Pharmacological Effects of Lupeol: Antitumor, Anti-inflammatory, Antioxidant, and Antidiabetic Activities

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Abstract Lupeol (LUP) is a natural triterpenoid compound derived from various plants, exhibiting pharmacological effects including tumor inhibition, inflammation suppression, antioxidant activity, and amelioration of type 2 diabetes mellitus. In terms of anti-tumor activity, LUP inhibits breast cancer by inducing ROS accumulation, suppresses liver cancer by inhibiting the Wnt/β-catenin pathway, exerts anti-lung cancer effects by regulating the Notch pathway, and blocks the colon cancer cell cycle by inhibiting the RhoA-ROCK1 pathway. In terms of anti-inflammatory activity, LUP alleviates osteoarthritis by activating the SIRT3/mTOR pathway to enhance autophagy. In terms of antioxidant activity, LUP alleviates oxidative stress and inflammation to protect against myocardial injury. Additionally, LUP improves type 2 diabetes mellitus by regulating glucose and lipid metabolism and protecting islet β-cells. LUP has high safety and multi-target regulatory effects. However, its specific molecular mechanisms and clinical studies still require further in-depth exploration.

Key words Lupeol, Anti-tumor, Anti-inflammatory, Antioxidant, Type 2 diabetes mellitus

0 Introduction

Lupeol (LUP) is a triterpenoid compound isolated and purified from various natural plants, including the seed coat of *Mesembryanthemum crystallinum*, dandelion, and white birch bark. It has a molecular formula of $C_{30}H_{50}O$ and appears as a white crystalline powder. Lupeol is soluble in organic solvents such as methanol, ethanol, and DMSO. Numerous studies have shown that LUP possesses various pharmacological effects, including tumor inhibition[1], inflammation suppression[2], oxidative stress relief[3], and type 2 diabetes improvement. In recent years, with the continuous development of traditional Chinese medicine in China, the use of herbal extracts for the treatment of malignant tumors and chronic diseases has become a research hotspot. Among them, LUP has gradually attracted the attention of researchers due to its advantages such as high safety, significant efficacy, and low toxicity and side effects. This article reviews the pharmacological effects and molecular mechanisms of LUP in recent years, providing a theoretical basis for further research and clinical application of LUP.

1 Antitumor effects

Cancer refers to malignant tumors originating from epithelial tissues. Cancer cells are highly invasive and metastatic, and are prone to developing resistance to drugs such as doxorubicin and paclitaxel, thereby increasing the difficulty of clinical treatment. Chinese herbal medicine exhibits unique advantages in cancer treatment due to its low drug resistance. Studies have shown that LUP has good inhibitory effects on breast cancer, liver cancer, lung cancer, and colon cancer.

1.1 Antitumor effect on breast cancer Breast cancer is a malignant tumor originating from the epithelial tissue of the breast. Its core pathological manifestation is the uncontrolled abnormal proliferation and differentiation of epithelial cells in the mammary ducts or lobules, leading to the formation of invasive malignant lesions that tend to infiltrate surrounding tissues and metastasize to distant sites. Studies have shown that LUP has a good antitumor effect against breast cancer.

Zhu *et al.*[4] evaluated the viability of human breast cancer MCF-7 cells using the MTT assay and calculated the half-maximal inhibitory concentration (IC_{50}) of LUP. The results showed that compared with the control group, treatment with 20, 40, 80, and 160 μmol/L LUP for 24, 48, and 72 h inhibited MCF-7 cell proliferation in a concentration- and time-dependent manner. The IC_{50} values of LUP against MCF-7 cells at 24, 48, and 72 h were (93.43 ± 1.87), (63.02 ± 1.56), and (41.58 ± 1.23) μmol/L, respectively, indicating that LUP had a marked inhibitory effect on the proliferation of MCF-7 cells. Based on these findings, the regulatory effect of LUP on MCF-7 cell apoptosis was examined by flow cytometry. The results showed that compared with the control group, LUP at concentrations of 60 and 120 μmol/L effectively induced apoptosis in MCF-7 cells, indicating that LUP could promote apoptosis of MCF-7 cells. Furthermore, flow cytometry was adopted to detect the level of reactive oxygen species (ROS) in MCF-7 cells treated with LUP. The results showed that compared with the control group, the ROS level in MCF-7 cells significantly increased with the concentration of LUP increasing, indicating that LUP induced a significant increase in ROS expression level in MCF-7 cells in a concentration-dependent manner. In summary, LUP can induce apoptosis by inducing a substantial accumulation of ROS in MCF-7 cells, activating apoptotic pathways, and subsequently regulating the expression levels of apoptosis-related proteins, and thereby inhibit the proliferation and growth of MCF-7

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cells. These findings demonstrate that LUP possesses good anti-breast cancer activity, which lays an experimental foundation for its subsequent mechanistic exploration, preparation research and clinical transformation research, and makes it hopeful to become a new natural anti-breast cancer drug.

