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Review

Chemical Constituents and Pharmacological Effects of *Camellia petelotii* (Merr.) Sealy: A Review

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Abstract

Camellia petelotii (Merr.) Sealy (Jinhuacha, JHC) is a rare plant species native to Guangxi, China, and northern Vietnam, renowned for its significant ornamental, nutritional, and medicinal properties. To date, 177 chemical constituents have been isolated and identified from JHC, including Flavonoids, Terpenoids, Steroids, Phenylpropanoids, Essential oils, Alkaloids, and Organic acids. Among these, Quercetin, Kaempferol, Luteolin, Catechin, Oleanolic acid, and Camelliaside A are the primary bioactive compounds. The JHC exhibits diverse pharmacological activities, including hypolipidemic and hypoglycemic effects, anti-inflammatory, antioxidant, antibacterial, antidepressant, and antitumor properties. Studies have demonstrated that extracts of JHC effectively inhibit pancreatic lipase (PL) and cholesterol esterase (CEase), with median inhibitory concentrations (IC_{50}) values of 320 μg/mL and 200 μg/mL, respectively. Additionally, the extracts show strong free radical scavenging activity, with 2,2-Diphenyl-1-picrylhydrazyl (DPPH) and 2,2′-azino-bis(3-ethylbenzothiazoline-6-sulfonic acid) (ABTS) radical clearance rates reaching 84.61% and 95.51%, respectively. Clinical applications indicate that JHC Hypoglycemic Capsules achieve a total effective rate of 93.33% in the adjunctive treatment of patients with type 2 diabetes. Nevertheless, the quality control markers and the precise mechanisms underlying certain pharmacological effects of JHC remain to be fully elucidated. This paper systematically reviews current advances in the study of the chemical constituents and pharmacological effects of JHC, and further proposes future research directions, aiming to provide a scientific foundation for the in-depth investigation of its active constituents and mechanisms of action.

Keywords: *Camellia petelotii* (Merr.) Sealy; chemical constituents; pharmacological effects

1. Introduction

Camellia petelotii (Merr.) Sealy (Jinhuacha, JHC) is an evergreen shrub or small tree belonging to the genus Camellia in the family Theaceae. It was first discovered in Guangxi, China, in the early 1960s and is mainly distributed in southwestern China and northern Vietnam (Fig. 1). Recognized as a rare and endangered species, it is renowned for its striking golden-yellow flowers (Fig. 1B) and is often referred to as the "Queen of Tea" [1–3]. Currently, JHC has been introduced to Japan, Australia, and North America as a valuable genetic resource for commercial camellia cultivation, drawing significant attention from horticulturists worldwide [4]. JHC possesses not only significant ornamental value but also considerable medicinal and edible applications. Its extract can serve as a natural food coloring agent, while its seeds can be processed for edible oil or utilized as industrial raw materials. Additionally, the leaves of JHC can be prepared as an infusion for consumption. This plant is traditionally used as a folk remedy by the Zhuang ethnic group in Guangxi, China [5]. According to authoritative references such as the "Guangxi Standards for Chinese Medicinal Materials" and the "Modern Compendium of Materia Medica", it has been documented for the prevention and treatment of pharyngitis, nephritis, dysentery, tumors, hematochezia, hypertension, and menstrual disorders. According to the Chinese Materia Medica, the flowers of JHC are administered in dosages of 3–9 g, prepared by boiling in water or as a decoction, primarily for the treatment of hematochezia and excessive menstruation. The leaves, with a recommended dosage of 9–15 g, are used to reduce fever, detoxify, and alleviate dysentery. Fresh plant materials may also be crushed and applied topically in appropriate amounts for hemostasis. The first record of JHC was made in 1552 in the Compendium of Materia Medica (Bencaogangmu) during the Ming Dynasty. The description in the book reads, "JHC is produced in the south.... It blossoms in late winter, with red petals and yellow stamens.... the flowers are golden yellow". The botanist Jinglie Zuo first discovered the wild JHC population in 1933 in Fangchenggang, Guangxi, China. In 1965, it was officially named *Camellia nitidissima* C. W. Chi by the botanist Jingwen Qi. In 1984, it was listed as a first-class

