Research Advances in the Pharmacological Effects and Molecular Mechanisms of Harmine

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Abstract This paper provides a systematic review of the anti-tumor, anti-inflammatory, antibacterial, and anti-echinococcosis effects of harmine, as well as its role in regulating osteogenic differentiation and protecting the central nervous system, along with the underlying molecular mechanisms. Harmine exhibits anti-tumor properties by inhibiting the proliferation of gastric cancer, lung cancer, and other cancer cell types, inducing apoptosis, and impeding migration and invasion through pathways such as PTEN/AKT/MDM2. Regarding its anti-inflammatory and antibacterial effects, harmine reduces inflammation by modulating the NF-κB/NLRP3 pathway and exerts antibacterial activity by disrupting bacterial structures. Furthermore, harmine regulates the Th1/Th2 balance to produce anti-echinococcosis effects, promotes osteogenic differentiation by mediating the antioxidant activity of CA-9, and ameliorates anxious behavior through the regulation of neuroinflammation. This review offers a theoretical foundation for the further development and clinical application of harmine.

Key words Harmine, Anti-tumor, Anti-inflammatory, Antibacterial, Anti-echinococcosis, Osteogenic differentiation, Neuroprotective

1 Introduction

Peganum harmala, a perennial herb, is extensively distributed across the Middle East, North Africa, and certain regions of Asia. It is traditionally utilized for its therapeutic properties, including alleviation of cough and asthma, elimination of dampness, detoxification, as well as the dispelling of wind and relief of itching. Harmine, a β-carboline alkaloid compound derived from *P. harmala*, has the molecular formula $C_{13}H_{12}N_2O$ and a molecular weight of 212.25 g/mol[1-2]. This compound typically appears as a white crystalline powder and is soluble in organic solvents such as chloroform and methanol. Numerous studies have demonstrated that harmine exhibits a variety of pharmacological effects, including anti-tumor, anti-inflammatory, antibacterial, anti-echinococcosis activities, as well as the regulation of osteogenic differentiation and the protection of the central nervous system[3]. This paper aims to review the research progress concerning the pharmacological effects and molecular mechanisms of harmine, with the objective of providing a theoretical foundation for its further study, development, and application.

2 Anti-tumor effects of harmine

A malignant tumor is a pathological condition characterized by the transformation of normal cells into cells that undergo uncontrolled and indefinite proliferation due to prolonged exposure to specific carcinogenic factors. Malignant tumors exhibit features such as unlimited growth, high invasiveness, and a strong propensity for metastasis[4]. Research has demonstrated that harmine can effectively inhibit the proliferation of various tumor cells, induce apoptosis, and suppress both invasion and migration.

2.1 Inhibitory effects of harmine on the proliferation of tumor cells Tao Tingting *et al.*[5] investigated the impact of harmine on the expression levels of proliferation-related proteins in human gastric cancer SGC-7901 cells using Western blot analysis. The results demonstrated that treatment with harmine significantly increased the expression of the *PTEN* gene in SGC-7901 cells, while the expression levels of protein kinase B (AKT), phosphorylated mouse double minute 2 (p-MDM2), and cyclooxygenase-2 (COX-2) were significantly decreased. The impact of harmine on the expression levels of proliferation-related proteins in human gastric cancer SGC-7901 cells following knockdown of the *PTEN*, *AKT*, and *MDM2* genes was investigated using cell transfection technology and Western blot analysis. The results demonstrated that *PTEN* gene knockdown effectively suppressed the inhibitory effect of harmine on the expression of AKT, p-MDM2, and COX-2. Conversely, *AKT* gene knockdown synergistically enhanced the inhibitory effects of harmine on p-MDM2 and COX-2 expression. Similarly, knockdown of *MDM2* synergistically suppressed COX-2 expression in combination with harmine treatment. These findings suggest that harmine downregulates COX-2 expression via the PTEN/AKT/MDM2 signaling pathway, thereby inhibiting the proliferation of gastric cancer cells.

