Proceedings of ICMMGH 2026 Symposium: Cell and Molecular Biology: Understanding Immune Cell Functions
DOI: 10.54254/2753-8818/2026.PJ30389

Researsh Progress on the Chemical Constituents, Pharmacological Activity and Related Signaling Pathways of Coptis Chinensis Franch

Guochuan Xu

School of Pharmacy and Medicine, Ocean University of China, Qingdao, China
15253334169@163.com

Abstract. Coptis chinensis Franch. is a representative herb in traditional Chinese medicine. Its core active components are isoquinoline alkaloids, with berberine being the most abundant, along with palmatine, jatrorrhizine, coptisine, and others. Coptis chinensis Franch. exhibits various pharmacological activities, including antibacterial and anti-endotoxin effects, antiviral activity, hypoglycemic action, and anticancer properties. Berberine, one of its key components, functions through multiple targets by modulating pathways such as the AMPK/PI3K-Akt signaling pathway, the AMPK/mTOR autophagy regulation pathway, and the TLR4/MyD88 inflammatory signaling pathway.

Keywords: Coptis chinensis Franch., berberine, alkaloid, signaling pathways

1. Introduction

Coptis chinensis Franch., as a classic traditional Chinese medicine, possesses the functions of clearing heat, drying dampness, purging fire, and detoxifying. Its modern pharmacological value is increasingly gaining widespread attention. This article systematically reviews the research progress on the chemical constituents, multiple pharmacological activities, and related molecular signaling pathways of Coptis chinensis Franch., aiming to provide a theoretical basis for its further development and clinical application. Studies on chemical constituents have shown that Coptis chinensis Franch. is rich in isoquinoline alkaloids [1], with berberine being the most abundant, along with other active components such as palmatine and coptisine, which collectively form the material basis for its efficacy [2]. Furthermore, components such as flavonoids, lignans, and organic acids also play synergistic roles [3]. In terms of pharmacological activities, Coptis chinensis Franch. exhibits broad-spectrum and significant biological activities. Research has confirmed that it possesses potent antibacterial [4-6] and antiviral [7-9]effects, can effectively improve insulin resistance and lower blood glucose [10-12], and also demonstrates anti-tumor effects by inhibiting cell proliferation [13] and inducing apoptosis [14], among other mechanisms [15,16]. Research on the mechanisms of action further reveals that berberine, the core active component of Coptis chinensis Franch., primarily functions by modulating several key signaling pathways. These include the AMPK/PI3K-Akt pathway involved in regulating energy metabolism [11,17], the AMPK/mTOR

DOI: 10.54254/2753-8818/2026.PJ30389

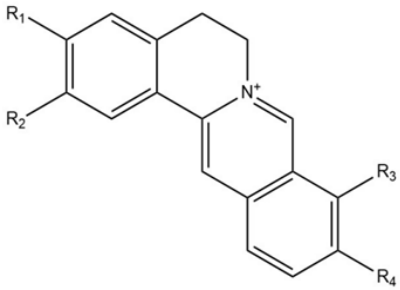
pathway regulating autophagy and cell growth [18-20], and the TLR4/MyD88/NF-κB pathway that inhibits inflammatory responses [21,22].

2. Chemical composition

Coptis chinensis Franch. contains various compounds, including alkaloids, flavonoids, lignans, and organic acids, among which alkaloids are the primary active constituents [1].

2.1. Alkaloids

Alkaloids are the primary active constituents in Coptis chinensis Franch., mainly comprising isoquinoline alkaloids, with berberine being the most abundant [23]. Li Feng conducted a study on the extraction and isolation of the main chemical components from the rhizomes of ten varieties of Coptis chinensis Franch. (Weilian) from Emeishan City.The six major protoberberine-type alkaloids in Coptis chinensis Franch. were quantified in the following descending order: berberine (1) > coptisine (2) ≥ palmatine (3) > epiberberine (4) > columbamine (5) ≥ jatrorrhizine (6). Their approximate ratios were 5.4~8.3 : 1.4~2.5: 1.3~2.4: 1: 0.4~0.8: 0.4~0.7 [24]. The 2015 edition of the Chinese Pharmacopoeia specifies coptisine, berberine, epiberberine, and palmatine as indicative components for Coptis chinensis Franch. [2]. The berberine content varies across different parts of the plant: rhizomes contain 5% ~ 8%, fibrous roots contain up to 5%, and leaves contain 1.4% ~ 2.9% [25]. Differences in berberine content are also observed among different varieties of Coptis chinensis Franch.: Coptis chinensis Franch. (Weilian) contains 5.56% ~ 7.25%, Coptis deltoidea C. Y. Cheng et Hsiao (Yalian) contains 5.20% ~ 5.32%, and Coptis teeta Wall. (Yunlian) contains 6.83% ~ 7.69% [26]. The structures of compounds 1~6 are shown in Figure 1.



1. R_1+R_2=-OCH_2O-, R_3=-OCH_3, R_4=-OCH_3
2. R_1+R_2=-OCH_2O-, R_3+R_4=-OCH_2O-
3. R_1=-OCH_3, R_2=-OCH_3, R_3=-OCH_3, R_4=-OCH_3
4. R_1=-OCH_3, R_2=-OCH_3, R_3+R_4=-OCH_2O-
5. R_1=-OCH_3, R_2=-OH, R_3=-OCH_3, R_4=-OCH_3
6. R_1=-OH, R_2=-OCH_3, R_3=-OCH_3, R_4=-OCH_3

Figure 1. Structures of the six protoberberine-type alkaloids in Coptis chinensis Franch.