1.2 Antitumor effect on liver cancer Liver cancer is a malignant tumor originating from hepatocytes or intrahepatic bile duct epithelial cells. Its core pathological manifestation is the abnormal proliferation and uncontrolled differentiation of hepatocytes or bile duct epithelial cells, leading to the formation of highly invasive malignant tissue that tends to invade normal liver tissue and metastasize to distant sites. Studies have shown that LUP acts effectively against liver cancer.

Shi *et al.* [5] evaluated the viability of HepG2 liver cancer cells using the MTT assay. The results showed that compared with the control group, LUP inhibited the proliferation of HepG2 cells in a concentration- and time-dependent manner, indicating that LUP could effectively inhibit the proliferation of HepG2 cells. Subsequently, flow cytometry was applied to assess the effect of LUP on the apoptosis rate of HepG2 cells. The results showed that compared with the control group, LUP induced apoptosis in HepG2 cells in a concentration- and time-dependent manner, indicating that LUP could induce apoptosis in HepG2 cells. Based on these findings, real-time quantitative polymerase chain reaction (RT-qPCR) was conducted to evaluate the effects of different concentrations of LUP on the mRNA expression levels of key molecules in the Wnt/β-catenin signaling pathway, specifically the anti-apoptotic protein Wnt3 and β-catenin, in HepG2 cells after 72 h of treatment. The results showed that compared with the control group, the mRNA expression levels of Wnt3 and β-catenin in the 10, 20, and 40 μmol/L LUP treatment groups decreased in a concentration- and time-dependent manner, indicating that LUP inhibited the activation of the Wnt/β-catenin signaling pathway at the transcriptional level. Furthermore, Western blotting (WB) was used to detect the expression levels of pathway-related proteins as well as proliferation- and apoptosis-associated proteins. The results showed that after 72 h, the expression levels of Wnt3, β-catenin, proliferating cell nuclear antigen (PCNA), and B-cell lymphoma-2 (Bcl-2) decreased significantly, while the expression of Bcl-2-associated X protein (Bax) increased significantly, in a concentration-dependent manner, which indicated that LUP can regulate the expression of proliferation- and apoptosis-related proteins while inhibiting the expression of Wnt/β-catenin pathway proteins. In summary, LUP inhibits the proliferation and induces the apoptosis of HepG2 cells in a concentration- and time-dependent manner. It also inhibits the Wnt/β-catenin pathway at both the transcriptional and translational levels, thereby regulating the expression of related proteins and playing an anti-liver cancer role. These findings provide a theoretical basis for its clinical application.

1.3 Antitumor effect on lung cancer Lung cancer is a primary malignant tumor of the lung originating from the bronchial mucosa or glands. Its core pathological manifestation is the abnormal proliferation and differentiation disorder of bronchial mucosal or alveolar epithelial cells, leading to the formation of invasive malignant tissue that invades surrounding normal lung tissue and metastasizes to distant sites. Studies have shown that LUP has a good antitumor effect against lung cancer.

Yin *et al.* [6] evaluated the cell viability of lung cancer A549 cells using the MTT assay and calculated the half-maximal inhibitory concentration (IC_{50}) of LUP. The results showed that compared with the control group, LUP inhibited the proliferation of A549 cells in a concentration- and time-dependent manner, indicating that LUP significantly inhibited the proliferation of A549 cells. Furthermore, flow cytometry was used to assess the effect of LUP on the apoptosis of A549 cells. The results showed that compared with the control group, LUP induced apoptosis in A549 cells in a concentration-dependent manner, indicating that LUP significantly promoted apoptosis in A549 cells. Based on these findings, the levels of cell migration and invasion were detected using Transwell assays. The results showed that compared with the control group, LUP significantly inhibited the number of migratory and invasive cells in a concentration- and time-dependent manner, indicating that LUP effectively inhibited the migration and invasion of lung cancer A549 cells. Furthermore, Western blotting was performed to detect the expression levels of proteins related to proliferation, apoptosis, invasion, and the Notch signaling pathway in lung cancer A549 cells after treatment with 15, 30, and 60 mg/L LUP for 48 h. The results showed that compared with the control group, LUP significantly reduced the expression levels of PCNA, Bcl-2, N-cadherin, Notch-1, and Hes-1, while significantly increasing the expression level of E-cadherin. These findings indicated that LUP inhibited the expression of pro-proliferation, anti-apoptotic, pro-invasion, and Notch pathway proteins, while upregulating the expression of anti-invasion proteins. In summary, LUP inhibits the proliferation, promotes apoptosis, and suppresses the migration and invasion of lung cancer A549 cells in a concentration- and time-dependent manner. Its molecular mechanism is closely associated with the downregulation of the Notch-1/Hes-1 signaling pathway, which leads to a significant decrease in the expression levels of PCNA, Bcl-2, and N-cadherin, as well as an increase in the expression level of E-cadherin.

