Review Article

Dietary Bioactives as NLRP3 Inflammasome Modulators: A Novel Adjunctive Approach in Myasthenia Gravis

Pratibha Daroi*, Sudhir Maurya, Dhananjay Raidas
LSHGCT's Gahlot Institute of Pharmacy, Koparkhairne, Navi Mumbai-400 709, India.
***Corresponding author's E-mail:** pratibhadaroi@gmail.com

Received: 12-02-2026; **Revised:** 28-04-2026; **Accepted:** 05-05-2026; **Published online:** 20-05-2026.

ABSTRACT

Myasthenia Gravis (MG) is a debilitating autoimmune neuromuscular disorder characterized by impaired cholinergic transmission due to autoantibody-mediated targeting of the acetylcholine receptor at the neuromuscular junction. Although immunosuppressive therapies provide symptomatic relief, they carry risks of systemic side effects and do not fully address underlying neuroinflammation. The nucleotide-binding oligomerization domain-like receptor family pyrin domain-containing 3 (NLRP3) inflammasome is a multiprotein complex that plays a pivotal role in innate immunity by mediating caspase-1 activation and subsequent interleukin-1β (IL-1β) and IL-18 secretion. Dysregulated NLRP3 activation is strongly associated with chronic inflammatory, autoiimmune and metabolic disorders, including MG. Emerging evidence implicates the NLRP3 inflammasome—a cytosolic multiprotein complex regulating IL-1β and IL-18 maturation—in perpetuating MG pathology through innate immune activation and pyroptotic pathways. Notably, naturally derived bioactive compounds—including polyphenols, flavonoid, omega-3 fatty acids, sulforaphane, and vitamin D—have shown robust ability to inhibit NLRP3 priming and activation. Preclinical models of autoimmune disease, including experimental autoimmune MG (EAMG), have demonstrated therapeutic benefit from such interventions. This review synthesizes mechanistic insights into NLRP3's role in MG and outlines natural modulators with translational promise. Nutritional strategies targeting NLRP3 may serve as valuable adjuncts to existing MG therapies, warranting rigorous clinical investigation.

Keywords: NLRP3 inflammosomes, Myasthenia Gravis, Dietary bioactive, Neuroinflammation, immune modulation, adjunctive therapy, oxidative stress.

INTRODUCTION

Autoimmune diseases (ADs) affect approximately 8% to 10% of the global population, with prevalence rising significantly due to environmental, lifestyle, and diagnostic changes. Among numerous autoimmune disorders, Myasthenia Gravis (MG) has shown remarkable increase in prevalence with recent estimates suggesting around 150 to 300 cases per million people (0.015%–0.03% of the population)[1]. MG is a debilitating chronic autoimmune neuromuscular disorder. It is marked by impaired cholinergic transmission caused by autoantibody-mediated targeting of the acetylcholine receptor at the neuromuscular junction which breaks down communication between nerves and muscles. As a result of this, patient experiences fluctuating, fatigable muscle weakness that worsens with exertion and improves with rest[2]. The underlying defect occurs at the neuromuscular junction (NMJ), where pathogenic autoantibodies impair synaptic transmission by targeting components essential for cholinergic signaling[3]. Among these, antibodies against the acetylcholine receptor (AChR) account for approximately 80–85% of generalized MG cases, leading to receptor internalization and complement-mediated destruction of the postsynaptic membrane[4]. In addition to AChR-specific antibodies, other antigenic targets such as muscle-specific kinase (MuSK) and low-density lipoprotein receptor-related protein 4 (LRP4) have been identified, contributing to disease heterogeneity and influencing therapeutic response[5]. These pathogenic antibodies originate from a breakdown in immune tolerance, involving autoreactive T and B cells, impaired regulatory T-cell function, and germinal center formation in the thymus, a hallmark in AChR-positive MG[6]. The immunopathogenesis of MG is characterized by complement activation, disruption of clustering proteins, and impaired neuromuscular signaling, ultimately resulting in failure of neuromuscular transmission and clinically evident muscle weakness. Current therapies aim to alleviate symptoms and suppress immune activity; however, long-term use of broad immunosuppressants carries risks of infection, metabolic complications, and organ toxicity, highlighting the need for novel, targeted, and well-tolerated approaches. Standard treatments of MG rely on acetylcholinesterase inhibitors, corticosteroids, and other immunosuppressive agents. While effective for many, these interventions are marred by systemic side effects and do not target the neuroimmune mechanisms perpetuating disease[7].