2.2. Flavonoids

Flavonoids are a class of compounds in nature characterized by the 2-phenylchromone structure. They typically exist in plants as glycosides conjugated with sugars, while a small portion occur in the form of aglycones. Coptis chinensis Franch. contains a wide variety of flavonoid compounds, such as rhamnetin, wogonin, 3,5,7-trihydroxy-6,8-dimethoxyflavone, coptis saponin I, 2',4,4'-trihydroxy-6'-methoxydihydrochalcone, evodioside XII, 7,4'-dihydroxy-5-methoxyflavanone, 6,8-dimethyl-3,5,7-trihydroxyphenone-coptis saponin II, among others [2].

2.3. Lignans

Lignans are natural compounds formed by the polymerization of two phenylpropanoid derivatives. Chen Liang et al. [27] isolated 13 compounds from a 90% ethanol extract of Coptis chinensis

DOI: 10.54254/2753-8818/2026.PJ30389

Franch., seven of which were lignans. These include erythro-guaiacylglycerol-8-O-4'-(coniferyl alcohol) ether (7), threo-guaiacylglycerol-8-O-4'-(coniferyl alcohol) ether (8), (±)-pinoresinol (9), (±)-medioresinol (10), (±)-lariciresinol (11), (±)-5'-methoxylariciresinol (12), and (±)-isolariciresinol (13). Among these, compounds 7, 8, 10, and 12 were isolated from the Coptis genus for the first time.

2.4. Organic acids

The acidic compounds in Coptis chinensis Franch. are mostly small molecules containing acidic groups such as hydroxyl and carboxyl groups, and they often form glycosides with glucose. Coptis chinensis Franch. contains a variety of organic acids, such as scopolin, protocatechuic acid, ferulic acid, chlorogenic acid, catechol, lactic acid, vanillic acid, and gentisic acid, among others [3]. Meng Fancheng et al. [28] isolated 12 compounds from the ethanol extract of dried rhizomes of Coptis teeta Wall. (Yunnan Huanglian), six of which were organic acids. These include ferulic acid (14), Z-octadecyl caffeate (15), protocatechuic acid (16), methyl-3,4-dihydroxyphenyl lactate (17), 3,4-dihydroxyphenethyl alcohol (18), and 3,5-dihydroxyphenethyl alcohol-3-O-β-D-glucopyranoside (19). All these compounds were isolated from Coptis teeta Wall. for the first time, with compounds 16 and 19 being the first reported from the Coptis genus. Li Xuegai et al. [29] isolated nine organic acid compounds from the aqueous extract of Coptis chinensis Franch., including 5-O-feruloyl D-quinic acid (20), 4-O-feruloyl D-quinic acid (21), 3-methoxy-4-hydroxybenzoic acid (22), ferulic acid (23), vanillic acid-4-O-β-D-glucopyranoside (24), 3-(3',4'-dihydroxyphenyl)-(2R)-lactic acid-4'-O-β-D-glucopyranoside (25), 3-(4'-hydroxyphenyl)-(2R)-lactic acid (26), 3-(3',4'-dihydroxyphenyl)-(2R)-lactic acid (27), and 3-(3',4'-dihydroxyphenyl)-(2R)-lactic acid methyl ester (28). The structures of compounds 14–19 are shown in Figure 2.

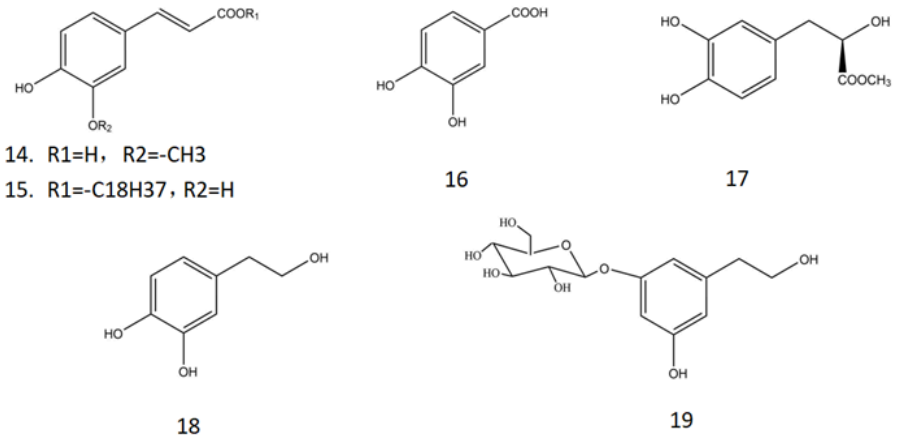


Figure 2. Structures of the six organic acids in Coptis chinensis Franch.