1.4 Antitumor effect on colon cancer Colon cancer is a malignant tumor originating from the epithelial tissue of the colonic mucosa. Its core pathological manifestation is the abnormal proliferation and uncontrolled differentiation of colonic mucosal epithelial cells, leading to the formation of invasive malignant lesions that tend to infiltrate surrounding tissues and metastasize to distant sites. Studies have shown that LUP has a good inhibitory effect against colon cancer.

Jiang *et al.* [7] used the CCK-8 assay to evaluate the effect of LUP at concentrations of 0, 20, 40, 60, 80, and 100 μmol/L on the proliferation of colon cancer HCT116 and SW620 cells. The results showed that LUP inhibited the proliferation of both HCT116 and SW620 cells in a concentration- and time-dependent manner, indicating that LUP significantly inhibits the proliferation and colo-

ny formation of these colon cancer cells. Subsequently, flow cytometry was applied to examine the effects of LUP on the cell cycle distribution and apoptosis rate of HCT116 and SW620 cells. The results showed that with increasing concentrations of LUP, the proportion of cells in the G_0/G_1 phase significantly increased, while the proportion of cells in the S phase significantly decreased, indicating that the cells were arrested in the G_0/G_1 phase. However, no significant change in the apoptosis rate was observed compared with the control group. These findings suggested that LUP inhibited the proliferation of colon cancer cells by inducing G_0/G_1 phase cell cycle arrest, without significantly affecting apoptosis. Based on these findings, RT-qPCR and Western blotting were performed to detect the mRNA and protein expression levels of Ras homolog family member A (RhoA), Rho-associated coiled coil-containing protein kinase 1 (ROCK1), β-catenin, and Cyclin D1 in HCT116 and SW620 cells after treatment with 0, 20, 40, and 60 μmol/L LUP for 48 h. The results showed that as the concentration of LUP increased, the mRNA and protein expression levels of RhoA, ROCK1, β-catenin, and Cyclin D1 all decreased significantly, indicating that LUP could significantly inhibit the RhoA-ROCK1 signaling pathway and the expression levels of its downstream molecules, thereby suppressing the proliferation of colon cancer cells. In summary, LUP inhibits the proliferation and colony formation of colon cancer HCT116 and SW620 cells in a concentration- and time-dependent manner. Its core mechanism involves inducing G_0/G_1 phase cell cycle arrest and suppressing the RhoA-ROCK1 signaling pathway, thereby reducing the expression of downstream molecules and ultimately exerting anti-proliferative effects against colon cancer HCT116 and SW620 cells.

2 Anti-inflammatory effects

Knee osteoarthritis (OA)[8] is a chronic degenerative disease highly prevalent in the elderly population. Its core pathological feature is the progressive degradation of articular cartilage, accompanied by synovial inflammatory infiltration and osteophyte formation. OA has a high incidence rate, and in advanced stages, patients experience restricted joint movement, which may even lead to disability. Studies have shown that LUP has a good improving effect on arthritis.