Rao Mingjun[6] investigated the effect of combining harmine with B-cell lymphoma-2 (*Bcl-2*) gene inhibitors (ABT-199/ABT-737) on the proliferative capacity of non-small cell lung cancer (NSCLC) cells using the sulforhodamine B assay. The results demonstrated that the cell survival rate was significantly reduced when harmine was combined with *Bcl-2* inhibitors compared to the use of either agent alone. The impact of harmine on the expression levels of proteins associated with the proliferation of NSCLC cells was further investigated using colony formation assays and Western

Received: February 12, 2026 Accepted: May 8, 2026

Supported by Central Government Funds for Local University Reform and Development (Talent Cultivation Program) (2020GSP16); Undergraduate Innovation and Entrepreneurship Training Program of Heilongjiang Province (202510223002); Postgraduate Innovation Research Project of Heilongjiang Bayi Agricultural University (YJSCX2025-KJQN66).

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blot analysis. The results demonstrated that treatment with harmine significantly downregulated the expression of myeloid leukemia-1 (Mcl-1) protein in NSCLC cells. These findings suggest that harmine can overcome resistance to *Bcl-2* inhibitors by promoting the degradation of Mcl-1 protein in NSCLC cells, thereby achieving a synergistic inhibitory effect on NSCLC cell proliferation when combined with *Bcl-2* inhibitors.

2.2 Apoptosis-inducing effects of harmine on tumor cells Sun Kun[7] investigated the apoptosis-inducing effects of harmine on gastric cancer cell lines BGC-823 and SGC-7901 using flow cytometry. The findings demonstrated that treatment with harmine at concentrations of 0, 4, 8, and 16 μmol/L resulted in a dose-dependent increase in the proportion of early apoptotic cells in both cell lines. Further analysis via Western blotting revealed that harmine treatment significantly decreased the expression of the protein Bcl-2, while significantly increasing the expression of the Bcl-2-associated X protein (Bax) in BGC-823 and SGC-7901 cells. These results suggest that harmine induces apoptosis in human gastric cancer BGC-823 and SGC-7901 cells by downregulating Bcl-2 expression.

Liu Junling *et al.*[8] investigated the apoptosis-inducing effect of harmine on acute T-lymphoblastic leukemia Jurkat cells using agarose gel electrophoresis. The results demonstrated that, following treatment with harmine, Jurkat cells exhibited characteristic ladder-like bands on agarose gels, whereas the DNA from the control group remained concentrated near the loading wells. These findings indicate that harmine induces significant DNA fragmentation in Jurkat cells, thereby promoting apoptosis. The impact of harmine on the expression levels of apoptosis-related proteins in Jurkat cells was further evaluated using Western blot analysis. The results demonstrated that treatment with harmine at concentrations of 0, 2.5, 5, and 10 μmol/L significantly reduced the expression levels of Bcl-2 and ICAD proteins in Jurkat cells. These findings suggest that harmine induces apoptosis in acute T-lymphoblastic leukemia Jurkat cells by modulating the expression of the apoptosis-related proteins Bcl-2 and ICAD.

Liu Jiren[9] investigated the apoptosis-inducing effects of harmine on human gastric adenocarcinoma SGC-7901 cells using flow cytometry. The findings demonstrated that harmine significantly increased the apoptosis rate of SGC-7901 cells in a concentration-dependent manner at concentrations of 0, 4, 8, and 16 μmol/L. Furthermore, the impact of harmine on the expression levels of apoptosis-related proteins in SGC-7901 cells was assessed via Western blot analysis. The results indicated that treatment with harmine at concentrations of 3, 4, 5, 6, and 7 mg/mL led to a significant increase in the expression of the pro-apoptotic protein Fas, alongside a significant decrease in the expression of the anti-apoptotic protein Bcl-2. These findings suggest that harmine induces apoptosis in human gastric adenocarcinoma SGC-7901 cells by modulating the expression of apoptosis-related proteins Fas and Bcl-2.