3. Pharmacological activities

3.1. Antibacterial effects

Berberine and other active components in Coptis chinensis Franch. exhibit broad-spectrum antimicrobial activity, demonstrating significant inhibitory effects against Gram-positive bacteria, Gram-negative bacteria, and fungi [3]. Huang Yanfei et al. [4] determined that the minimum inhibitory concentration (MIC) of the aqueous extract of Coptis chinensis Franch. against Shigella dysenteriae was 5 mg/mL, and found that the antibacterial effect strengthened with increasing

DOI: 10.54254/2753-8818/2026.PJ30389

concentration and prolonged exposure time. Li Qiaoru et al. [5] used in vitro screening to evaluate the antimicrobial activity of aqueous and ethanol extracts of Coptis chinensis Franch. , Daqingcao, Donglingcao and 17 other Chinese herbal medicines against Staphylococcus aureus, Neisseria gonorrhoeae, and Candida albicans. The study found that the aqueous and ethanol extract of Coptis chinensis Franch. show strong antibacterial activity against Staphylococcus aureus and significant inhibitory effect on Candida albicans, while their antibacterial activity against Neisseria gonorrhoeae is weak, with a MIC greater than 1:32.Yang Xingtang et al. [6] conducted a susceptibility test of berberine against Helicobacter pylori and found a correlation between the diameter of the inhibition zone and the concentration of berberine within a certain range. In the 250 μg berberine group, the inhibition zone diameter reached 19.78 mm, with an effectiveness rate of 88.5%, comparable to that of clarithromycin and amoxicillin. The 100 μg berberine group showed a similar effectiveness rate to antibiotics, but the inhibition zone diameter was significantly smaller. The 25 μg and 50 μg berberine groups exhibited significantly lower efficacy compared to the antibiotic group.

3.2. Antiviral effects

Coptis chinensis Franch. exhibits inhibitory effects against multiple viruses, such as herpes simplex virus, influenza virus, and cytomegalovirus. Zhang Yan et al. [7] discovered through in vitro experiments that berberine hydrochloride significantly inhibited the cytopathic effect (CPE) induced by HSV-1 in a dose-dependent manner. In the berberine hydrochloride treatment group, the expression level of gD mRNA was significantly reduced 16 hours after viral infection. Berberine hydrochloride also markedly suppressed the expression of viral proteins ICP27 and ICP8, with a stronger and dose-dependent inhibition observed for ICP27 expression. Chen Xiaoming et al. [8] found in their study that berberine inhibits influenza virus-induced endothelial cell cytoskeletal remodeling and reduces endothelial cell permeability by stabilizing the cytoskeleton and decreasing stress fiber formation. Yang Liumeng et al. [9] systematically compared the anti-HIV-1 activity and cytotoxicity of berberine, palmatine, and their 13-hexyl derivatives (13-hexylberberine and 13-hexylpalmatine). The results showed that berberine and palmatine strongly inhibited HIV-1 reverse transcriptase (RT), with EC_{50} values of 63.1μg/mL and 44.52μg/mL, respectively. In contrast, the hexyl derivatives completely lost their RT inhibitory activity. However, 13-hexylberberine and 13-hexylpalmatine demonstrated significantly stronger anti-HIV-1 activity, with EC_{50} values of 0.16 μg/mL and 0.08 μg/mL, respectively.

3.3. Hypoglycemic effects

Coptis chinensis Franch. can improve insulin resistance and reduce blood glucose levels through multiple pathways. Berberine primarily exerts its hypoglycemic effects by stimulating glycolysis independently of the AMPK signaling pathway, enhancing the expression of insulin receptor substrate-1 and PI3K proteins, and modulating the LKB1/AMPK/TORC2 signaling pathway, among other mechanisms [23]. Jiang Miao [10] established a type 2 diabetes insulin resistance (IR) rat model and found that the herbal pair of Coptis chinensis Franch. and ginseng effectively suppressed weight gain in these rats, significantly reduced fasting blood glucose (FBG) and fasting insulin (FINS) levels, and demonstrated IR-improving effects comparable to pioglitazone. This treatment effectively alleviated hyperglycemia and hyperinsulinemia in type 2 diabetic IR rats and improved the insulin sensitivity index. Huang Xiufang et al. [30] developed an IR rat model using high-fat, high-sugar, and high-salt feed to investigate the effects of Huanglian Jiedu Decoction (HLJD) on inflammatory factors (IL-6, TNF-α) and oxidative stress markers (SOD, MDA). Compared to the

DOI: 10.54254/2753-8818/2026.PJ30389

model group, all HLJD groups showed significant reductions in fasting plasma glucose (FPG) and FINS, along with a notable increase in the insulin sensitivity index (ISI). The high- and medium-dose HLJD groups exhibited superior effects to the metformin group. Additionally, all HLJD dose groups demonstrated significantly decreased levels of IL-6, TNF-α, and MDA, and a marked increase in SOD activity. Chen Guang et al. [31] conducted in vivo experiments using a type 2 diabetes mellitus (T2DM) rat model and discovered that berberine significantly inhibited weight gain in the model rats, markedly lowered fasting blood glucose levels, promoted the increased expression of PI3K p85 and GLUT4 proteins, and alleviated insulin resistance in target tissues. Qin Xiaojun et al. [12] established a diabetic rat model with cerebral ischemia-reperfusion injury to explore the impact of Coptis chinensis Franch. on the expression of apoptosis-related proteins Bax and Bcl-2. The results indicated that Coptis chinensis Franch. downregulated the pro-apoptotic protein Bax and upregulated the anti-apoptotic protein Bcl-2. Furthermore, a high dose of Coptis chinensis Franch. significantly improved neurological deficits and reduced blood glucose levels.