Ma *et al.*[9] evaluated the effects of different concentrations and treatment durations of LUP on the viability of primary mouse chondrocytes using the CCK-8 assay. The results showed that compared with the control group, LUP at concentrations of 2.5, 5, 10, and 20 μmol/L had no significant effect on cell viability, while 40 μmol/L LUP significantly inhibited cell viability. After treatment with 50 μmol/L TBHP, cell viability decreased significantly, but 20 μmol/L LUP significantly restored it. Treatment with 20 μmol/L LUP for both 24 and 48 h enhanced cell viability, with the effect at 24 h being significantly better than that at 48 h. These findings indicated that LUP at 20 μmol/L for 24 h exerted the most significant effect of chondrocyte intervention. Subsequently, immunofluorescence and transmission electron microscopy were adopted to assess the regulation of autophagy in TBHP-treated chondrocytes by LUP, and the autophagy inhibitor CQ was used to verify the role of autophagy. The results showed that in the TBHP group, P62 protein increased, the LC3BⅡ/LC3BⅠ ratio decreased, and autophagosomes were reduced. LUP reversed the inhibition of autophagy. After the addition of CQ, the protective effect of LUP was inhibited, cell viability decreased, and indicators of senescence, apoptosis, and inflammation increased, indicating that LUP alleviated TBHP-induced chondrocyte injury by activating autophagy. Furthermore, si-RNA interference experiments were carried out to investigate the role of the SIRT3/mTOR pathway in LUP-mediated autophagy regulation. The results showed that in the TBHP group, the level of SIRT3 protein decreased, while the level of phosphorylated mTOR (p-mTOR) increased. LUP upregulated SIRT3 expression and downregulated p-mTOR expression. After SIRT3 silencing, the LUP-induced activation of autophagy was blocked, indicating that LUP exerted its protective effect by inducing autophagy via the SIRT3/mTOR pathway. In summary, LUP enhances autophagy through the activation of the SIRT3/mTOR pathway, thereby reducing TBHP-induced oxidative stress and senescence in chondrocytes, and ultimately alleviating the progression of osteoarthritis in mice. These findings provide a potential therapeutic target for the treatment of osteoarthritis.

3 Antioxidant effects

Oxidative stress[10] results from excessive production of reactive oxygen species (ROS) in the body or impaired function of the antioxidant defense system. Its core pathological manifestation is the overproduction of ROS and decreased activity of antioxidant enzymes, leading to lipid peroxidation, protein damage, and DNA mutation. Studies have shown that LUP has good antioxidant effects.

Cui *et al.*[11] evaluated the effect of LUP on cardiac function in MI/RI rats using echocardiography and hemodynamic measurements. The results showed that compared with the I/R model group, the heart rate (HR), left ventricular systolic pressure (LVSP), left ventricular ejection fraction (LVEF), left ventricular fractional shortening (FS), and left ventricular wall thickness (LVWT) in the 20 and 50 mg/kg LUP groups and the positive control group all increased significantly. It indicated that LUP at 20 and 50 mg/kg significantly improved both systolic and diastolic function in MI/RI rats. Subsequently, serum cardiac injury markers were measured using biochemical methods to evaluate the protective effect of LUP against myocardial injury. The results showed that compared with the I/R group, the levels of creatine kinase isoenzyme (CK-Mb), myoglobin (Mb), and cardiac troponin I (cTnI) in the 20 and 50 mg/kg LUP groups decreased significantly, indicating that LUP alleviated cardiomyocyte membrane damage and exerted a cardioprotective effect. Based on these findings, immunohistochemistry was performed to examine the effect of LUP on myocardial morphology and apoptosis. The results showed that in the I/R group, myocardial fibers were disorganized and edema

was pronounced, whereas LUP at 20 and 50 mg/kg significantly ameliorated the myocardial structure, indicating that LUP protected myocardial tissue architecture, promoted cardiomyocyte proliferation, and inhibited apoptosis. Furthermore, biochemical methods were adopted to measure inflammatory cytokines and oxidative stress markers. The results showed that compared with the I/R group, the levels of TNF-α, IL-1β, iNOS, IL-6, and MDA increased significantly, while SOD activity increased significantly in the 20 and 50 mg/kg LUP groups. These findings indicate that LUP could suppress the inflammatory response, enhance antioxidant capacity, and alleviate myocardial oxidative injury. In summary, LUP can improve cardiac function, reduce myocardial injury markers, alleviate myocardial pathological damage, and inhibit inflammation and oxidative stress.

4 Ameliorative effects on type 2 diabetes mellitus

Type 2 diabetes mellitus[12] is a common endocrine syndrome characterized by glucose metabolism disorder, often accompanied by dyslipidemia and complicated by hyperlipidemia. Its core pathological manifestations include dysregulation of blood glucose, decreased insulin sensitivity, abnormalities in glucose and lipid metabolism, and chronic inflammatory responses. Studies have shown that LUP has a good improving effects on type 2 diabetes mellitus.