Innate immune activation, and specifically the NLRP3 inflammasome, has gained recognition for its role in autoimmune and neuroinflammatory diseases[8]. Triggered by stress signals such as mitochondrial dysfunction, reactive oxygen species (ROS), or protein aggregates, the NLRP3 complex activates caspase-1, liberating IL-1β and IL-18—potent drivers of inflammation and autoimmunity[9,10]. Recent studies suggest a contributory role for NLRP3 in MG pathogenesis, including genetic associations and evidence of inflammasome activation in patient samples and experimental models[10,11].

©Copyright protected. Unauthorised republication, reproduction, distribution, dissemination and copying of this document in whole or in part is strictly prohibited.

Despite advances in synthetic NLRP3 inhibitors, concerns regarding long-term safety, high cost, and potential immunosuppression limit their widespread use[12]. In contrast, natural bioactive compounds derived from dietary sources offer a promising, safer, and cost-effective alternative for chronic disease management. Nutritional modulators—such as polyphenols, flavonoids, omega-3 fatty acids, and terpenoids—not only exert anti-inflammatory effects but also provide ancillary health benefits, aligning with preventive and integrative medicine approaches[13].

This review aims to bridge mechanistic insights with translational opportunity by evaluating natural dietary compounds known to inhibit NLRP3 activation—such as polyphenols, omega-3 fatty acids, sulforaphane, and vitamin D—highlighting their pharmacology, safety, and relevance to MG. It also covers preclinical and clinical evidence supporting their efficacy. Furthermore, we highlight existing challenges and future perspectives for translating these findings into clinical practice.

MECHANISTIC BASIS: NLRP RECEPTORS IN MG PATHOGENESIS

The NLRP family, particularly the NLRP3 inflammasome, plays a pivotal role in innate immune activation and is emerging as a central mediator in autoimmune neuromuscular disorders, including Myasthenia Gravis (MG). MG is primarily driven by autoantibodies against acetylcholine receptors (AChRs) or muscle-specific kinase (MuSK), leading to impaired neuromuscular transmission. However, adaptive immunity is not the sole contributor; innate immune components such as inflammasomes create a permissive inflammatory milieu that sustains autoreactive T and B cell responses.

1. NLRP3 inflammasome assembly and activation

The NLRP3 inflammasome is a cytosolic multiprotein complex composed of NLRP3, ASC (apoptosis-associated speck-like protein), and caspase-1. Its activation is triggered by various danger-associated molecular patterns (DAMPs) and pathogen-associated molecular patterns (PAMPs)[14,15]. In MG, oxidative stress, mitochondrial dysfunction, and circulating immune complexes act as stimuli, initiating a two-signal activation mechanism:

- Signal 1 (Priming): Toll-like receptor (TLR)-mediated NF-κB activation upregulates NLRP3 and pro-IL-1β gene transcription.
- Signal 2 (Activation): Cellular stressors, such as ATP release and ROS accumulation, induce NLRP3 oligomerization and inflammasome assembly.

2. Downstream cytokine cascade

Activated NLRP3 inflammasomes cleave pro-caspase-1 into active caspase-1, facilitating the maturation of IL-1β and IL-18. These cytokines amplify Th1 and Th17 cell responses, perpetuating autoantibody generation and complement-mediated damage at the neuromuscular junction. Studies have demonstrated elevated IL-1β and IL-18 levels in MG patients, correlating with disease severity, indicating a pathogenic contribution of inflammasome activation.

3. Crosstalk with autophagy and cell death pathways

Autophagy, a cellular housekeeping mechanism, exerts a regulatory effect on inflammasome activity. Defective autophagy observed in MG enhances ROS accumulation, promoting NLRP3 activation. Pyroptosis, an inflammatory form of cell death driven by caspase-1-mediated gasdermin-D cleavage, may further exacerbate neuromuscular junction inflammation and antigen release.

4. Interaction with Adaptive Immunity

NLRP3 activation creates a pro-inflammatory microenvironment that favors antigen-presenting cell (APC) activation and epitope spreading, ultimately supporting autoreactive B cell maturation. This mechanistic interplay suggests that controlling NLRP3 activity could dampen both innate and adaptive immune drivers in MG.

These discrete activation steps offer multiple intervention points for natural compounds: attenuation of NF-κB signaling, antioxidant mitigation of mitochondrial dysfunction, blockade of NEK7-mediated assembly, or inhibition of caspase-1 and GSDMD activity (Figure 1).