3.4. Antitumor effects

Active components in Coptis chinensis Franch., such as berberine, have been found to exert antitumor effects by inhibiting tumor cell proliferation, migration, and adhesion, as well as promoting apoptosis [13]. The mechanisms by which berberine inhibits tumor cell proliferation primarily include: arresting the cell cycle, inhibiting the activity of related proteins and enzymes, modulating signaling pathways, inducing changes in mitochondrial membrane potential, reducing IL-6 levels, and blocking potassium ion channels. Huang Linqing et al. [15] demonstrated through in vivo and in vitro antitumor experiments that berberine significantly inhibits S-180 cells both in vitro and in vivo. Bao Jianzhong et al. [14] found that berberine can inhibit the proliferation and differentiation of human colon cancer cells, with the inhibitory effect positively correlating with drug concentration and exposure time. Berberine promotes apoptosis through mechanisms related to the Bcl-2 protein family and the Caspase cascade reaction. Furthermore, multiple studies have indicated that berberine from Coptis chinensis Franch. exhibits inhibitory and cytotoxic effects on various tumor cells, including gastric cancer, osteosarcoma, prostate cancer, bladder cancer, mouse S-180 tumor cells, HL-60, MGC-803, HepG2, NT2/D1, and YES-2 cells, among others [16].

4. Related signaling pathways

The pharmacological effects of Coptis chinensis Franch. and its active components are closely associated with the modulation of multiple key signaling pathways. These pathways are extensively involved in core biological processes such as energy metabolism, autophagy, and inflammatory responses. The following sections will review critical pathways including AMPK/PI3K-Akt, AMPK/mTOR, and TLR4/MyD88, elucidating the multi-target mechanisms of Coptis chinensis Franch. and its role in improving insulin resistance, thereby providing a basis for further exploration of its pharmacological value, as summarized in Table 1.

DOI: 10.54254/2753-8818/2026.PJ30389

Table 1. Signaling pathways related to Coptis chinensis Franch.

Signaling Pathway	Status and Function in Type 2 Diabetes	Core Mechanisms	Key Molecules and Processes
AMPK/PI3K-Akt Glucose Metabolism Signaling Pathway [32,33]	Status: PI3K-Akt is inhibited; AMPK activity is reduced. Function: Coordinately regulates cellular glucose uptake and utilization.	Inflammatory factors and lipid accumulation activate kinases that inhibit IRS-1, thereby blocking insulin signaling.	AMPK Pathway: Increased AMP/ATP ratio → AMPK → TBC1D1/AS160 → GLUT4 translocation PI3K-Akt Pathway: Insulin receptor → IRS → PI3K → Akt → Promotes GLUT4 translocation and glycogen synthesis
AMPK/mTOR Autophagy Regulatory Pathway[34, 17,20]	Status: mTORC1 activity is overactivated; autophagy level is suppressed. Function: AMPK and mTOR act antagonistically to coordinately regulate cell growth and catabolism.	Nutrient excess leads to overactivation of mTORC1, which suppresses autophagy, resulting in the accumulation of damaged mitochondria and misfolded proteins, thereby promoting insulin resistance. AMPK directly inhibits mTORC1 while simultaneously activating autophagy, thereby improving cellular function.	Energy insufficiency → AMPK → inhibits mTORC1 + activates ULK1 → initiates autophagy Sufficient nutrition → Akt → mTORC1 → promotes anabolic processes and suppresses autophagy
TLR4/MyD88 Inflammatory Signaling Pathway [21,35]	Status: The pathway is chronically activated. Function: Serves as a core pathway in innate immunity and links nutrient excess to insulin resistance.	Free fatty acids, endotoxins, and other ligands activate the TLR4 receptor, leading to the production of inflammatory factors, disruption of insulin signaling, and ultimately triggering the development of type 2 diabetes.	Ligand → TLR4 → MyD88 → IKK → NF-κB nuclear translocation → inflammatory gene transcription
Gut Hormones/ Gut-Pancreas Axis Signaling Pathway [36]	Status: Insufficient secretion or accelerated degradation of GLP-1. Function: Postprandial intestinal hormone secretion promotes insulin release and suppresses glucagon secretion in a glucose concentration-dependent manner.	GLP-1 activates GLP-1R on β cells, promotes insulin gene transcription and secretion via the cAMP-PKA pathway, while also inhibiting beta-cell apoptosis, delaying gastric emptying, and suppressing appetite.	Food intake → L cells in the intestine secrete GLP-1 → binds to GLP-1R on beta cells → Gs protein → adenylate cyclase → increased cAMP → PKA → promotes insulin secretion and cell proliferation
Endoplasmic Reticulum Stress Unfolded Protein Response (UPR) [37,38]	Status: Chronic, unresolved endoplasmic reticulum stress. Function: The UPR is an adaptive cellular response to alleviate the accumulation of misfolded proteins in the endoplasmic reticulum.	Under endoplasmic reticulum stress from unfolded/misfolded protein accumulation, UPR is activated. IRE1, PERK, ATF6 alleviate stress by reducing protein synthesis and enhancing folding capacity.	IRE1α: Splices XBP1 or activates JNK. PERK: Phosphorylates eIF2α while activating ATF4/CHOP. ATF6: Transcriptionally regulates molecular chaperones.

