

Research Advances in the Biological Activities of Astragalin

Xichun HUANG, Quan QUAN, Junhao LI, Zhao NING, Hongye LI, Wenhui ZHAO, Chenghao JIN*

College of Life Science and Biotechnology, Heilongjiang Bayi Agricultural University, Daqing 163319, China

Abstract Astragalins exhibit a range of biological activities. This review summarizes recent research on the effects of astragalins in anti-inflammatory, anti-tumor, antioxidant, neuroprotective, and cardiovascular protective contexts. Regarding its anti-inflammatory properties, astragalins primarily mitigate inflammatory responses by inhibiting the MAPK and NF- κ B signaling pathways. In terms of anti-tumor activity, it modulates pathways such as PI3K/AKT and NF- κ B to suppress the proliferation and migration of various cancer cell types. Concerning neuroprotection, astragalins ameliorate neuronal damage in Alzheimer's disease models by activating PI3K/AKT-mTOR-mediated autophagy. Additionally, it reduces oxidative stress and ameliorates myocardial fibrosis associated with chronic heart failure. This review provides a theoretical foundation for the further investigation, development, and application of astragalins.

Key words Astragalins, Anti-inflammatory, Anti-tumor, Antioxidant, Neuroprotective, Heart failure

1 Introduction

Astragalins, also referred to as kaempferol-3-O-glucoside, kaempferol-3-glucoside, or kaempferol-3-O-glucoside, is a naturally occurring plant-derived compound. It is predominantly found in medicinal plants including *Potentilla discolor*^[1], *Cuscuta australis*^[2], *Rubus chingii*^[3], and *Thesium chinense*^[4]. The molecular formula of astragalins is $C_{21}H_{20}O_{11}$, and it has a molecular weight of 448.38 g/mol. This compound is readily soluble in organic solvents such as dimethyl sulfoxide (DMSO). Numerous studies have demonstrated that astragalins exhibit a variety of pharmacological effects within the field of biomedicine, including anti-inflammatory, anti-tumor, antioxidant, hypoglycemic, neuroprotective, and hepatoprotective activities. It has emerged as a prominent monomer in traditional Chinese medicine, attracting sustained research interest from both domestic and international scholars in recent years^[5]. This article provides a comprehensive review of the pharmacological effects and molecular mechanisms of astragalins, aiming to establish a theoretical foundation for its further development and clinical application.

2 Anti-inflammatory effects of astragalins

Inflammation is a natural defense mechanism elicited by biological tissues in response to harmful stimuli. Excessive inflammatory responses can contribute to the onset and progression of various diseases. Common clinical manifestations include redness, swelling, fever, and pain. Conventional treatment approaches primarily involve pharmacological and physical therapies. Recent studies have

demonstrated that astragalins exhibit significant anti-inflammatory properties.

Xing *et al.*^[6] examined the effects of astragalins on the progression of inflammatory bone destruction and elucidated its underlying mechanisms using experimental techniques including cell counting kit-8 (CCK-8) colorimetry, flow cytometry, and Western blot analysis. Their findings demonstrated that treatment with astragalins significantly reduced the mRNA and protein expression levels of cathepsin K, Fos proto-oncogene (c-Fos), activated T-cell nuclear factor 1 (NFATc1), and tartrate-resistant acid phosphatase (TRAP) at various stages of osteoclast differentiation. Additionally, the expression of reactive oxygen species (ROS) scavenging enzymes regulated by nuclear factor E_2 -related factor 2 (Nrf2) was markedly upregulated. These results suggest that astragalins mitigate inflammatory bone destruction by down-regulating the mitogen-activated protein kinase (MAPK) signaling pathway, thereby exerting anti-inflammatory effects.

Peng *et al.*^[7] utilized experimental approaches, including colitis mouse model, histopathological observation, 16S rRNA high-throughput sequencing, and Western blotting, to investigate the intervention effects and molecular mechanisms of astragalins on DSS-induced acute experimental colitis. Their findings demonstrated that astragalins treatment significantly ameliorated colitis-associated phenotypes, such as weight loss, colonic shortening, and histopathological damage in mice. Furthermore, astragalins effectively restored the balance of the intestinal microbiota and markedly inhibited the activation of the nuclear factor- κ B (NF- κ B) signaling pathway in colonic tissue. These results suggest that astragalins mitigate intestinal flora dysbiosis and suppresses NF- κ B pathway activation, thereby attenuating intestinal inflammatory responses and ultimately conferring a protective effect against DSS-induced acute colitis in mice.