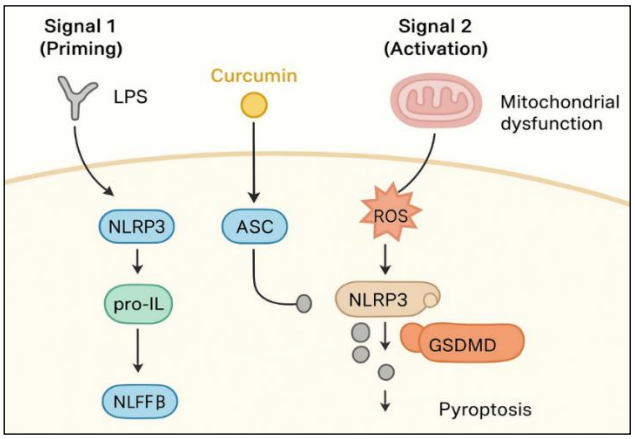

Figure 1: Schematic of NLRP3 inflammasome activation pathway, indicating signal 1 (priming), signal 2 (activation) and pyroptosis

The sum of genetic, molecular, and preclinical data positions the NLRP3 inflammasome as a plausible contributor to MG pathogenesis, especially where inflammation, oxidative stress, and autoimmune amplification converge. This mechanistic basis establishes a justified rationale for targeting NLRP3 via safe, naturally derived agents.

NUTRITIONAL MODULATORS OF NLRP3 WITH TRANSLATIONAL POTENTIAL IN MG

Dietary bioactive compounds with anti-inflammatory and antioxidant properties are gaining recognition as potential adjuncts in autoimmune disease management. In MG, where NLRP3-mediated inflammation contributes to disease progression, nutritional interventions may

©Copyright protected. Unauthorised republication, reproduction, distribution, dissemination and copying of this document in whole or in part is strictly prohibited.

attenuate inflammasome activation and cytokine-driven tissue injury.

I. Polyphenols: Curcumin, Resveratrol, EGCG

i) Curcumin (Curcuma longa)

Curcumin is a polyphenolic compound with established NLRP3 inhibitory activity. It suppresses NF-κB activation (priming step) and reduces ROS-mediated NLRP3 oligomerization (activation step)[16]. In preclinical models of autoimmune disorders, curcumin downregulated IL-1β secretion and improved muscle strength, suggesting its utility in MG[17]. However, However, its clinical translation is severely hindered by poor aqueous solubility, rapid metabolism, and negligible oral bioavailability, warranting nanoformulations or adjuvant delivery strategies[18].

ii) Resveratrol (Grapes, Berries)

Resveratrol activates Sirtuin1 (SIRT1) and promotes autophagy, indirectly suppressing inflammasome assembly. Its ability to restore mitochondrial homeostasis reduces oxidative stress, mitigating NLRP3 activation[19]. In autoimmune contexts, resveratrol reduces oxidative stress and immune activation, by shifting Th17/Treg balance, which is relevant for MG pathogenesis[20,21].

iii) Epigallocatechin Gallate (EGCG, Green Tea)

EGCG exerts dual anti-oxidant and anti-inflammatory actions. It inhibits ROS accumulation, downregulates NLRP3 and caspase-1 expression, and decreases IL-1β secretion[22]. Preclinical studies in other autoimmune diseases (e.g., rheumatoid arthritis) have shown improved inflammatory profiles, making EGCG a promising candidate for MG-focused trials[23].

iv) Quercetin

Quercetin, a flavonol abundant in fruits and vegetables, downregulates NLRP3 expression and caspase-1 activation by scavenging free radicals and enhancing antioxidant defense pathways such as Nrf2 signaling, which collectively mitigate oxidative damage [24]. Quercetin inhibits the NLRP3 inflammasome at multiple points: it interferes with Apoptosis-associated Speck-like protein Containing a CARD (Caspase Recruitment Domain) ASC oligomerization/ “speck” formation, blunting caspase-1 activation and IL-1β/IL-18 release; it also dampens priming signals through NF-κB/MAPK pathways and lowers NLRP3 expression in infected/activated cells[25]. Across diverse in-vitro and in-vivo inflammation models, quercetin reduces NLRP3-dependent cytokines and pyroptosis, consistent with a class effect on inflammasome signaling[26]. Direct quercetin intervention trials in MG have not been reported. However, network-pharmacology/TCM studies of MG formulas repeatedly identify quercetin as a high-probability active constituent acting on immune/inflammatory nodes (e.g., PI3K/Akt), and one EAMG study tied a quercetin-containing formula to Akt pathway suppression—indirect, hypothesis-generating signals rather than efficacy proof[27].

II. Omega-3 Fatty Acids: DHA, EPA

Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) modulate inflammasome signaling by incorporating into cell membranes and altering lipid raft composition[28]. They also activate peroxisome proliferator-activated receptors (PPARs), reducing NF-κB activity and thereby limiting NLRP3 priming[29].