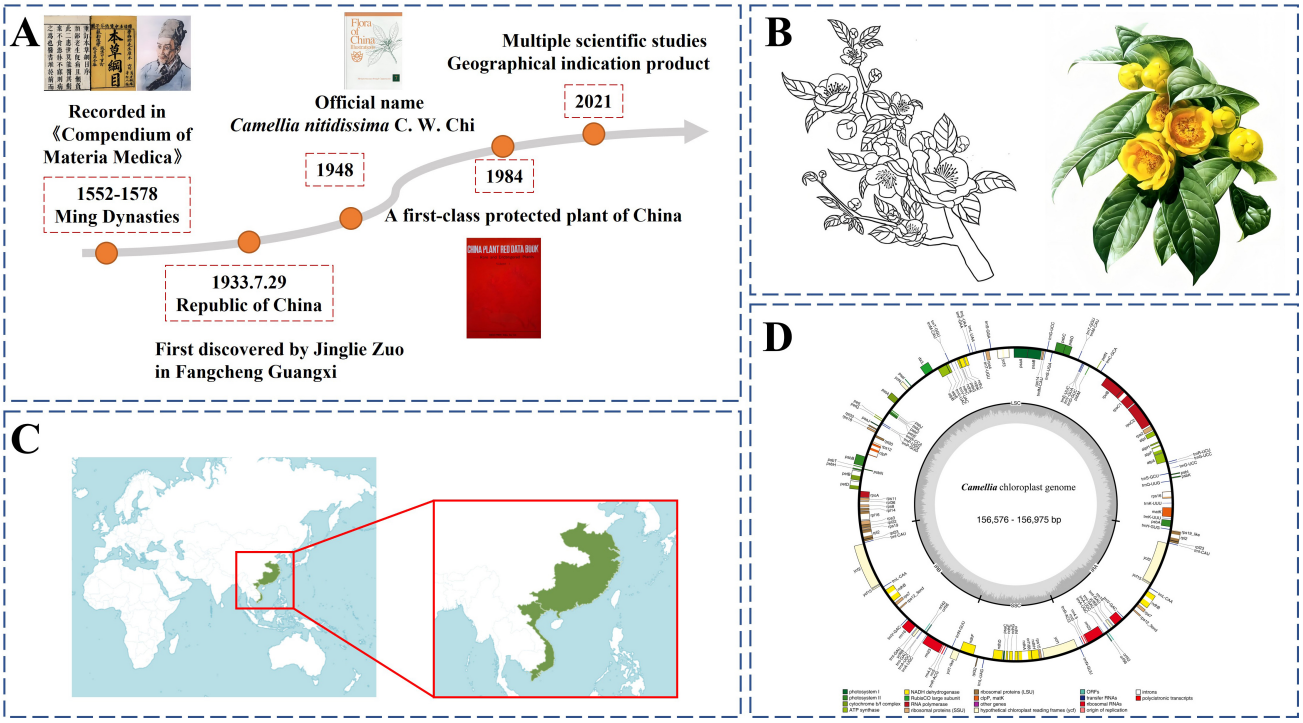


Fig. 1. Introduction to JHC. (A) The history of consumption and medicinal use of JHC. (B) Flower and leaf morphology of JHC. (C) Geographical distribution of JHC. (D) The chloroplast genome size of JHC. JHC, Jinhuacha.

protected plant of China. In 2010, the Chinese National Health Commission changed its scientific name to *Camellia petelotii* (Merr.) Sealy. In 2021, it became a geographical indication brand of Fangchenggang, Guangxi, China (Fig. 1A), with great development potential.

At present, research on JHC mainly focuses on germplasm resources, genetic diversity, cultivation physiology, isolation and identification of chemical components, and activity analysis. Among them, research in molecular biology mainly concentrates on gene sequencing and the role of genes in substance synthesis and metabolism. For instance, some research reports indicate that the total length of the chloroplast genome of JHC is between 156,575 and 156,975 bp, providing a research basis for the identification of JHC species [6]. In recent years, with the continuous breakthroughs in cultivation techniques, the output of JHC has steadily increased, and various functional products have emerged on the basis of various scientific researches (Fig. 2) [7]. More and more chemical components have been isolated and identified from JHC, e.g., Flavonoids, Terpenoids, Steroids, and Phenylpropanoids. These are confirmed to have multiple pharmacological effects, such as lowering of blood sugar, reduction of swelling, and anti-tumor effects [8]. Notably, there are significant differences in the component contents among different species of Camellia petelotii [9–11]. However, the quality markers of JHC and the specific mechanisms by which it exerts its pharmacological effects remain unclear, and there is no comprehensive overview of its chemical components and pharmacological effects. This has led to the public's insufficient understanding of the components and benefits of JHC, restricting the enhancement of its brand awareness and the development of the industry.