2.3 Effects of harmine on the migration and invasion of tumor cells Wang Meilin *et al.*[10] investigated the effect of harmine on the migratory capacity of human breast cancer MCF-7 cells using a cell scratch assay. The results demonstrated that treatment with harmine at concentrations of 0, 5, 10, and 20 μmol/L significantly decreased the migration rate of MCF-7 cells. Additionally, the impact of harmine on the invasive ability of MCF-7 cells was assessed via a Transwell chamber assay. The findings indicated that harmine treatment significantly reduced the number of cells penetrating the Matrigel matrix. The impact of harmine on the expression levels of proteins associated with migration and invasion in MCF-7 cells was further evaluated using Western blot analysis. The results demonstrated that treatment with harmine significantly reduced the intracellular levels of transforming growth factor-β (TGF-β), Smad2, Smad3, Smad4, N-cadherin, vimentin, and signal transducer and activator of transcription 3 (Stat3) in MCF-7 cells. These findings suggest that harmine inhibits the migration and invasion of human breast cancer MCF-7 cells by modulating the TGF-β signaling pathway, thereby exerting an anti-tumor effect.

Sun Hongyu *et al.*[11] investigated the effects of harmine on the migratory and invasive capacities of human osteosarcoma Saos-2 cells using cell scratch and Transwell chamber assays. The findings demonstrated that treatment with harmine at concentrations of 0, 5, 10, and 20 μmol/L significantly reduced both the migration rate and the number of invading Saos-2 cells. Furthermore, the anti-tumor efficacy of harmine was evaluated in a nude mouse tumor model via TUNEL immunostaining. Results indicated that harmine treatment led to a significant increase in apoptosis rates of Saos-2 cells, accompanied by a marked decrease in their migratory ability. These findings suggest that harmine exerts anti-tumor effects by inhibiting the migration and invasion of osteosarcoma Saos-2 cells.

3 Anti-inflammatory and antibacterial effects of harmine

3.1 Anti-inflammatory effects of harmine Inflammation is a fundamental pathological process primarily serving as a defense mechanism that occurs in the body in response to damage induced by various inflammatory stimuli. The inflammatory response involves multiple components, including inflammatory cells, cytokines, mediators, adhesion molecules, chemokines, and growth factors[12]. Research has demonstrated that harmine can alleviate inflammatory symptoms by inhibiting the expression of associated inflammatory factors.

Tan Yanjie *et al.*[13] investigated the protective effects of harmine on the intestinal mucosal barrier in a murine model of inflammatory bowel disease (IBD) using HE staining. Their findings demonstrated that harmine treatment significantly ameliorated pathological damage to the colonic mucosa and submucosal epithelium, accompanied by a marked reduction in inflammatory cell in-

filtration. Further analysis via Western blotting revealed that harmine administration led to a significant upregulation of the proteins ZO-1 and Occludin in the intestinal tissues of IBD model mice. These results suggest that harmine exerts anti-inflammatory effects by modulating the expression of ZO-1 and Occludin, thereby enhancing intestinal barrier function and decreasing mucosal permeability.

Niu Xiaofeng *et al.* [14] investigated the effects of harmine on mouse serum and kidney tissue using enzyme-linked immunoblotting. Their findings demonstrated that harmine treatment significantly reduced the secretion levels of tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) in mouse serum. The potential anti-inflammatory mechanism of harmine in the context of LPS-induced acute kidney injury was further examined through Western blotting and immunohistochemical analyses. Results indicated that harmine treatment induced phosphorylation of nuclear factor κB p65 (NF-κB p65) and nuclear factor κB inhibitor protein α (IκBα), accompanied by a marked decrease in IκBα expression in mice with acute kidney injury. Moreover, harmine significantly suppressed LPS-induced activation of the NLRP3 inflammasome by downregulating the expression of nuclear nucleotide-binding and oligomerization domain-like receptor 3 (NLRP3), Caspase-1, and interleukin-1β (IL-1β) in renal tissue. These findings suggest that harmine mitigates LPS-induced acute kidney injury in mice by suppressing the TLR4-NF-κB/NLRP3 signaling pathway.

3.2 Antibacterial effects of harmine Microbe refers to the general term for tiny organisms that are not easily visible to the naked eye, encompassing bacteria, fungi, viruses, *etc.* Microbes are characterized by their small size, diverse species, rapid reproduction rates, and strong adaptability to various environments[15]. Research has demonstrated that harmine exerts antibacterial effects by disrupting the structural integrity of bacteria, fungi, and other microbes, as well as interfering with their metabolic processes.