III. Vitamins and minerals: vitamin D, zinc

Vitamin D suppresses pro-inflammatory cytokines and enhances autophagy, indirectly regulating NLRP3 activation[30,31]. Observational studies indicate vitamin D deficiency in MG patients correlates with worse outcomes, suggesting supplementation could have a dual benefit—immune modulation and bone health preservation under corticosteroid therapy[31–33].

IV. Isothiocyanates: Sulforaphane

Sulforaphane, an Nrf2 activator, enhances antioxidant defenses and suppresses NLRP3 via Nrf2-driven expression of thioredoxin and heme oxygenase-1[34]. Rodent inflammatory models demonstrate robust suppression of IL-1β release and protection against pyroptosis[35].

V. Emerging Botanicals

Phytochemicals such as berberine and luteolin exert significant inhibitory effects on the NLRP3 inflammasome through multiple converging mechanisms, primarily targeting oxidative stress and preserving mitochondrial homeostasis. Berberine has been shown to attenuate NLRP3 activation by reducing reactive oxygen species (ROS) generation and stabilizing mitochondrial membrane potential, thereby preventing the release of mitochondrial danger signals that act as triggers for inflammasome assembly [36–38]. Similarly, Luteolin, another potent flavonoid, exhibits dual regulatory action by suppressing mitochondrial dysfunction and inhibiting upstream NF-κB signaling, a critical priming step for NLRP3 activation, thus reducing the release of pro-inflammatory cytokines like IL-1β and IL-18[39]. Through these integrated antioxidant and mitochondrial protective effects, these phytochemicals emerge as promising natural modulators for controlling NLRP3-driven inflammatory responses. Their potential in MG requires systematic preclinical validation and clinical investigation.

PHARMACOLOGY, DELIVERY CHALLENGES, AND FORMULATION STRATEGIES

Substantial amount of scientific research exploring natural NLRP3 inhibitors indicate their potent in vitro activity against critical modulators involved in neuroinflammation which are crucial for MG. however, clinical translation of these natural bioactive compounds faces pharmacokinetic hurdles:

- Low bioavailability: Curcumin, quercetin and resveratrol show poor absorption and rapid metabolism. EGCG undergoes extensive first-pass clearance[29,46].

©Copyright protected. Unauthorised republication, reproduction, distribution, dissemination and copying of this document in whole or in part is strictly prohibited.

To overcome these, innovative formulation strategies such as Nanoencapsulation, phytosomes, liposomal and cyclodextrin technologies can be employed which will significantly enhance plasma and tissue levels of these molecules.

- Off-target effects and safety: High-dose curcumin may affect CYP enzymes; similarly, resveratrol can interfere with anticoagulants; omega-3s have antiplatelet effects; vitamin D supplementation must avoid hypercalcemia.
- Drug–nutrient interaction: Synergy or antagonism with AChE inhibitors and immunosuppressants must be evaluated carefully, especially regarding CYP-mediated metabolism or immune modulation.

Thus, for successfully employing the bioactive molecules discussed in this review, syatematic optimization of form, dose, and co-administration timing is critical.

Table 1: Summary of dietary components and their potential role in Myasthenia Gravis