Therefore, it is necessary to conduct this review to fully explore its chemical components and pharmacological effects and clarify the future research direction. In view of this, we take "*Camellia petelotii*", "*Camellia nitidissima*" and "*Yellow Camellia*" as the key words. The chemical components and pharmacological activities of JHC are systematically screened in the Web of Science, PubMed, Elsevier, ScienceDirect, Google Scholar, Baidu Scholar, and China National Knowledge Infrastructure, CNKI, thus providing a reference for the further development and utilization of JHC.

2. Traditional Uses and Clinical Applications

A review of the classical literature and historical records reveals that traditional applications of JHC primarily focus on the treatment of dysentery, faucitis, hepatitis, and cardiovascular diseases. According to the "Guangxi List of Medicinal Plants", JHC leaves can reduce fever to promote salivation and are effective in the treatment of dysentery. As outlined in the "Guangxi Traditional Chinese Medicine Standards", JHC has the capacity to remove heat and toxic material and can induce diuresis to reduce edema. It can be used for the treatment of pharyngitis, dysentery, nephritis, edema, urinary tract infections, icteric hepatitis, ascites associated with liver cirrhosis, hyperten-

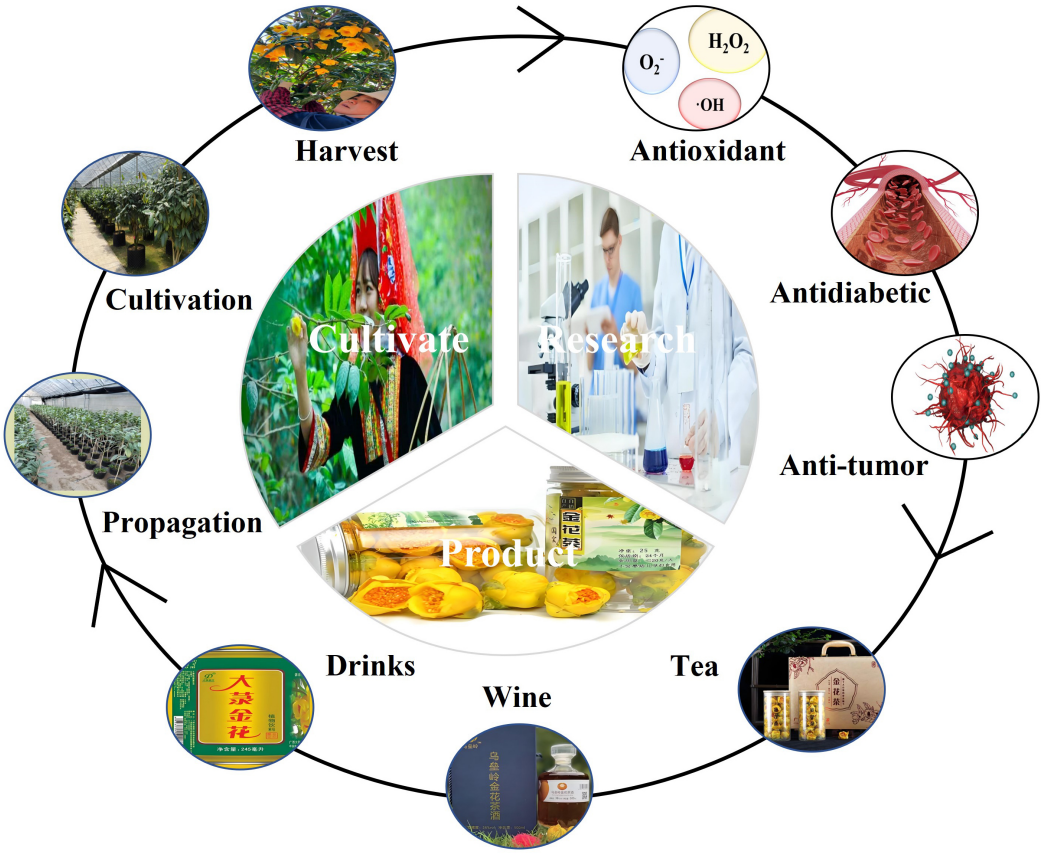


Fig. 2. The main research focus of JHC including propagation and cultivation, activity research and product development.