DOI: 10.54254/2753-8818/2026.PJ30389

4.1. AMPK/PI3K-Akt glucose metabolism signaling pathway

AMPK is a heterotrimeric protein composed of a catalytic α subunit and regulatory β and γ subunits. Its activity is tightly regulated by the intracellular AMP/ATP ratio. Under cellular energy deficiency, elevated AMP stimulates interactions with regulatory proteins, promoting AMPK phosphorylation at Thr172 by tumor suppressor LKB1 to activate it [40]. Activated AMPK then phosphorylates downstream targets to regulate metabolism and restore cellular energy homeostasis Additionally, the AMPK pathway can be activated through cascade reactions involving calcium/calmodulin-dependent protein kinase β (CaMKKβ) and liver kinase B1 (LKB1), among others [39].

When cellular energy supply is insufficient, AMP stimulates interactions with axial proteins, leading to the phosphorylation of AMPK at the Thr172 site by the tumor suppressor liver kinase B1 (LKB1), thereby activating it [40].

When insulin molecules bind to the extracellular domain of the α subunit of the insulin receptor (InsR), the insulin binding induces a conformational change in the α subunit. This brings the intracellular domains of the two β subunits closer together, stabilizing the receptor's dimeric state. The proximity of the β subunit intracellular domains allows the kinase domain of one β subunit to phosphorylate key tyrosine residues on the intracellular domain of the other β subunit. IRS proteins are recruited and anchored near the activated InsR, and tyrosine residues (Tyr) on the IRS proteins are phosphorylated by the InsR kinase domain. The phosphorylated IRS proteins act as a platform to recruit and activate two major signaling pathways: the PI3K-Akt pathway and the Ras-MAPK pathway. The PI3K-Akt pathway is the core pathway for insulin signal transduction. PI3K is recruited to the receptor, and the p85 regulatory subunit of PI3K specifically binds to certain phosphorylated tyrosine residues on the IRS protein, leading to PI3K activation. The activated catalytic subunit of PI3K then converts phosphatidylinositol 4,5-bisphosphate (PIP_2) into phosphatidylinositol (3,4,5)-trisphosphate (PIP_3) [41]. PIP_3 acts as a second messenger, binding to the PH domain of Akt and recruiting Akt to the inner surface of the plasma membrane. The kinases PDK1 and mTORC2 phosphorylate Akt at Thr308 and Ser473, respectively. The activated Akt then dissociates from the plasma membrane and enters the cytoplasm and nucleus, where it phosphorylates numerous downstream substrates.

Under energy stress conditions, activated AMPK negatively regulates the insulin/PI3K-Akt signaling pathway by phosphorylating specific sites on IRS or activating phosphatases to dephosphorylate IRS, thereby reducing unnecessary anabolic processes. Under energy-sufficient conditions, Akt phosphorylates AMPKα at Ser485/491, which inhibits LKB1-mediated phosphorylation at Thr172, consequently suppressing catabolic activity. As shown in Figure 3.

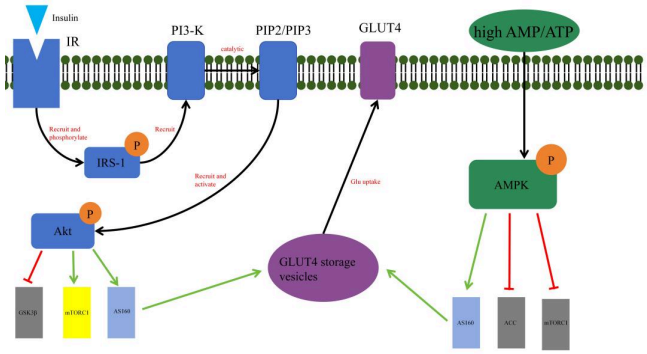


Figure 3. Schematic diagram of the AMPK/PI3k-Akt glucose metabolism signaling pathway

DOI: 10.54254/2753-8818/2026.PJ30389

4.2. AMPK/mTOR autophagy regulatory pathway

The mammalian target of rapamycin (mTOR) is a serine/threonine protein kinase composed of two structurally and functionally distinct complexes, mTORC1 and mTORC2. These complexes regulate the expression and activity of transcription factors such as HIF-1α and the myelocytomatosis oncogene (MYC) to promote glycolysis, glutaminolysis, and the pentose phosphate pathway [42].

Under energy stress conditions, AMPK is activated and strongly inhibits mTORC1 activity, thereby blocking metabolic pathways such as glucose utilization and protein synthesis, which restricts the proliferation of tumor cells. Conversely, when this pathway is impaired and mTOR remains persistently activated, it not only weakens the positive regulation of tumor suppressors like p16 but also relieves the inhibition of various pro-oncogenic factors such as HIF-1α and β-catenin. This ultimately exacerbates metabolic abnormalities, inflammatory responses, and tumor microenvironment dysregulation, creating favorable conditions for the development of malignancies such as liver cancer [18]. Liu Yang et al. [19] demonstrated through in vitro experiments that, compared to the control group, the expression levels of AMPK and mTOR genes were significantly increased in the berberine-treated group. Additionally, berberine inhibited the proliferation and metabolism of EC9706 cells, indicating that berberine exerts its antitumor effects by modulating the AMPK/mTOR pathway.