Bioactive	Plant source	Active component	NLRP3-related action	Relevance to MG (effects / evidence level)
Curcumin	Turmeric (*Curcuma longa*)	Curcumin (diferuloylmethane)	Suppresses NF-κB priming, reduces ROS and P2X7R/TLR4 signaling that drive NLRP3 assembly; decreases caspase-1 activation and IL-1β release[16].	Attenuation of IL-1β and oxidative priming suggests potential to blunt inflammasome-driven muscle/nerve injury in MG. Evidence: strong preclinical inflammasome data; no definitive MG clinical trials[16].
Resveratrol	Grapes, berries, peanuts	Resveratrol	Activates SIRT1 and autophagy, restores mitochondrial homeostasis and limits mtROS—indirectly suppresses NLRP3 assembly and caspase-1 activation[40].	By reducing oxidative priming and modulating Th17/Treg balance, resveratrol may impact adaptive autoimmune pathways relevant to MG. Evidence: preclinical immune-modulation and inflammasome suppression[40].
EGCG (Epigallocatechin-3-gallate)	Green tea (*Camellia sinensis*)	EGCG	Decreases intracellular ROS, downregulates NLRP3 and caspase-1 expression, and lowers IL-1β release in macrophage models.	Anti-inflammatory signals (less IL-1β) and systemic antioxidant effects may reduce inflammasome-driven exacerbation risk; MG-specific data absent (Preclinical)[41].
Quercetin	Onions, apples, many fruits /vegetables	Quercetin (flavonol) & glycosides	Scavenges ROS, activates Nrf2/mitophagy pathways, blocks ASC oligomerization and caspase-1 activation —reduces IL-1β/IL-18 release[25].	Mechanistic fit for MG (reducing IL-1 family cytokines and modulating T-cell milieu)[25]. Direct MG trials are lacking.
omega-3 PUFA)	Marine oils, algal oils	DHA, EPA	Incorporate into membranes, alter lipid raft composition and TLR signaling, activate PPARs → reduce NF-κB priming and decrease NLRP3 activation; pro-resolution mediators also limit inflammasome triggers[42].	May dampen systemic innate immune priming and shift adaptive immunity (Th17/Treg), theoretically lowering MG inflammatory burden[42]. Clinical MG evidence limited; plausible adjunct
Vitamin D	Dietary + cutaneous synthesis (cholecalciferol)	25(OH)D → $1,25(OH)_2D$	Vitamin D receptor (VDR) signaling inhibits NLRP3 (reduces caspase-1/IL-1β), promotes autophagy and restrains NF-κB priming[30,31].	Observational MG data link deficiency to worse outcomes; vitamin D could reduce NLRP3-mediated IL-1 signals and also help bone health during steroid therapy[43]. Direct RCT evidence in MG is lacking (Observational + mechanistic).
Sulforaphane	Cruciferous vegetables (broccoli sprouts)	Sulforaphane (isothiocyanate)	Activates Nrf2 → upregulates antioxidant enzymes (HO-1, thioredoxin), inhibits caspase-1 activation and IL-1β maturation downstream of NLRP3.	Strong antioxidant/Nrf2 activity may protect NMJ from inflammasome-driven injury; translational data promising in neuroinflammation but MG-specific evaluation absent (Preclinical).
Berberine	*Berberis* spp., Coptis	Berberine (isoquinoline alkaloid)	Reduces mtROS, stabilizes mitochondrial membrane potential, downregulates NLRP3 expression and caspase-1 activation; multiple studies show decreased IL-1β[37].	Mitochondrial stabilization and IL-1β reduction are relevant to MG inflammasome biology; evidence currently preclinical and model-based (Preclinical)[38].
Luteolin	Celery, parsley, chamomile	Luteolin (flavone)	Lowers ROS/mtROS, inhibits TXNIP-NLRP3 axis and ASC oligomerization, directs macrophage polarization (M1→M2) and reduces caspase-1/IL-1β[44].	By shifting innate inflammatory phenotype and reducing IL-1/IL-18, luteolin could modulate pathways implicated in MG; MG-specific data are not yet available (Preclinical)[44].
Ferulic acid	Cereal grains, rice bran, oats, fruits	Ferulic acid (phenolic acid)	Induces autophagy and SIRT1 signaling, reduces NLRP3/ASC /caspase-1 expression and IL-1β secretion in neuro inflammation models[45].	Autophagy/SIRT1-mediated suppression of NLRP3 is relevant to limiting IL-1 driven inflammation in MG; evidence currently preclinical (Preclinical)[45].

©Copyright protected. Unauthorised republication, reproduction, distribution, dissemination and copying of this document in whole or in part is strictly prohibited.

CONCLUSION

Targeting the NLRP3 inflammasome through dietary bioactives—including polyphenols, omega-3 fatty acids, sulforaphane, and vitamin D—represents a mechanistically rational adjunctive strategy for the management of myasthenia gravis. These compounds exert multi-level regulatory effects on inflammasome signaling, including suppression of NF-κB–mediated priming, attenuation of mitochondrial ROS generation, inhibition of ASC oligomerization, and modulation of caspase-1–dependent maturation of interleukin-1β and interleukin-18. Through these interconnected pathways, they hold potential to mitigate neuroinflammation and restore immune homeostasis in MG.

Despite compelling preclinical evidence, clinical translation remains in its early stages. Addressing key challenges such as limited bioavailability, pharmacokinetic variability, and incomplete understanding of drug–nutrient interactions will be critical. Future research should adopt mechanism-driven, biomarker-guided clinical trial designs, incorporating inflammasome-specific endpoints (e.g., IL-1β, caspase-1 activity, and pyroptosis markers) to establish target engagement and therapeutic efficacy. Additionally, advances in formulation science—such as nano-delivery systems and lipid-based carriers—may enhance systemic exposure and improve clinical outcomes.

Looking ahead, integrating NLRP3-targeted nutritional interventions with existing immunotherapies may enable a precision medicine approach, tailored to patient-specific inflammatory and immunological profiles. Such strategies have the potential to reduce treatment burden, improve safety, and enhance long-term disease control. Overall, while significant translational gaps remain, the convergence of immunology, nutrition science, and drug delivery technologies positions dietary bioactives as promising candidates in the evolving therapeutic landscape of myasthenia gravis.