sion, sores, ulcers, and for tumor prevention. The "Modern Compendium of Materia Medica" documented its efficacy in treating dysentery, with the flowers indicated for hematochezia and excessive menstrual bleeding. According to the "Chinese Materia Medica", the JHC flower has astringent and hemostatic properties and is used primarily in the treatment of hematochezia and excessive menstruation. JHC leaves were known to clear heat, detoxify, and arrest dysentery, and were thus commonly used to treat dysentery, pharyngitis, and skin ulcers. Furthermore, JHC was widely utilized in clinical practice by ethnic minorities in Guangxi as a hepatoprotective agent (Fig. 3). Contemporary clinical data suggest that JHC hypoglycemic capsules could serve as an adjunct in the treatment of type 2 diabetes, with a reported overall efficacy of 93.33% [12]. Continuous consumption of JHC over a three-month period by patients with hypertension could reduce systolic blood pressure by 10–15 mmHg and diastolic blood pressure by 5–10 mmHg. The antihypertensive effects of JHC usually begin to manifest within 1–2 h after administration [13]. These traditional and modern therapeutic applications are closely linked to the rich array of chemical components found in JHC, particularly flavonoids, triterpenoids, and saponins, which constitute the biochemical foundation of its pharmacological effects. The aim of this review is to summarize the chemical composition and pharmacological activities of JHC, thereby providing a scientific basis for understanding its traditional medicinal uses.

3. Chemical Constituents

JHC has attracted much attention for its unique phytochemical resources. This review systematically summarizes the compounds isolated and identified from the flowers and leaves of JHC. Table 1 presents the categories, proportions, representative compounds and their possible biological activities of the components currently isolated and identified from JHC. Including Flavonoids (**1–60**), Terpenoids (**61–131**), Steroids (**132–136**), Phenylpropanoids (**137–149**), Essential oil (**150–163**), Organic acid (**164–168**) and Others (**169–177**). The **Supplementary Table 1** was details of the component.

3.1 Flavonoids

Flavonoids are characteristic secondary metabolites in JHC and show significant structural diversity in chemical taxonomy and biological activity. The flavonoids identified to date mainly include Flavonols (**1–3**), Flavanols (**5–8**), Biflavonoids (**16**), and their derivatives (Fig. 4). The highest content is catechin (39.31%) and quercetin (38.20%) [14]. From the perspective of structural characteristics, this type of component typically has the following substitution patterns: hydroxyl substitution at the C-5 position of the par-