Han *et al.*^[8] examined the effects of astragalins on the NF- κ B signaling pathway in human colonic epithelial cells and its therapeutic potential in experimental colitis. Utilizing methods such as Western blot analysis, histopathological staining, human colonic

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Xichun HUANG, master's degree candidate, research fields: pharmacology of anti-cancer drugs. *Corresponding author. Chenghao JIN, doctoral degree, professor, research fields: pathogenesis of cancer and drug development.

epithelial cell culture, and the establishment of a mouse model of experimental colitis, the study demonstrated that astragalín treatment significantly inhibited NF- κ B pathway activation in human colonic epithelial cells. Furthermore, astragalín effectively ameliorated symptoms associated with experimental colitis in mice, reducing intestinal mucosal inflammatory damage and inflammatory cell infiltration. These findings suggest that astragalín mitigates intestinal inflammatory responses by suppressing NF- κ B signaling activation in human colonic epithelial cells, thereby exerting a protective effect in experimental colitis models.

Ma Xiaofei *et al.* [9] examined the effects and underlying mechanisms of astragalín in mitigating airway inflammation in asthmatic mice using experimental approaches including the establishment of a mouse asthma model, histopathological staining, enzyme-linked immunosorbent assay (ELISA), and Western blot analysis. Their findings demonstrated that astragalín significantly alleviated pathological alterations such as airway inflammatory cell infiltration, mucus hypersecretion, and airway remodeling in asthmatic mice. Additionally, astragalín reduced the levels of airway inflammatory mediators and inhibited the activation of associated inflammatory signaling pathways. These results suggest that astragalín ameliorates airway pathological damage by attenuating airway inflammatory responses and suppressing the activation of inflammation-related signaling pathways, thereby exerting an inhibitory effect on airway inflammation in asthmatic mice.

3 Anti-tumor effects of astragalín

Malignant tumors are diseases characterized by the uncontrolled proliferation of abnormal cells. A defining feature of these tumors is their ability to proliferate indefinitely, and invade surrounding tissues and organs. Common clinical treatment modalities include surgery, radiotherapy, and targeted therapy. Recent studies have reported that astragalín exhibits significant anti-tumor effects.

Yang *et al.* [10] examined the effects of astragalín on the proliferation and migration of human colon cancer HCT116 cells, as well as its underlying mechanisms, employing experimental techniques including cell counting, cell scratch assay, Transwell migration assay, and Western blot analysis. Their findings demonstrated that treatment with astragalín significantly inhibited the proliferation and migration capacities of HCT116 cells, markedly reduced the phosphorylation level of NF- κ B p65 protein, and substantially prevented the degradation of I κ B- α protein. These results suggest that astragalín suppresses the proliferation and migration of human colon cancer HCT116 cells by modulating the NF- κ B signaling pathway and downregulating the expression of downstream proteins associated with proliferation and migration.

Wang *et al.* [11] examined the anti-tumor effects and underlying mechanisms of astragalín on gastric cancer cells using experimental approaches including cell proliferation assays, colony formation assays, Transwell assays, and Western blot analysis. Their findings demonstrated that treatment with astragalín significantly

inhibited the proliferation, colony formation, migration, and invasion capabilities of gastric cancer cells, while markedly increasing apoptosis levels. Additionally, astragalín treatment resulted in a significant downregulation of the phosphorylation levels of PI3K and AKT proteins. These results suggest that astragalín suppresses the malignant phenotypes of gastric cancer cells by inhibiting the activation of the PI3K/AKT signaling pathway, thereby exerting an anti-tumor effect.

Liang Yong *et al.* [12] examined the effects of astragalín on the proliferation, migration, apoptosis, and oxidative stress of lung cancer A549 cells, as well as its underlying mechanisms, employing experimental techniques including CCK-8 colorimetry, cell scratch assay, flow cytometry, Western blotting, and assessment of oxidative stress-related markers. Their findings demonstrated that treatment with astragalín significantly inhibited the proliferation and migration capacities of A549 cells, markedly increased the apoptosis rate, significantly reduced intracellular ROS levels, and notably enhanced superoxide dismutase (SOD) activity. Furthermore, astragalín treatment resulted in a significant downregulation of Bcl-2 protein expression and upregulation of Bax and Caspase-3 protein levels. These results suggest that astragalín suppresses proliferation and migration while promoting apoptosis in lung cancer A549 cells by modulating apoptosis-related protein expression and mitigating oxidative stress, thereby exerting an anti-lung cancer effect.