Source of Support: The author(s) received no financial support for the research, authorship, and/or publication of this article

Conflict of Interest: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES

1. Salari N, Fatahi B, Bartina Y, Kazeminia M, Fatahian R, Mohammadi P, et al. Global prevalence of myasthenia gravis and the effectiveness of common drugs in its treatment: a systematic review and meta-analysis. J Transl Med. 2021 Dec;19(1):516. doi:10.1186/s12967-021-03185-7

2. Phillips WD, Vincent A. Pathogenesis of myasthenia gravis: update on disease types, models, and mechanisms. F1000Res. 2016 Jun 27;5:1513. doi:10.12688/f1000research.8206.1

3. Huda R. Inflammation and autoimmune myasthenia gravis. Front Immunol. 2023 Jan 30;14:1110499. doi:10.3389/fimmu.2023.1110499

4. Lazaridis K, Tzartos SJ. Autoantibody Specificities in Myasthenia Gravis; Implications for Improved Diagnostics and Therapeutics. Front Immunol. 2020 Feb 14;11:212. doi:10.3389/fimmu.2020.00212

5. Lazaridis K, Tzartos SJ. Myasthenia Gravis: Autoantibody Specificities and Their Role in MG Management. Front Neurol. 2020 Nov 30;11:596981. doi:10.3389/fneur.2020.596981

6. Marx A, Ströbel P, Weis CA. The pathology of the thymus in myasthenia gravis. Mediastinum. 2018 Dec;2:66–66. doi:10.21037/med.2018.12.04

7. Menon D, Barnett C, Bril V. Novel Treatments in Myasthenia Gravis. Front Neurol. 2020 Jun 30;11:538. doi:10.3389/fneur.2020.00538

8. Chen Y, Ye X, Escames G, Lei W, Zhang X, Li M, et al. The NLRP3 inflammasome: contributions to inflammation-related diseases. Cell Mol Biol Lett. 2023 Jun 27;28(1):51. doi:10.1186/s11658-023-00462-9

9. Wang L, Fu H, Nanayakkara G, Li Y, Shao Y, Johnson C, et al. Novel extracellular and nuclear caspase-1 and inflammasomes propagate inflammation and regulate gene expression: a comprehensive database mining study. J Hematol Oncol. 2016 Dec;9(1):122. doi:10.1186/s13045-016-0351-5

10. He Y, Hara H, Núñez G. Mechanism and Regulation of NLRP3 Inflammasome Activation. Trends in Biochemical Sciences. 2016 Dec;41(12):1012–21. doi:10.1016/j.tibs.2016.09.002

11. Li Y, Wang S, Liu Y, Zhang Y. Serum Pentaxin 3 (PTX3) Promotes NLRP3 Inflammasome and Pyroptosis in Patients with Up-Regulated Myasthenia Gravis. Front Biosci (Landmark Ed). 2023 Nov 28;28(11):306. doi:10.31083/j.fbl2811306

12. Coll RC, Hill JR, Day CJ, Zamoshnikova A, Boucher D, Massey NL, et al. MCC950 directly targets the NLRP3 ATP-hydrolysis motif for inflammasome inhibition. Nat Chem Biol. 2019 Jun;15(6):556–9. doi:10.1038/s41589-019-0277-7

13. Ma ZF, Fu C, Lee YY. The Modulatory Role of Bioactive Compounds in Functional Foods on Inflammation and Metabolic Pathways in Chronic Diseases. Foods. 2025 Feb 27;14(5):821. doi:10.3390/foods14050821

14. Schroder K, Tschopp J. The Inflammasomes. Cell. 2010 Mar;140(6):821–32. doi:10.1016/j.cell.2010.01.040

15. Zhang Y, Yang W, Li W, Zhao Y. NLRP3 Inflammasome: Checkpoint Connecting Innate and Adaptive Immunity in Autoimmune Diseases. Front Immunol. 2021 Oct 11;12:732933. doi:10.3389/fimmu.2021.732933

16. Benameur T, Frota Gaban SV, Giacomucci G, Filannino FM, Trotta T, Polito R, et al. The Effects of Curcumin on Inflammasome: Latest Update. Molecules. 2023 Jan 11;28(2):742. doi:10.3390/molecules28020742

17. Wang S, Li H, Zhang M, Yue LT, Wang CC, Zhang P, et al. Curcumin ameliorates experimental autoimmune myasthenia gravis by diverse immune cells. Neuroscience Letters. 2016 Jul;626:25–34. doi:10.1016/j.neulet.2016.05.020