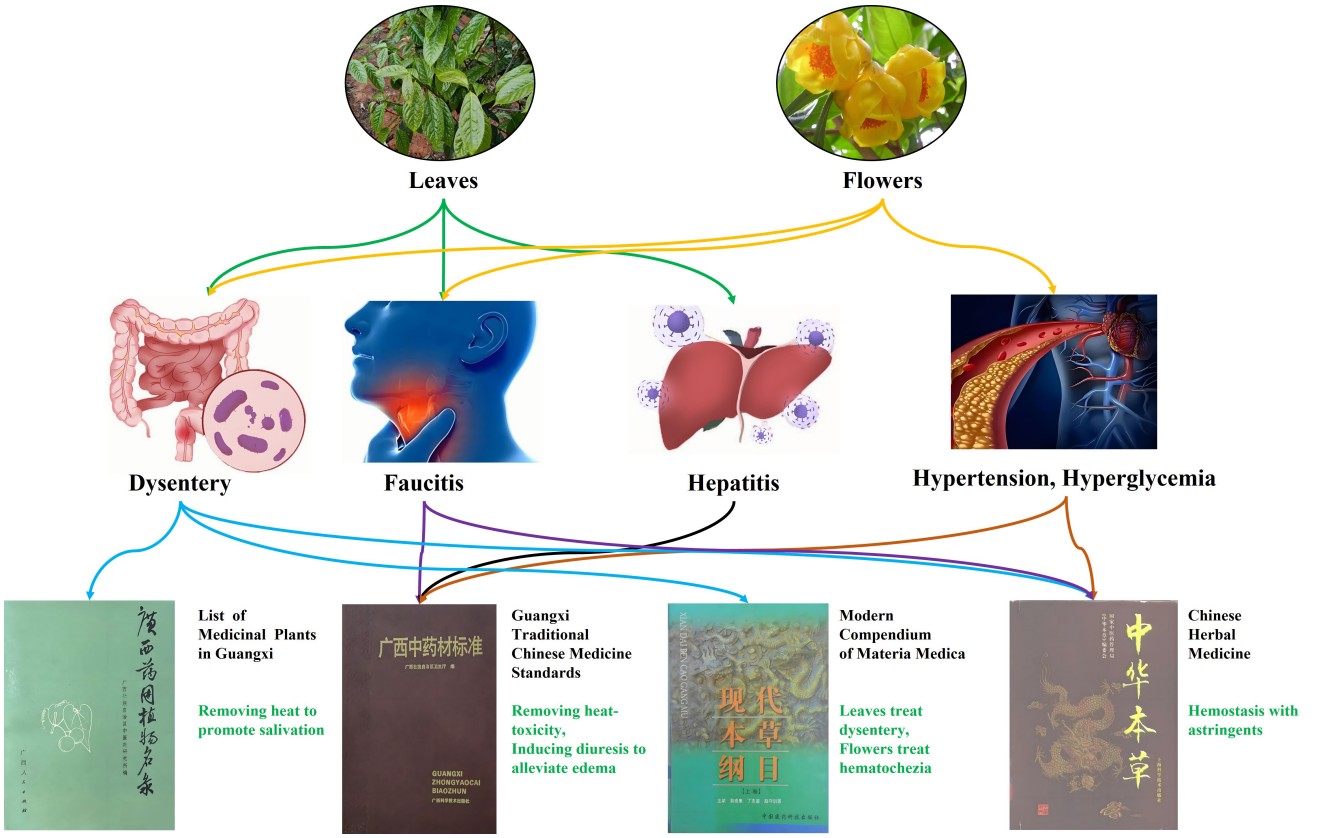


Fig. 3. The uses of JHC recorded in traditional Chinese materia medica throughout history. It is mainly used for the treatment of dysentery, faucitis, hepatitis, and cardiovascular diseases.

Table 1. The categories, proportion and representative compounds of components in JHC and their effects.

Categories	Proportion	Representative Compounds	Effects
Flavonoids	34%	Quercetin[1]	Reduces blood pressure, enhances capillary resistance, decreases capillary fragility, lowers blood lipid levels, dilates coronary arteries, and increases coronary blood flow
		Luteolin[4]	Anti-inflammatory, anti-allergic, uric acid-lowering, anti-tumor, antibacterial and antiviral
		Epigallocatechin gallate[13]	Antioxidant, antibacterial, antiviral, antiparasitic and enhancing the immune capacity of the body
		Rutin[20]	Anti-inflammatory, antioxidant and antiviral
Terpenoids	40%	Ginsenoside Rg1[110]	Promote neurogenesis, enhance learning and memory abilities, combat fatigue and regulate immunity
		Campetelosides A[116]	Neuroprotective and anti-inflammatory
		Betulin[130]	Anti-inflammatory and anti-tumor
Steroids	3%	β-sitosterol[132]	Lower cholesterol, fight tumors and repair tissues
Phenylpropanoids	7%	Ferulic acid[139]	Antibacterial, antiviral and antioxidant
Essential oil	8%	Vanillin[158]	Antibacterial
Organic acid	3%	Shikimic acid[167]	Anti-inflammatory, antiviral and antioxidant
Others	5%	Theobromine[176]	Stimulating effect on the cardiovascular system

The superscript numbers correspond to the compound numbers mentioned later in the text.

ent nucleus A ring (**1–10**), with the B ring mainly characterized by a single hydroxyl substitution (**19**, **23–28**). In terms of glycosylation modification, the flavonoids in JHC mostly exist in the form of glycosides (**18–60**) (Fig. 5), with their glycosylation connection sites showing obvious regularity. The C-3 hydroxyl group often forms β-configuration side bonds with hexose sugars, e.g., glucose and galactose (**18–34**), while the C-7 hydroxyl group tends to be differ-

ent from monosaccharides or disaccharides (**35–41**). This differentiated sugar group substitution pattern has a significant impact on molecular polarity. From the perspective of structure-activity relationships, the α,β-unsaturated ketone conjugated system formed by the 2,3-position double bond of the C ring of the flavonoid parent nucleus with the 4-position carbonyl group (**1–4**, **16**) can effectively stabilize the free radical intermediate through π-π stacking. This constitutes the core pharmacophore of antioxidant activity.