4.3. TLR4/MyD88 inflammatory signaling pathway

Toll-like receptors (TLRs) are a class of type I transmembrane protein receptors comprising thirteen members (TLR1-TLR13) [43]. Among them, TLR1, 2, 4, 5, 6, and 10 are involved in recognizing cell surface molecules and are expressed on the cell membrane, whereas TLR3, 7, 8, and 9 recognize nucleic acids and are localized intracellularly within endosomes [44]. TLR4, a key member of the TLR family, specifically recognizes lipopolysaccharide (LPS) from pathogens and stimulates immune-inflammatory responses by activating adaptive immune-related genes.

MyD88 is an adapter protein containing a TIR domain and serves as a downstream signaling factor in the TLR signaling pathway [44]. The MyD88-dependent signaling pathway is commonly present in most TLR family members, though signal transduction in some TLRs does not entirely rely on MyD88. Thus, TLR signaling can be categorized into MyD88-dependent and MyD88-independent pathways. In the MyD88-dependent pathway, signaling is specifically activated by TLR4. The activated TIR domain associates with MyD88, leading to the recruitment of IRAK4 to the TLR-MyD88 complex. Once activated, IRAK4 phosphorylates and recruits IRAK1 and IRAK2, thereby initiating downstream signaling cascades [45]. Ji Lingyun et al. [21] pretreated cells with serum containing Scutellaria-Coptis drug pair at different concentrations, followed by induction of pyroptosis with LPS + ATP. Their findings revealed that the Scutellaria-Coptis drug pair inhibits macrophage pyroptosis by suppressing the TLR4/MyD88/NF-κB signaling pathway and downregulating NLRP3 inflammasome activation. Similarly, Zhang Hongjie et al. [22] established a neuroinflammation model using LPS and demonstrated that the Scutellaria-Coptis drug pair reduces the release of pro-inflammatory cytokines by inhibiting the activation of the TLR4/MyD88/NF-κB signaling pathway in microglia.

5. Conclusion

This article systematically reviews the research progress on the chemical constituents, pharmacological activities, and related molecular signaling pathways of Coptis chinensis Franch.

DOI: 10.54254/2753-8818/2026.PJ30389

Studies have shown that as a classic traditional Chinese medicine, the modern pharmacological value of Coptis chinensis Franch. is primarily reflected in the multi-component, multi-target synergistic mechanisms centered on isoquinoline alkaloids. In terms of chemical composition, Coptis chinensis Franch. contains not only various alkaloids such as berberine, palmatine, and coptisine but also active components including flavonoids, lignans, and organic acids, which collectively form the material basis for its efficacy. In pharmacological activities, Coptis chinensis Franch. demonstrates broad therapeutic potential. Its antibacterial effects are mainly achieved by disrupting bacterial cell membrane permeability and inhibiting key enzyme activities. Its antiviral activity is reflected in inhibiting viral replication and protein expression. The hypoglycemic effect is closely related to improving insulin resistance and regulating glucose metabolism, while its antitumor activity operates through multiple mechanisms such as inducing apoptosis and inhibiting proliferation. Furthermore, multiple studies have found that berberine, the core active component of Coptis chinensis Franch., exerts its effects by regulating several keys signaling pathways. Among them, the AMPK/PI3K-Akt pathway plays a central role in regulating energy metabolism and insulin sensitivity; the AMPK/mTOR pathway is involved in the regulation of autophagy and cell growth; and the TLR4/MyD88 pathway plays an important role in suppressing inflammatory responses. The crosstalk between these pathways further illustrates the complexity of the mechanisms of action of Coptis chinensis Franch.

However, several issues in current research remain to be addressed. Most mechanistic studies are still at the preclinical stage, lacking high-quality clinical research data. The understanding of the synergistic mechanisms among various active components is insufficient, and the bioavailability of berberine needs further improvement. Future research should focus on conducting well-designed clinical trials, delving deeper into the synergistic mechanisms of multiple components, and developing novel drug delivery systems to enhance bioavailability, thereby providing important insights and resources for innovative drug development.