18. Fu Y, Ge Y, Yi S, Peng Q, Jiang H, Zhou J. Smart nanoparticle delivery systems for curcumin: a targeted strategy to enhance anticancer efficacy and bioavailability. J Mater Sci: Mater Med. 2026 Feb 9;37(1):38. doi:10.1007/s10856-026-07012-7

19. Yu X, Chen M, Wu J, Song R. Research progress of SIRTs activator resveratrol and its derivatives in autoimmune diseases. Front Immunol. 2024 Jun 19;15:1390907. doi:10.3389/fimmu.2024.1390907

20. Yao J. Effect of resveratrol on Treg/Th17 signaling and ulcerative colitis treatment in mice. WJG. 2015;21(21):6572. doi:10.3748/wjg.v21.i21.6572

21. Guo NH, Fu X, Zi FM, Song Y, Wang S, Cheng J. The potential therapeutic benefit of resveratrol on Th17/Treg imbalance in immune thrombocytopenic purpura. International Immunopharmacology. 2019 Aug;73:181–92. doi:10.1016/j.intimp.2019.04.061

22. Lee HE, Yang G, Park YB, Kang HC, Cho YY, Lee HS, et al. Epigallocatechin-3-Gallate Prevents Acute Gout by Suppressing NLRP3 Inflammasome Activation and Mitochondrial DNA Synthesis. Molecules. 2019 Jun 6;24(11):2138. doi:10.3390/molecules24112138

23. Lee F, Bae KH, Ng S, Yamashita A, Kurisawa M. Hyaluronic acid–green tea catechin conjugates as a potential therapeutic agent for rheumatoid arthritis. RSC Adv. 2021;11(24):14285–94. doi:10.1039/D1RA01491A

©Copyright protected. Unauthorised republication, reproduction, distribution, dissemination and copying of this document in whole or in part is strictly prohibited.

24. Alattar A, Alshaman R, Althobaiti YS, Soliman GM, Ali HS, Khubrni WS, et al. Quercetin Alleviated Inflammasome-Mediated Pyroptosis and Modulated the mTOR/P70S6/P6/eIF4E/4EBP1 Pathway in Ischemic Stroke. Pharmaceuticals. 2023 Aug 21;16(8):1182. doi:10.3390/ph16081182

25. Domiciano TP, Wakita D, Jones HD, Crother TR, Verri WA, Arditi M, et al. Quercetin Inhibits Inflammasome Activation by Interfering with ASC Oligomerization and Prevents Interleukin-1 Mediated Mouse Vasculitis. Sci Rep. 2017 Feb 2;7(1):41539. doi:10.1038/srep41539

26. Tőzsér J, Benkő S. Natural Compounds as Regulators of NLRP3 Inflammasome-Mediated IL-1 β Production. Mediators of Inflammation. 2016;2016:1–16. doi:10.1155/2016/5460302

27. Liu P, Qi G, Gu S, Dong H, Liu C, Yang H. Single-cell transcriptomics and network pharmacology reveal therapeutic targets of Jianpi Yiqi Bugan Yishen decoction in immune cell subsets of children with myasthenia gravis. Transl Pediatr. 2022 Dec;11(12):1985–2003. doi:10.21037/tp-22-593

28. Shaikh SR, Rockett BD, Carraway K. Docosahexaenoic Acid Modifies the Clustering and Size of Lipid Rafts and the Lateral Organization and Surface Expression of MHC Class I of EL4 Cells. The Journal of Nutrition. 2009 Sep;139(9):1632–9. doi:10.3945/jn.109.108720

29. McCarty MF, Iloki Assanga SB, Lewis Luján L, O'Keefe JH, DiNicolantonio JJ. Nutraceutical Strategies for Suppressing NLRP3 Inflammasome Activation: Pertinence to the Management of COVID-19 and Beyond. Nutrients. 2020 Dec 25;13(1):47. doi:10.3390/nu13010047

30. Rao Z, Chen X, Wu J, Xiao M, Zhang J, Wang B, et al. Vitamin D Receptor Inhibits NLRP3 Activation by Impeding Its BRCC3-Mediated Deubiquitination. Front Immunol. 2019 Dec 4;10:2783. doi:10.3389/fimmu.2019.02783

31. Tunbridge M, França Gois PH. Vitamin D and the NLRP3 Inflammasome. Applied Sciences. 2020 Nov 27;10(23):8462. doi:10.3390/app10238462

32. Girgis CM, Cha KM, Houweling PJ, Rao R, Mokbel N, Lin M, et al. Vitamin D Receptor Ablation and Vitamin D Deficiency Result in Reduced Grip Strength, Altered Muscle Fibers, and Increased Myostatin in Mice. Calcif Tissue Int. 2015 Dec;97(6):602–10. doi:10.1007/s00223-015-0054-x