For instance, the antioxidant activity of Quercetin (**1**) is primarily attributed to the α,β-unsaturated ketone conjugated system formed by the double bond at the 2,3-position and the carbonyl group at the 4-position, as well as the 3′,4′-ortho-dihydroxyl group in the B ring. Additionally, the hydroxyl groups at the 5-position and 7-position in the A ring and the free hydroxyl group at the C-3 position play a significant regulatory role in electron delocalization, metal chelation, and stabilization of free radicals. The antibacterial activity of Quercetin is mainly due to the insertion of its hydrophobic flavonoid backbone into bacterial cell membranes, resulting in membrane disruption, along with enzyme inhibition mediated by the diphenolic hydroxyl group on the B ring, and interference with bacterial biofilm formation by the entire molecular structure. The antioxidant activity of kaempferol (**2**) is largely derived from the catechol-like structure in the B ring and the conjugated system involving the C2=C3 double bond, which enables efficient scavenging of free radicals and stabilization of phenoxyl radicals. Its antibacterial properties are associated with the molecule's hydrophobicity, the positioning of phenolic hydroxyl groups, and molecular planarity—features that facilitate disruption of microbial membrane integrity and inhibition of essential enzymatic activities. The primary antioxidant capacity of (-)-Catechin (**5**) and (+)-catechin (**6**) is rooted in the 3′,4′-dihydroxyl configuration on the B ring [15]. Their antibacterial effects are predominantly mediated through a synergistic interaction between this catechol moiety and the overall hydrophobic character of the molecule, acting via mechanisms including induction of oxidative stress, metal ion chelation, and perturbation of cellular membranes.

3.1.1 Flavones

3.1.2 Flavone Glycosides

The above-mentioned structural specificity establishes chemical classification markers for JHC, e.g., the combined characteristics of C-5 hydroxyl-substituted flavonols and B-ring monohydroxylation mode. More importantly, it also provides a clear molecular framework for the targeted structural modification of flavonoid components. Based on existing structure-activity relationships, the lipophilicity and bioavailability of JHC can be regulated through strategies such as directed glycosylation/acylation and selective hydroxyl protection, while its antioxidant efficacy can be enhanced through the design of metal chelating sites. This lays a solid chemical foundation for the development of new flavonoid functional molecules.

3.2 Terpenoids

The structural diversity and chemical characteristics of terpenoids in JHC have become an important direction in phytochemical research. From the perspective of biosynthesis, the molecular skeletons of terpenoids all originate from the directional polymerization and c-ring modification of isoprene units. Moreover, their structural complexity increases significantly as the degree of polymerization increases.

Monoterpenoids are based on the head-tail connection of two isoprene units and form various types of skeletons through different cyclization mechanisms. These include acyclic monoterpenoids (**61–68**), which have characteristic terminal allyl alcohol groups within their molecules, as well as monocyclic and iridoid glycosides (**69–76**) (Fig. 6). The structural characteristics of sesquiterpenes are reflected in their unique ring system combinations and oxidation modification sites. Their skeletal structure includes chain-like and single-ring sesquiterpenoids (**77–88**), and bicyclic sesquiterpenes (**89–100**) (Fig. 7). The sesquiterpene components undergo oxidation at C4, C7, or C11 positions to form polar groups such as hydroxyl, ketone, or epoxide groups, highlighting their structural diversity. Triterpenoids are characteristic secondary metabolites of higher plants and exhibit a significant structural evolution from linear to highly cyclized. They are typically represented by the oleane-type pentacyclic system (**102–103**), with the carboxyl group at the C17 position often forming an ester bond with a sugar group to constitute a saponin (**116–123**) (Fig. 8). The diverse structures of terpenoids in JHC not only reveal the biosynthesis rules of its secondary metabolites, but also provide a theoretical basis at the molecular level for further in-depth development of its medicinal value.

3.2.1 Monoterpenoids

3.2.2 Sesquiterpenes

3.2.3 Triterpenoids

3.3 Steroids

The aglycone types of steroidal saponins are based on cholestane and stigmastane as the basic framework (**132–133**). The hydroxyl group at the C-3 position is combined with the sugar chain through glycosidic bonds. The length of the sugar chain ranges from monosaccharides to disaccharides (**134–136**) (Fig. 9). The structural diversity provides a key molecular basis for the classification of chemical components and the evolution of secondary metabolic pathways in JHC.