Xu Yan *et al.* [16] investigated the effects of harmine on six bacterial species—*Staphylococcus aureus*, beta-hemolytic streptococcus, *Escherichia coli*, *Pseudomonas aeruginosa*, *Shigella fortunei*, and *Salmonella typhi*—as well as one fungal species, *Candida albicans*, using the minimum bactericidal concentration (MBC) assay. The findings indicated that the MBC of harmine ranged from 125 to 250 μg/mL for the six bacterial species and was 800 μg/mL for *C. albicans*. The bactericidal rate of harmine was evaluated at various time points (0, 0.5, 1, 2, 4, 6, 8, 10, 24, and 48 h) by constructing *in vitro* time-kill curves for *P. aeruginosa*, *S. aureus*, and *C. albicans*. The results demonstrated that treatment with harmine resulted in bactericidal rates exceeding 90% for all three microorganisms within 4 h. Furthermore, the bactericidal rates for *S. aureus* and *P. aeruginosa* reached 100% within 24 h. These findings suggest that harmine holds significant potential for application in combating bacterial and fungal infections, particularly those affecting superficial tissues.

Xia Hongkai *et al.* [17] evaluated the bactericidal activity of harmine against *Ralstonia solanacearum* using the minimum inhibitory concentration (MIC) assay. The findings demonstrated that treatment with harmine at 60 mg/L for 1 and 2 h resulted in mortality rates of 85% and 90%, respectively. When treated with harmine at 180 mg/L for 1 and 2 h, mortality rates increased to 96% and 98%, respectively. Furthermore, exposure to harmine at 240 mg/L for 2 h yielded a mortality rate of 99.3%. The impact of harmine on the expression of virulence-related genes in *R. solanacearum* was further assessed via quantitative real-time polymerase chain reaction (qPCR). Results indicated that harmine treatment significantly inhibited the expression of the bacterial virulence-related gene (*xpsR*) and reduced the expression of the extracellular protein secretion E gene (*espE*). These findings suggest that harmine exerts its antibacterial effects by downregulating the expression of *xpsR* and *espE* in *R. solanacearum*.

4 Other effects of harmine

4.1 Anti-echinococcosis effects of harmine Echinococcosis, also referred to as hydatid disease, is a zoonotic infection caused by the larvae of *Echinococcus granulosus* parasitizing humans and certain animal hosts. This condition is also characterized by immune system dysregulation[18]. Research has demonstrated that harmine can inhibit the growth of parasites by suppressing the expression of specific cytokines.

Chen Bei *et al.* [19] investigated the effects of harmine on T lymphocyte subsets in the peripheral blood of mice following *E. granulosus* infection using flow cytometry. Their findings demonstrated that harmine treatment resulted in a significant decrease in the proportion of CD4 + T cells and a significant increase in CD8 + T cells in the peripheral blood. Additionally, the impact of harmine on Th1/Th2-related cytokine levels in mouse serum was assessed through blood cell analysis. The results indicated that harmine administration significantly elevated the expression of interleukin-2 (IL-2) and interferon-gamma (IFN-γ), cytokines secreted by Th1 cells, while significantly reducing the levels of interleukin-4 (IL-4) and interleukin-10 (IL-10), which are secreted by Th2 cells. These findings suggest that harmine modulates the Th1/Th2 cell imbalance induced by *E. granulosus* infection by altering the expression of Th1/Th2-associated cytokines, thereby inhibiting parasite growth.

4.2 Neuroprotective effects of harmine The nervous system serves as the regulatory center for the activities of the body's organs. When degenerative changes occur within the nervous system, an inflammatory response is triggered. Excessive neuroinflammation can inflict further damage on surrounding tissues, thereby posing a serious threat to human health. Research has demonstrated that harmine exerts significant therapeutic effects by protecting nerve cells and ameliorating neurological dysfunction. Zheng Zhiheng *et al.* [20] examined the effects of harmine on excitatory synaptic transmission using whole-cell patch-clamp recordings. Their findings demonstrated that harmine treatment caused a

significant rightward shift in the cumulative probability curve of the frequency of miniature excitatory postsynaptic currents (mEPSCs), whereas the amplitude distribution did not exhibit a significant rightward shift. Inhibition of LPS increased the frequency of mEPSCs in projection neurons of the basolateral amygdala (BLA PNs). To further assess harmine's impact on synaptic transmission inhibition in BLA PNs, miniature inhibitory postsynaptic currents (mIPSCs) were recorded. The results indicated no significant changes in either the frequency or amplitude of mIPSCs across all experimental groups. These findings suggest that harmine exerts an anxiolytic effect by modulating neuroinflammation and neuronal plasticity within the basolateral amygdala.

