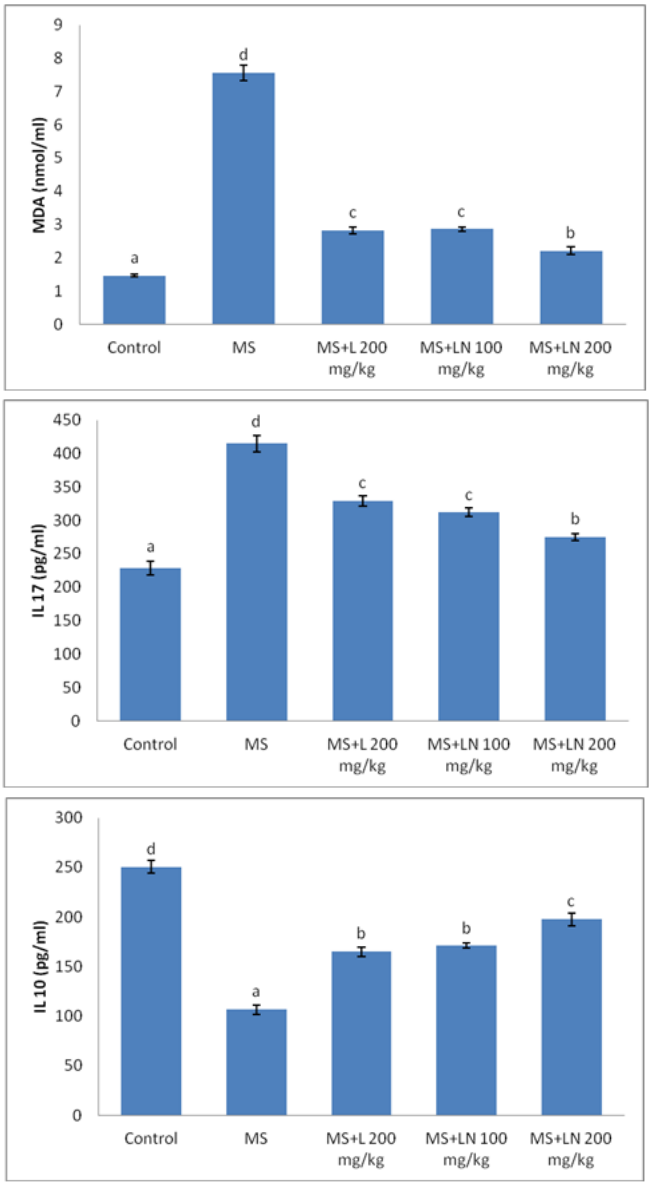

Figure 2. Comparison of inflammatory cytokines among treatment groups. MDA: Malondialdehyde, MS: Multiple Sclerosis, L: Lycopene, N: Noisome, IL: Interleukin. The means within each column that share at least one common letter do not differ significantly according to Duncan's test, whereas comparisons with no letters in common are statistically significant.

stress (22). Furthermore, Kirişçi et al confirmed the protective effects of lycopene against ischemia/reperfusion injury in a rat hind limb muscle model by improving inflammatory and oxidative stress markers (23).

Although our study did not directly evaluate intracellular signaling, the observed reduction in oxidative and inflammatory markers is compatible with mechanisms proposed in previous lycopene research. Experimental work has shown that lycopene can activate Nrf2-dependent antioxidant responses and inhibit NF-κB signaling, thereby decreasing oxidative damage and the production of pro-inflammatory cytokines in neuronal and systemic tissues (22). Other studies suggest that lycopene modulates pathways such as TLR4/NF-κB, PPAR-γ, PGC-1α, and PON-1, contributing to improved cellular redox homeostasis and dampened inflammatory signaling in heart, kidney, liver, and brain (9,11,17,21).

Overall, lycopene supplementation in a rat model of MS attenuated systemic oxidative stress and partially corrected an adverse cytokine profile characterized by reduced IL-10 and elevated IL-17, with the most robust effects observed for high-dose noisome-encapsulated lycopene. These results are in line with a growing body of experimental and translational evidence indicating that lycopene exerts antioxidant and anti-inflammatory effects in the nervous system and in systemic inflammatory states, including models that more directly mimic MS pathogenesis. While our data support lycopene, particularly in nano-formulated form, as a promising adjunctive candidate for modulating oxidative and immune pathways relevant to MS, more comprehensive mechanistic, histopathological, and behavioral studies, followed by carefully designed

clinical investigations, are needed before any therapeutic recommendations can be made.