Zhu Zhiheng *et al.* [13] examined the effects of astragalín on the apoptosis of undifferentiated gastric cancer cells and elucidated its underlying mechanisms using experimental techniques including CCK-8 colorimetry, flow cytometry, and Western blot analysis. Their findings demonstrated that treatment with astragalín significantly inhibited the proliferation of undifferentiated gastric cancer cells and markedly increased the apoptosis rate. Additionally, astragalín treatment resulted in a significant downregulation of Bcl-2 protein expression, alongside significant upregulation of Bax, Caspase-3, and Caspase-9 protein levels, thereby activating apoptosis-related signaling pathways. These results suggest that astragalín promotes apoptosis in undifferentiated gastric cancer cells by modulating the expression of apoptosis-associated proteins and activating apoptotic signaling cascades, ultimately exerting an anti-tumor effect against undifferentiated gastric cancer.

Wang Yu *et al.* [14] examined the effects of astragalín on apoptosis in the diffuse large B-cell lymphoma (DLBCL) OCI-LY8 cell line and elucidated its underlying mechanisms using experimental techniques including CCK-8 colorimetry, flow cytometry, and Western blot analysis. Their findings demonstrated that treatment with astragalín significantly inhibited the proliferative capacity of OCI-LY8 cells and markedly increased the apoptosis rate. In addition, astragalín treatment resulted in a significant downregulation of Bcl-2 protein expression, alongside significant upregulation of Bax, Caspase-3, and Caspase-9 protein levels. Concurrent activation of apoptosis-related signaling pathways was also observed.

These results suggest that astragaloside induces apoptosis in the DLBCL OCI-LY8 cell line by modulating the expression of apoptosis-associated proteins and activating apoptotic signaling pathways, thereby exerting an anti-DLBCL effect.

4 Neuroprotective effects of astragaloside

Alzheimer's disease (AD) is a prevalent neurodegenerative disorder commonly affecting the elderly population. It is typically characterized by cognitive decline, emotional disturbances, social withdrawal, and impaired self-care abilities, all of which significantly compromise patients' quality of life. Currently, clinical treatment primarily focuses on symptomatic management and slowing disease progression. Recent studies have indicated that astragaloside exhibits notable neuroprotective properties.

Utilizing experimental approaches including the development of APP/PS1 transgenic mouse models, immunohistochemical staining, Western blot analysis, and assessment of autophagy-related markers, Yang *et al.*^[15] investigated the neuroprotective effects and underlying mechanisms of astragaloside in APP/PS1 mice through activation of PI3K/AKT-mTOR-mediated autophagy. Their findings demonstrated that astragaloside treatment significantly mitigated neuronal damage in the cortex and hippocampus, markedly reduced senile plaque deposition, increased the LC3 II/LC3 I ratio, upregulated Beclin-1 protein expression, and downregulated p62 protein expression. Additionally, phosphorylation levels of PI3K, AKT, and mTOR proteins were significantly elevated, indicating activation of the PI3K/AKT-mTOR signaling pathway. These results suggest that astragaloside exerts neuroprotective effects by activating PI3K/AKT-mTOR-mediated autophagy and modulating the expression of autophagy-related proteins, thereby alleviating neuronal injury and senile plaque accumulation in APP/PS1 mice.

Wang Xiaonan *et al.*^[16] examined the protective effects and underlying mechanisms of astragaloside on bone marrow microvascular endothelial cells (BMECs) subjected to ischemic injury using experimental approaches including cell culture, CCK-8 colorimetry, lactate dehydrogenase (LDH) activity assays, and Western blot analysis. Their findings demonstrated that astragaloside treatment significantly increased the survival rate of ischemic BMECs, markedly reduced LDH release, and decreased the apoptosis rate. Furthermore, astragaloside significantly upregulated the expression of the anti-apoptotic protein Bcl-2 while downregulating the pro-apoptotic proteins Bax and Caspase-3. Additionally, astragaloside alleviated ischemia-induced inflammatory responses and oxidative stress, concomitantly modulating and activating related signaling pathways. These results suggest that astragaloside confers protection to BMECs under ischemic injury by regulating apoptosis-related protein expression, mitigating inflammation and oxidative damage, thereby exerting an ischemic protective effect.