4.3 Effect of harmine on osteogenic differentiation Osteogenic differentiation is the process by which precursor cells, such as mesenchymal stem cells (MSCs), progressively differentiate into osteoblasts in response to various intrinsic and extrinsic stimuli. These osteoblasts are responsible for secreting the bone matrix and regulating bone mineralization. Research has demonstrated that harmine can mitigate the impairment of osteogenic differentiation capacity by modulating the expression levels of associated cytokines.

Xu Wen[21] investigated the effects of harmine on the expression of osteogenic genes and proteins in stem cells derived from human exfoliated deciduous teeth (SHEDs) under hypoxic conditions using protein immunoblotting. The findings demonstrated that, under hypoxia, harmine treatment significantly upregulated the expression of carbonic anhydrase 9 (CA-9) in SHEDs. Following the evaluation of CA-9 inhibitory activity using a CA-9 inhibitor, the impact of harmine on the osteogenic capacity of the cells was assessed. The results demonstrated that the expression levels of the osteogenic markers Runt-related transcription factor 2 (RUNX2), type I collagen (COL-1), and osteopontin (OPN) were significantly downregulated, resulting in the loss of the osteoprotective effect of harmine on SHEDs. Additionally, fluorescence microscopy was employed to evaluate the effects of harmine on reactive oxygen species (ROS) and malondialdehyde (MDA) levels in SHEDs. The results indicated that harmine treatment significantly suppressed the production of ROS and MDA. These findings suggest that harmine mitigates hypoxia-induced damage in SHEDs by modulating the antioxidant activity of CA-9.

5 Prospects

Harmine, a natural compound exhibiting diverse pharmacological activities, holds significant potential for applications in antitumor, anti-inflammatory, antibacterial therapies, and the regulation of osteogenic differentiation. Although numerous studies have investigated harmine, most have been confined to preliminary examinations of its pharmacological effects, lacking comprehensive and systematic analyses of the underlying molecular mechanisms and clinical applications. Therefore, it is imperative to integrate disciplines such as molecular biology, structural biology, physiology, molecular pharmacology, and biopharmaceutics to conduct more in-depth fundamental research at the molecular, cellular, and animal levels. Such efforts will provide a robust theoretical foundation for the further development and utilization of harmine.

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sponse. Consequently, LPS is frequently employed in the development of various inflammatory models[8]. Macrophages, as primary immune cells, play an essential role in immune defense by phagocytosing and eliminating foreign pathogens and maintaining immune homeostasis. However, under pathological conditions, hyperactivation of macrophages can amplify inflammatory responses, leading to tissue damage and contributing significantly to the pathogenesis of inflammatory diseases such as pneumonia, arthritis, and pancreatitis[9]. Under LPS stimulation, macrophages exhibit a significant increase in the release of inflammatory cytokines, including TNF-α, IL-1β, and IL-6, thereby initiating an inflammatory response[10]. The present study demonstrated that the concentrations of TNF-α, IL-1β, and IL-6 in the supernatant of RAW264.7 cells were markedly elevated following LPS induction. Treatment with anwulignan effectively inhibited the release of these inflammatory cytokines in LPS-stimulated RAW264.7 cells. These findings suggest that anwulignan can attenuate the inflammatory response induced by LPS. Furthermore, the observed anti-inflammatory activity of anwulignan indicates that it may represent a key bioactive component of *K. longipedunculata* responsible for its anti-inflammatory properties and potential therapeutic effects in rheumatoid arthritis. Future studies will aim to validate its efficacy in animal models.

In summary, this study established the characteristic HPLC chromatogram of *K. longipedunculata* medicinal materials and developed a method for determining the content of anwulignan. Anwulignan may represent the active component of *K. longipedunculata* responsible for its anti-inflammatory effects. This research offers a more comprehensive reference for the quality evaluation of *K. longipedunculata* medicinal materials.

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