References

[1] Wang W, Zhang QW, Ye WC, et al. Isoquinoline Alkaloids from the Rhizoma of Coptis chinensis Franch. [J]. Chinese Journal of Natural Medicines, 2007, 2007(05): 348.
[2] National Pharmacopoeia Commission. Pharmacopoeia of the People's Republic of China (Volume I) [S]. Beijing: China Medical Science and Technology Press, 2015: 303-305.
[3] Fu L, Fu Q, Li J, et al. Research Progress on Chemical Constituents and Pharmacological Effects of Coptis chinensis Franch. [J]. Acta Chinese Medicine and Pharmacology, 2021, 49(2): 88.
[4] Huang YF. Study on Bacteriostatic Mechanism of Aqueous Extract of Coptis chinensis Franch. against Shigella [D]. Hefei: Anhui Agricultural University, 2016.
[5] Li QR, Ren JK, Liu ZZ, et al. Screening of antibacterial activity of 18 Chinese herbal medicines [J]. Shaanxi Journal of Traditional Chinese Medicine, 2002, 23(6): 555.
[6] Yang XT, Wang ZR, Wang ZJ, et al. Experimental study on antibacterial effect of berberine against Helicobacter pylori [J]. Journal of Tongji University (Medical Science), 2014, 35(6): 63-66+112.
[7] Zhang Y, Chen T, Huang HY, et al. In vitro anti-herpes simplex virus type 1 activity and mechanism of berberine hydrochloride [J]. Pharmacology and Clinics of Chinese Materia Medica, 2017, 33(1): 33-37.
[8] Chen XM, Zhang SJ, Qi XY, et al. Effect of berberine on cytoskeleton and permeability of HUVEC infected by influenza virus [J]. Journal of Liaoning University of Traditional Chinese Medicine, 2017, 19(12): 28-31.
[9] Yang LM, Wang RR, Li JJ, et al. In vitro anti-HIV-1 activity of four berberine analogues [J]. Chinese Journal of Natural Medicines, 2007, 5(3): 225-228.
[10] Jiang M. Study on the mechanism of Coptis chinensis and Panax ginseng herb pair in improving insulin resistance of type 2 diabetes mellitus [D]. Beijing: Beijing University of Chinese Medicine, 2006.
[11] Chen G, Lu FE, Wang ZS, et al. Correlation between berberine-improved insulin resistance and PI-3K, GLUT4 proteins in type 2 diabetic rats [J]. Chinese Pharmacological Bulletin, 2008, 24(8): 1007-1010.

DOI: 10.54254/2753-8818/2026.PJ30389

[12] Qin XJ, Feng AP, Huang QJ, et al. Effect of Coptidis Rhizoma on the expression of Bax and Bcl-2 proteins in diabetic rats with cerebral ischemia-reperfusion model [J]. Practical Geriatrics, 2017, 31(11): 2021-2024.
[13] Gai XH, Liu SX, Ren T, et al. Research progress on chemical constituents of Coptidis Rhizoma and its pharmacological activities [J]. Chinese Traditional and Herbal Drugs, 2018, 49(20): 4924.
[14] Bao JZ, Zhang ZY, Ye HB, et al. Mechanism of berberine on proliferation and apoptosis of human colon cancer cells [J]. Chinese Journal of Control of Endemic Diseases, 2017, 37(2): 191-192.
[15] Huang LQ, Xu CF, Zhou SW, et al. Experimental study on the antitumor effect of berberine [J]. Chinese Pharmacological Bulletin, 1997, 13(2): 1.
[16] Siqin Gaowa, Baolachaolu, Li M, et al. Research progress on the pharmacological activities of berberine in Coptis chinensis Franch. [J]. Journal of Medicine & Pharmacy of Chinese Minorities, 2014, 20(1): 50.
[17] Zhang M, Liang B, Yang ZM. Oxidized low-density lipoprotein induces autophagy in HUVECs via the AMPK/mTOR signaling pathway [J]. Chinese Journal of Biochemistry and Molecular Biology, 2019, 35(6): 662-668.
[18] Hu Y, Hu SQ, Zeng S, et al. Research progress on Chinese medicine monomers against hepatocellular carcinoma based on the AMPK/mTOR signaling pathway [J/OL]. Chinese Journal of Experimental Traditional Medical Formulae, 2025, (): 1-14. https: //doi.org/10.13422/j.cnki.syfjx.20251528. [Accessed 4 November 2025].
[19] Liu Y, Chen X, Zhou ZX, et al. Effect of berberine on energy metabolism of EC9706 cells via the AMPK/mTOR pathway [J]. Chinese Archives of Traditional Chinese Medicine, 2025, 43(01): 97-101+270-271. https: //doi.org/10.13193/j.issn.1673-7717.2025.01.018.
[20] Li CY, Li SR, Zhang W, et al. Kidney-tonifying Chinese medicines enhance autophagic capacity of Leydig cells in rats with kidney essence deficiency via the AMPK-mTOR pathway [J]. Journal of Hebei University (Natural Science Edition), 2024, 44(6): 633-642.
[21] Ji LY, Chen WD, Ge CL, et al. Mechanism of Scutellaria baicalensis and Coptis chinensis herb pair in treating atherosclerosis based on macrophage pyroptosis mediated by the TLR4/MyD88/NF-κB signaling pathway [J]. China Journal of Traditional Chinese Medicine and Pharmacy, 2024, 39(5): 2266-2272.
[22] Zhang HJ, Su D, Zhu GH, et al. Compatibility advantage of Scutellariae Radix-Coptidis Rhizoma in anti-neuroinflammation and its potential targets in regulating the TLR4/MyD88/NF-κB signaling pathway [J]. Chinese Journal of Experimental Traditional Medical Formulae, 2022, 28(22): 58-67.
[23] Chen ZX, Zhai XM. Research progress on chemical constituents and pharmacological effects of Coptis chinensis Franch. [J]. Harbin Medical Journal, 2025, 45(1): 131.
[24] Li F. Study on chemical constituents and quality standards of Coptis chinensis Franch. [D]. Chengdu: Sichuan University, 2007.
[25] Du QB. Overview of studies on chemical constituents and pharmacological activities of Coptis chinensis Franch. [J]. Journal of Baotou Medical College, 2015, 31(5): 153.
[26] Xu JT, Wang LQ, Xu B. Research progress on Coptis chinensis Franch. [J]. Acta Academiae Medicinae Sinicae, 2004, 26(6): 705..
[27] Chen L, Wang L, Zhang QW, et al. Study on non-alkaloid chemical constituents from Coptis chinensis Franch. [J]. China Journal of Chinese Materia Medica, 2012, 37(9): 1241-1244.
[28] Meng FC, Wang L, Zhang J, et al. Study on non-alkaloidal constituents from Coptis teeta [J]. Journal of China Pharmaceutical University, 2013, 44(4): 307-310.
[29] Li XG, Yang LG, Chen LX, et al. Isolation and identification of chemical constituents from the aqueous extract of Coptis chinensis Franch. [J]. Journal of Shenyang Pharmaceutical University, 2013, 29(3): 195-198.
[30] Huang XF, Tao YG, Zhang RL, et al. Effects of Huanglian Jiedu Decoction on inflammatory factors and oxidative stress levels in insulin-resistant rats [J]. Chinese Journal of Traditional Medical Science and Technology, 2017, 24(5): 572-574+583.
[31] Chen G, Lu FE, Wang ZS, et al. Correlation between the amelioration of insulin resistance and protein expression of PI-3K and GLUT4 in type 2 diabetic rats treated with berberine [J]. Chinese Pharmacological Bulletin, 2008, 24(8): 1007-1010.
[32] Zhang Y, Cao Y, Wang F, Wang L, et. Polysaccharide from Momordica charantia L. Alleviates Type 2 Diabetes Mellitus in Mice by Activating the IRS1/PI3K/Akt and AMPK Signaling Pathways and Regulating the Gut Microbiota [J]. Agric Food Chem, 2025 Mar 26; 73(12): 7298-7309.
[33] Tao R, Gong J, Luo X, et. AMPK exerts dual regulatory effects on the PI3K pathway. J Mol Signal. 2010 Feb 18; 5(1): 1.
[34] Kim J, Kundu M, Viollet B, et. AMPK and mTOR regulate autophagy through direct phosphorylation of Ulk1. Nat Cell Biol. 2011 Feb; 13(2): 132-41.