Conclusion

In conclusion, lycopene supplementation effectively mitigates oxidative stress and inflammation in a rat model of MS, with lycopene-loaded noisome at the highest dosage demonstrating superior therapeutic efficacy in restoring biochemical profiles toward normal levels. These findings also suggest that nano-formulated lycopene may offer enhanced protective effects against disease-associated metabolic imbalances compared with conventional lycopene treatment.

Limitations of the study

Several limitations should be considered when interpreting these findings. First, the study was performed in a rat model, so the results may not fully translate to human MS. Second, the sample size was relatively small, and the intervention period was limited, which may restrict the generalizability of the outcomes. Third, the analysis focused mainly on serum oxidative and inflammatory biomarkers, without detailed histopathological or mechanistic assessment of demyelination and remyelination. Finally, because only two doses of lycopene noisome and one dose of conventional lycopene were tested, the optimal therapeutic dose and long-term safety remain uncertain.

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Authors' contribution

Conceptualization: Shekoofeh Atashpour.
Data curation: Mohamadnavid Vanda.
Formal analysis: Shekoofeh Atashpour.
Investigation: Shekoofeh Atashpour.
Methodology: Mohamadnavid Vanda.
Project management: Mohamadnavid Vanda.
Resources: All authors.
Supervision: All authors.
Validation: Shekoofeh Atashpour.
Writing–original draft: All authors.
Writing–review and editing: All authors.

Ethical issues

This study was derived from the thesis of Mohamadnavid Vanda, a general medicine student (Thesis No. 13971101020), and was conducted at the animal house facility of Jahrom University of Medical Sciences, Jahrom, Iran. The study received ethical approval under code IR.JUMS.REC.1397.004 (available at: https://ethics.research.ac.ir/form/8df5gig5wh1lx4.pdf). The research protocol adhered to guidelines for animal studies. The experimental procedures were also conducted under the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986). We also followed the guidelines related to animal experiments, approved by the United States National Institutes of Health (NIH, 1978). Besides, the authors have ultimately observed ethical issues (including plagiarism, data fabrication, and double publication).

Conflicts of interest

The authors declare no conflict of interest.

Data availability statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of generative artificial intelligence (AI) and AI-assisted technologies in the writing process

While preparing this work, the authors utilized AI (Perplexity and Grammarly) to refine grammar points and language style. Subsequently, they thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

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References

1. Arachchige A, El Choueiri J, Pellicanò F, Laurelli F, Alves GAM, Stomeo N. A review of multiple sclerosis: From pathophysiology to latest therapeutic advances. AIMS Neurosci. 2025;12:514–38. doi: 10.3934/Neuroscience.2025026.
2. Nissen JC, Tsirka SE. Preclinical model of multiple sclerosis: Methods in autoimmune demyelination. Methods Cell Biol. 2022;168:67–86. doi: 10.1016/bs.mcb.2021.12.009.
3. Walton C, King R, Rechtman L, Kaye W, Leray E, Marrie RA, et al. Rising prevalence of multiple sclerosis worldwide: Insights from the Atlas of MS, third edition. Mult Scler. 2020;26:1816–21. doi: 10.1177/1352458520970841.
4. Pegoretti V, Swanson KA, Bethea JR, Probert L, Eisel ULM, Fischer R. Inflammation and Oxidative Stress in Multiple Sclerosis: Consequences for Therapy Development. Oxid Med Cell Longev. 2020;2020:7191080. doi: 10.1155/2020/7191080.
5. Atuk Kahraman T, Yılmaz M, Aslan K, Canatan H, Kara A, Nalbantoglu OU, et al. Lycopene Supplemented Mediterranean Diet Ameliorates Experimental Autoimmune Encephalomyelitis (EAE) in Mice and Changes Intestinal Microbiome. J Neuroimmune Pharmacol. 2025;20:50. doi: 10.1007/s11481-025-10212-7.
6. Dąbrowska-Bouta B, Strużyńska L, Sidoryk-Węgrzynowicz M, Sulkowski G. Memantine Modulates Oxidative Stress in the Rat Brain following Experimental Autoimmune Encephalomyelitis. Int J Mol Sci. 2021;22:11330. doi: 10.3390/ijms222111330.
7. Dimitrijević M, Kotur-Stevuljević J, Stojić-Vukanić Z, Vujnović I, Pilipović I, Nacka-Aleksić M, et al. Sex Difference in Oxidative Stress Parameters in Spinal Cord of Rats with Experimental Autoimmune Encephalomyelitis: Relation to Neurological Deficit. Neurochem Res. 2017;42:481–92. doi: 10.1007/s11064-016-2094-7.
8. Ugbaja RN, James AS, Ugwor EI, Akamo AJ, Thomas FC, Kosoko AM. Lycopene suppresses palmitic acid-induced brain oxidative stress, hyperactivity of some neuro-signalling enzymes, and inflammation in female Wistar rat. Sci Rep. 2021;11:15038. doi: 10.1038/s41598-021-94518-5.
9. Shafe MO, Gumede NM, Nyakudya TT, Chivandi E. Lycopene: A Potent Antioxidant with Multiple Health Benefits. J Nutr Metab. 2024;2024:6252426. doi: 10.1155/2024/6252426.
10. Karaköy Z, Cadirci E, Dincer B. A New Target in Inflammatory Diseases: Lycopene. Eurasian J Med. 2022;54:23–8. doi: 10.5152/eurasianjmed.2022.22303.
11. Albrahim T. Lycopene modulates oxidative stress and inflammation in hypercholesterolemic rats. Pharmaceuticals