Zhang *et al.*[13] evaluated the effect of LUP on blood glucose regulation in diabetic mice by measuring fasting blood glucose (FBG). The results showed that compared with the model control (MC) group, LUP significantly reduced the FBG levels in db/db mice, indicating that LUP effectively could maintain glucose homeostasis. Subsequently, fasting insulin (FINS) levels were measured, and the homeostatic model assessment of insulin resistance (HOMA-IR) index was calculated. The results showed that compared with the MC group, LUP at 20, 40, and 80 mg/kg reduced FINS levels and decreased the HOMA-IR index in a dose-dependent manner, indicating that LUP could enhance insulin sensitivity. Based on these findings, immunofluorescence staining was performed to evaluate the protective effect of LUP on pancreatic tissue and islet β-cells in diabetic mice. The results showed that in the MC group, islets were atrophied, cell numbers were reduced, and insulin fluorescence signals were significantly weakened. After LUP intervention, islet morphology was restored to a regular shape, and the number of cells increased, indicating that LUP could protect islet structure, alleviate β-cell damage, and restore insulin secretion function. Further serum biochemical analysis was performed to evaluate the regulatory effect of LUP on the lipid profile in diabetic mice. The results showed that a high dose of LUP (80 mg/kg) significantly reduced TC, TG, and LDL-C levels while increasing HDL-C levels, thereby comprehensively restoring lipid homeostasis. These findings indicate that LUP can significantly ameliorate the lipid metabolism disorders associated with type 2 diabetes, thereby interrupting the vicious cycle of glucose and lipid metabolism. In summary, LUP can reduce fasting blood glucose, improve glucose tolerance and insulin resistance, and protect the structure and function of islet β-cells. It can also reduce visceral fat accumulation, and correct lipid disorders. By synergistically regulating the homeostasis of glucose and lipid metabolism, LUP exerts a significant intervening and alleviating effect on type 2 diabetes, demonstrating its potential to be developed as a candidate drug for lowering blood glucose and lipids.

5 Conclusions and prospects

LUP exhibits various pharmacological effects, including resisting tumors and inflammation, alleviating oxidative stress injury, and improving type 2 diabetes mellitus. As an important triterpenoid active compound of natural origin, the optimization of its extraction and isolation processes, as well as the investigation of its pharmacological activities, have achieved preliminary success. However, the in-depth analysis of its specific pharmacological mechanisms and clinical translational application are still in the early stages. It is necessary to continuously integrate knowledge theories and experimental techniques from molecular biology, cell biology, experimental zoology, pharmacology, basic medicine, and related fields to conduct more comprehensive and in-depth studies of lupeol at the molecular, cellular, and animal levels. These efforts will provide a theoretical basis and data support for the further development of natural pharmaceutical preparations of LUP.

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3.4 Constructing a virtual medical experiment platform (VMC-100) The VMC-100 2.0 virtual simulation teaching software employs a combined online and offline model, playing a positive role in stimulating students to explore problems, solve problems, and think independently.

3.5 Constructing an intelligent management system (ILS-100) The ILS-100 smart laboratory system can connect multiple sets of different laboratory equipment, thereby forming a comprehensive laboratory solution with configurability and scalability. This smart laboratory system realizes the smartification of personnel management, course teaching, equipment management, experimental evaluation, environmental monitoring, and information management.

4 Conclusions

The implementation of smart functional experiments has brought significant multi-dimensional results to modern medical education. Relying on a highly integrated and automated system, it optimizes the experimental teaching process in all aspects, improving teaching efficiency and quality. Meanwhile, leveraging functions such as intelligent formative assessment, it achieves multi-dimensional automatic analysis and feedback on students' operational skills and experimental results, significantly enhancing the objectivity of the teaching process and the pertinence of guidance.

Furthermore, smart functional experimental teaching enhances the standardized collection and in-depth analysis capabilities of experimental data, improves the scientific nature and reliability of experimental results, and achieves intelligent scheduling and management of laboratory resources, effectively reducing operating costs.

In comparison, the traditional functional experimental model typically relies on non-integrated equipment and manual operations; due to scattered equipment and complicated steps, it often leads to low operational efficiency. In terms of teaching evaluation, it lacks a systematic automatic assessment mechanism, relying mostly on teachers' subjective experience, making it difficult to conduct comprehensive and objective formative assessments. Simultaneously, there are obvious defects in data integration and resource management, thus facing significant challenges in adapting to the needs of informatization and refined development in modern medical education.

In summary, this project is closely linked to the construction of smart functional experiments at our university. After rigorous research, the relevant schemes were submitted to the college and university levels. During this period, experts were organized multiple times for discussion and demonstration, ultimately forming the Construction Plan and Implementation Scheme for the "Four-in-one" Smart Functional Experimental Teaching Center of Hubei University of Medicine. This scheme conforms to the development trend of "Internet + Education", helps promote the deep integration of information technology with education and teaching, and has a significant driving effect on improving the level of informatization construction and application at our university.

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