33. Askmark H, Haggård L, Nygren I, Punga AR. Vitamin D deficiency in patients with myasthenia gravis and improvement of fatigue after supplementation of vitamin D 3: a pilot study. Euro J of Neurology. 2012 Dec;19(12):1554–60. doi:10.1111/j.1468-1331.2012.03773.x

34. Sailo BL, Liu L, Chauhan S, Girisa S, Hegde M, Liang L, et al. Harnessing Sulforaphane Potential as a Chemosensitizing Agent: A Comprehensive Review. Cancers. 2024 Jan 5;16(2):244. doi:10.3390/cancers16020244

35. Gibbs A, Schwartzman J, Deng V, Alumkal J. Sulforaphane destabilizes the androgen receptor in prostate cancer cells by inactivating histone deacetylase 6. Proc Natl Acad Sci USA. 2009 Sep 29;106(39):16663–8. doi:10.1073/pnas.0908908106

36. Jiang Y, Huang K, Lin X, Chen Q, Lin S, Feng X, et al. Berberine Attenuates NLRP3 Inflammasome Activation in Macrophages to Reduce the Secretion of Interleukin-1β. Vol. 47. 2017;47(6).

37. Sun J, Zeng Q, Wu Z, Huang L, Sun T, Ling C, et al. Berberine inhibits NLRP3 inflammasome activation and proinflammatory macrophage M1 polarization to accelerate peripheral nerve regeneration. Neurotherapeutics. 2024 Jul;21(4):e00347. doi:10.1016/j.neurot.2024.e00347

38. Sarbadhikary P, George BP, Abrahamse H. Inhibitory Role of Berberine, an Isoquinoline Alkaloid, on NLRP3 Inflammasome Activation for the Treatment of Inflammatory Diseases. Molecules. 2021 Oct 15;26(20):6238. doi:10.3390/molecules26206238

39. Park CM, Jin KS, Cho CW, Lee YW, Huh GH, Cha YS, et al. Luteolin inhibits inflammatory responses by downregulating the JNK, NF-κB, and AP-1 pathways in TNF-α activated HepG2 cells. Food Sci Biotechnol. 2012 Feb;21(1):279–83. doi:10.1007/s10068-012-0037-x

40. Luo CJ, Li T, Li HL, Zhou Y, Li L. Resveratrol pretreatment alleviates NLRP3 inflammasome-mediated cardiomyocyte pyroptosis by targeting TLR4/MyD88/NF-κB signaling cascade in coronary microembolization-induced myocardial damage. Korean J Physiol Pharmacol. 2023 Mar 1;27(2):143–55. doi:10.4196/kjpp.2023.27.2.143

41. Mokra D, Joskova M, Mokry J. Therapeutic Effects of Green Tea Polyphenol (–)-Epigallocatechin-3-Gallate (EGCG) in Relation to Molecular Pathways Controlling Inflammation, Oxidative Stress, and Apoptosis. IJMS. 2022 Dec 25;24(1):340. doi:10.3390/ijms24010340

42. Baazm M, Behrens V, Beyer C, Nikoubashman O, Zendedel A. Regulation of Inflammasomes by Application of Omega-3 Polyunsaturated Fatty Acids in a Spinal Cord Injury Model. Cells. 2021 Nov 12;10(11):3147. doi:10.3390/cells10113147

43. Lu L, Zou G, Chen L, Lu Q, Wu M, Li C. Elevated NLRP3 Inflammasome Levels Correlate With Vitamin D in the Vitreous of Proliferative Diabetic Retinopathy. Front Med. 2021 Oct 15;8:736316. doi:10.3389/fmed.2021.736316

44. Zhang BC, Li Z, Xu W, Xiang CH, Ma YF. Luteolin alleviates NLRP3 inflammasome activation and directs macrophage polarization in lipopolysaccharide-stimulated RAW264.7 cells.

45. Liu Y, Shi L, Qiu W, Shi Y. Ferulic acid exhibits anti-inflammatory effects by inducing autophagy and blocking NLRP3 inflammasome activation. Mol Cell Toxicol. 2022 Oct;18(4):509–19. doi:10.1007/s13273-021-00219-5

46. Cai X, Fang Z, Dou J, Yu A, Zhai G. Bioavailability of Quercetin: Problems and Promises. CMC. 2013 May 1;20(20):2572–82. doi:10.2174/09298673113209990120

For any questions related to this article, please reach us at: globalresearchonline@rediffmail.com

New manuscripts for publication can be submitted at: submit@globalresearchonline.net and submit_ijpsrr@rediffmail.com

©Copyright protected. Unauthorised republication, reproduction, distribution, dissemination and copying of this document in whole or in part is strictly prohibited.