DOI: 10.54254/2753-8818/2026.PJ30389

[35] Sun FJ, Dong WB. Research progress on the correlation between the TLR4/MyD88 signaling pathway and kidney diseases [J]. Journal of Southwest Medical University, 2010, 33(1): 100-102.
[36] Müller T D, Finan B, Bloom S R, et. Glucagon-like peptide 1 (GLP-1) [J]. Physiological Reviews, 2019, 99(3): 1339-1381.
[37] Xu F, Wang L. Deciphering ER stress-unfolded protein response relationship by visualizing unfolded proteins in the ER. Cell Rep. 2024 Jun 25; 43(6): 114358.
[38] Sak F, Sengul F, Vatansev H. The Role of Endoplasmic Reticulum Stress in Metabolic Diseases. Metab Syndr Relat Disord. 2024 Sep; 22(7): 487-493.
[39] Lian K, Meng LC, Wang XQ, et al. Exploration of traditional Chinese medicine treatment for chronic heart failure based on the AMPK signaling pathway [J/OL]. Chinese Journal of Experimental Traditional Medical Formulae, 2025. https: //doi.org/10.13422/j.cnki.syfjx.20250915. [Accessed 4 November 2025].
[40] Zhou C, Wang LY, Zhou ZH, et al. Role of the AMPK signaling pathway in cerebral ischemia-reperfusion injury and research progress of related drugs [J]. Chinese Journal of Modern Applied Pharmacy, 2025, 42(14): 2510-2517.
[41] Cai RX, Tang JY, Zhu H, et al. Research progress on Chinese medicine targeting the PI3K/Akt signaling pathway against hypertrophic cardiomyopathy [J]. Journal of Qiqihar Medical University, 2025, 46(15): 1464.
[42] Gu BB, Meng Y. Research progress on the regulation of the PI3K/Akt/mTOR signaling pathway by traditional Chinese medicine in anti-lung cancer effects [J/OL]. Journal of Liaoning University of Traditional Chinese Medicine. https: //link.cnki.net/urlid/21.1543.R.20250722.1420.010. [Accessed 4 November 2025].
[43] Zhu SZ. Mechanism of traditional Chinese medicine mediating the TLR4/MyD88/NF-κB signaling pathway in the treatment of diabetic nephropathy [J/OL]. Acta Chinese Medicine. https: //link.cnki.net/urlid/41.1411.r.20250326.1607.004. [Accessed 4 November 2025].
[44] Wang RJ, Wang G, Zhang L. Role of the TLR4/MyD88/NF-κB signaling pathway in rheumatoid arthritis and research progress in Chinese medicinal intervention [J]. Information on Traditional Chinese Medicine, 2023, 40(8): 85.
[45] Bao LL, Cui LH. Advances in the TLR4/MyD88/NF-κB signaling pathway [J]. Chinese Journal of Gastroenterology and Hepatology, 2019, 28(5): 568-569.