3.4 Phenylpropanoids

The phenylpropyl compounds in JHC include three major structural types: phenylpropionic acid, coumarin,

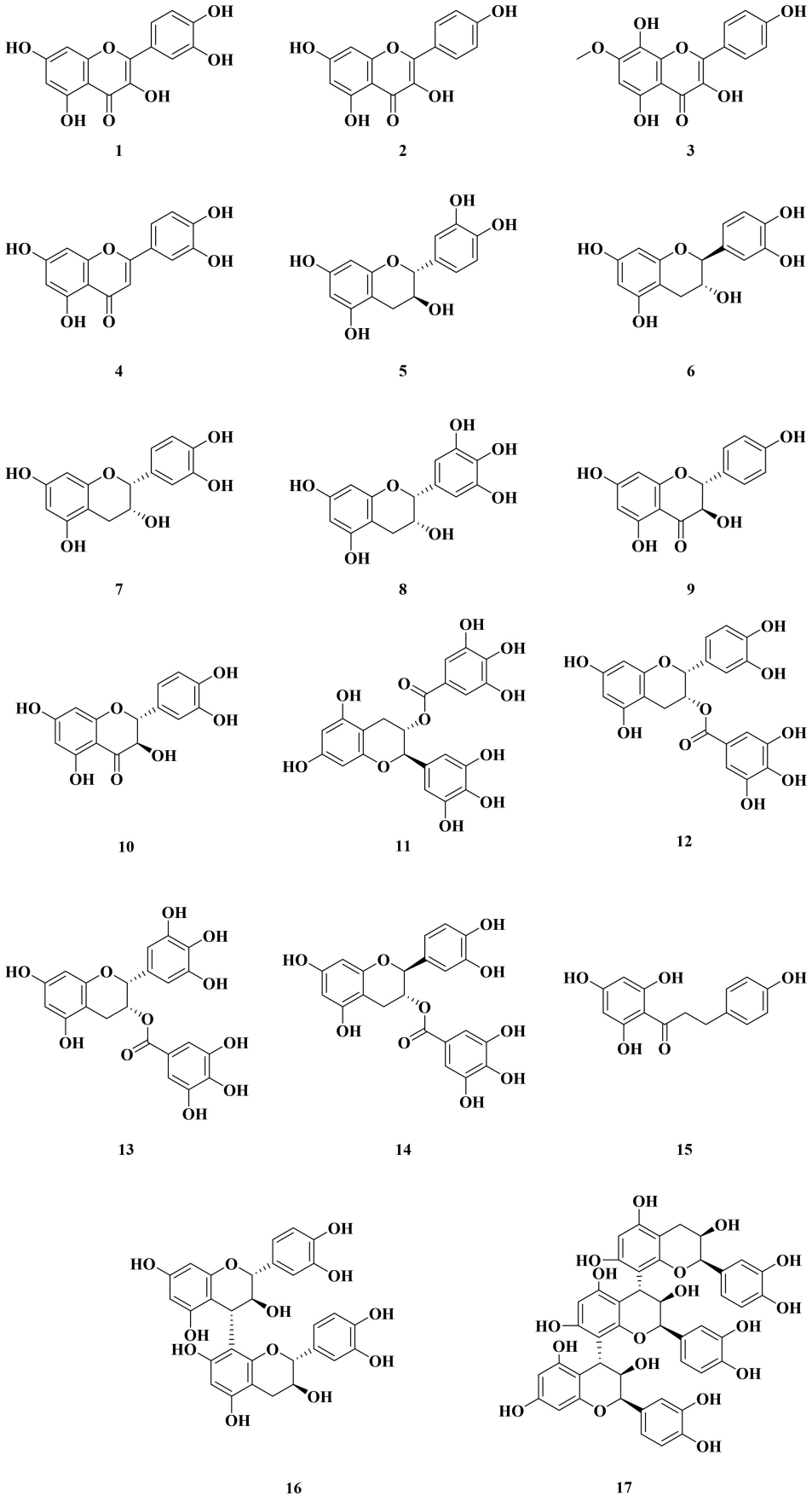
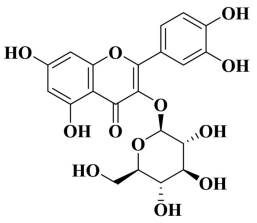


Fig. 4. Flavones compounds in JHC.