5 Antioxidant effects of astragaloside

Oxidative stress is a pathological condition characterized by an ex-

cessive accumulation of ROS within the body, surpassing its intrinsic antioxidant defenses. This imbalance leads to impaired cellular functions and the development of various diseases. Free radicals can induce a range of damages, including oxidative injury to cell membranes, DNA strand breaks, and disruption of protein structures. The antioxidant function denotes the body's capacity to counteract such damage. Recent studies have demonstrated that astragaloside exhibits significant antioxidant properties.

Luo Haijing *et al.*^[17] examined the impact of astragaloside on the expression of LncRNA-MEG3 in the muscle tissue of mice subjected to oxidative damage, as well as the underlying mechanisms. Their study employed experimental approaches including the establishment of a mouse model of oxidative damage, real-time quantitative PCR (qRT-PCR), and the assessment of oxidative stress-related biomarkers. The findings demonstrated that astragaloside treatment significantly reduced oxidative stress levels in the muscle tissue of affected mice, markedly enhanced antioxidant capacity, and substantially downregulated LncRNA-MEG3 expression. These results suggest that astragaloside mitigates oxidative damage in mouse muscle tissue by downregulating LncRNA-MEG3 expression and alleviating oxidative stress injury, thereby exerting a protective effect on the damaged muscle tissue.

Zhang Wentian *et al.*^[18] investigated the reparative effects and underlying mechanisms of astragaloside on hepatic oxidative damage in a chronic sleep deprivation mouse model. The study employed biochemical assays, histopathological staining, and qRT-PCR analyses. The results demonstrated that astragaloside treatment significantly decreased malondialdehyde (MDA) levels in liver tissue, increased the activities of antioxidant enzymes such as SOD and glutathione peroxidase (GSH-Px), and markedly alleviated liver pathological damage. Furthermore, oxidative stress-related signaling pathways were effectively modulated. These findings suggest that astragaloside enhances hepatic antioxidant capacity, reduces lipid peroxidation-induced damage, and thereby mitigates liver oxidative injury induced by chronic sleep deprivation, ultimately conferring hepatoprotective effects.

6 Anti-heart failure effects of astragaloside

Heart failure is a highly prevalent cardiovascular condition characterized primarily by the heart's inadequate pumping capacity, resulting in insufficient blood and oxygen delivery to tissues and organs throughout the body. It frequently arises secondary to primary cardiac diseases, including coronary artery disease, hypertension, and cardiomyopathy. Common clinical manifestations include dyspnea, fatigue, and peripheral edema. In severe cases, heart failure may precipitate sudden cardiac death. Therapeutic strategies primarily focus on enhancing cardiac function and slowing disease progression. Recent studies have indicated that astragaloside exhibits significant cardioprotective effects.

Shao Huimin *et al.*^[19] examined the effects of astragaloside on myocardial fibrosis and the AMPK/Nrf2 signaling pathway in a rat

model of chronic heart failure, employing histopathological staining, ELISA, and Western blot analyses to elucidate the underlying mechanisms. Their findings demonstrated that astragalín treatment significantly attenuated myocardial fibrosis and collagen deposition. Additionally, it markedly increased AMPK protein phosphorylation and Nrf2 nuclear translocation, upregulated the expression of downstream antioxidant proteins, and reduced myocardial inflammation and oxidative stress levels. These results suggest that astragalín mitigates myocardial fibrosis by activating the AMPK/Nrf2 pathway and enhancing antioxidant defenses, thereby conferring cardioprotective effects in rats with chronic heart failure.

7 Conclusions and prospects

In recent years, numerous studies have demonstrated that astragalín, a natural bioactive compound with diverse pharmacological effects, exhibits minimal toxicity and side effects in humans, indicating significant potential for development and application. However, the majority of research on astragalín remains confined to pharmacological characterization and animal studies. Comprehensive and systematic investigations into its mechanisms of action and clinical applications are necessary to establish a robust theoretical foundation for the development of astragalín as a safe, effective, and low-toxicity therapeutic agent.

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