(Basel). 2022;15:1420. doi: 10.3390/ph15111420.
12. Zha Z, Liu S, Liu Y, Li C, Wang L. Potential Utility of Natural Products against Oxidative Stress in Animal Models of Multiple Sclerosis. Antioxidants (Basel). 2022;11. doi: 10.3390/antiox11081495.
13. Lavecchia R, Zuorro A. Enhancement of lycopene extraction from tomato peels by enzymatic treatment. Chem Engineering Transactions. 2008;14:301–8.
14. Kushwaha A, Murthy RN, Murthy SN, Elkeeb R, Hui X, Maibach HI. Emerging therapies for the treatment of ungual onychomycosis. Drug Dev Ind Pharm. 2015;41:1575–81. doi: 10.3109/03639045.2015.1033426.
15. Sharma P, Saxena P, Jaswanth A, Chalamaiah M, Tekade K, Balasubramaniam A. Novel encapsulation of lycopene in niosomes and assessment of its anticancer activity. J Bioequiv Availab. 2016;8:224–32. doi: 10.4172/jbb.1000300.
16. Wang XD. Lycopene metabolism and its biological significance. Am J Clin Nutr. 2012;96:1214s–22s. doi: 10.3945/ajcn.111.032359.
17. Tešić Rajković S, Rančić A, Stojanović M, Živadinović J, Ramić I, Nestorović M, et al. Lycopene Mitigates Rat Liver Damage Induced by Lipopolysaccharide via Mechanisms Involving Oxidative Stress, Inflammation, and Apoptosis. Curr Issues Mol Biol. 2025;47. doi: 10.3390/cimb47110914.
18. Fioretto JR, Klefens SO, Carpi MF, Moraes MA, Bonatto RC, Ferreira ALA, et al. Lycopene supplementation reduces inflammatory, histopathological and DNA damage in an acute lung injury rabbit model. Crit Care Sci. 2025;37:e20250250. doi: 10.62675/2965-2774.20250250.
19. Erten F. Lycopene ameliorates propionic acid-induced autism spectrum disorders by inhibiting inflammation and oxidative stress in rats. J Food Biochem. 2021;45:e13922. doi: 10.1111/jfbc.13922.
20. Gao X, Lin B, Chen C, Fang Z, Yang J, Wu S, et al. Lycopene from tomatoes and tomato products exerts renoprotective effects by ameliorating oxidative stress, apoptosis, pyroptosis, fibrosis, and inflammatory injury in calcium oxalate nephrolithiasis: the underlying mechanisms. Food Funct. 2024;15:4021–36. doi: 10.1039/d4fo00042k.
21. Petyaev IM, Dovgalevsky PY, Klochkov VA, Chalyk NE, Pristensky DV, Chernyshova MP, et al. Effect of lycopene supplementation on cardiovascular parameters and markers of inflammation and oxidation in patients with coronary vascular disease. Food Sci Nutr. 2018;6:1770–7. doi: 10.1002/fsn3.734.
22. Zhao Q, Yang F, Meng L, Chen D, Wang M, Lu X, et al. Lycopene attenuates chronic prostatitis/chronic pelvic pain syndrome by inhibiting oxidative stress and inflammation via the interaction of NF-κB, MAPKs, and Nrf2 signaling pathways in rats. Andrology. 2020;8:747–55. doi: 10.1111/andr.12747.
23. Kirişçi M, Güneri B, Seyithanoğlu M, Kazancı Ü, Doğaner A, Güneş H. The protective effects of lycopene on ischemia/reperfusion injury in rat hind limb muscle model. Ulus Travma Acil Cerrahi Derg. 2020;26:351–60. doi: 10.14744/tjtes.2020.81456.