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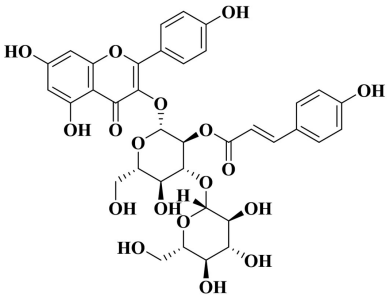

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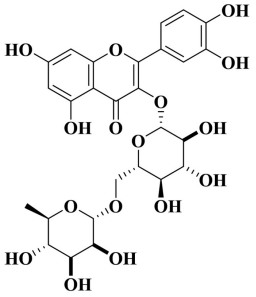

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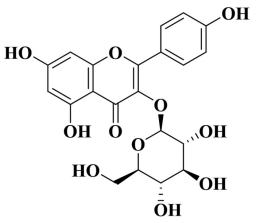

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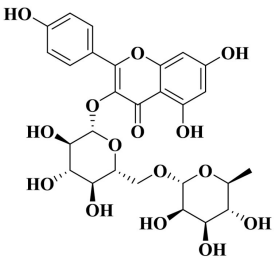

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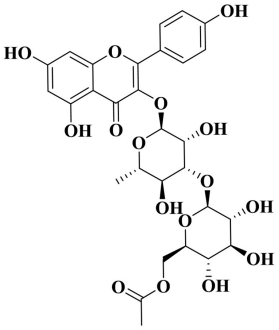

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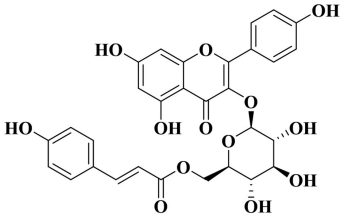

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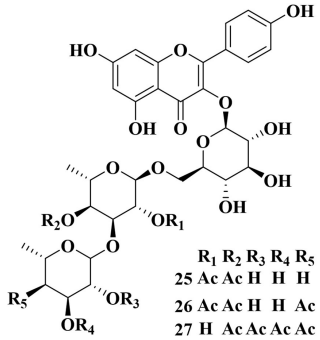
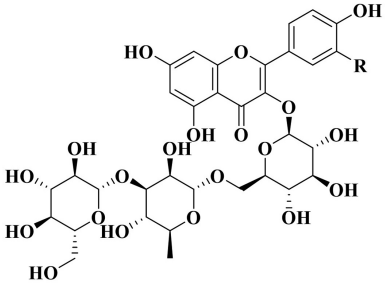
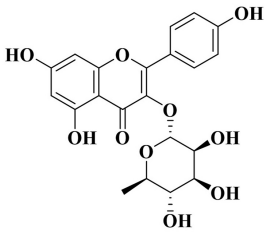


	R_1	R_2	R_3	R_4	R_5
25	Ac	Ac	H	H	H
26	Ac	Ac	H	H	Ac
27	H	Ac	Ac	Ac	Ac
28	Ac	Ac	Ac	Ac	Ac



	29	30
R	H	OH



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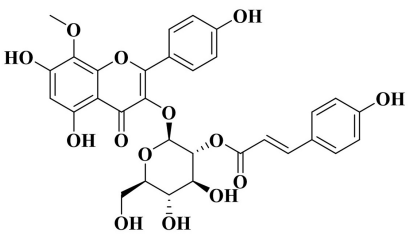

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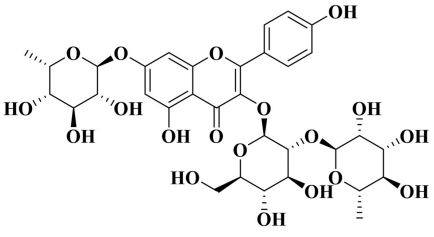

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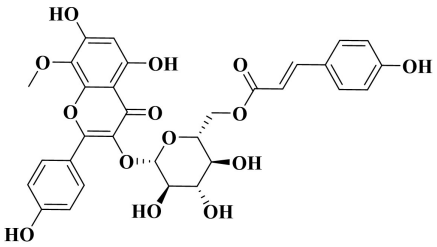

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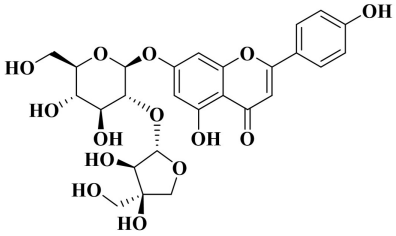

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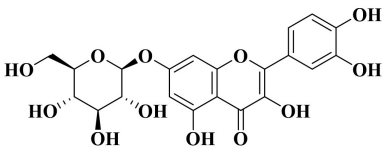

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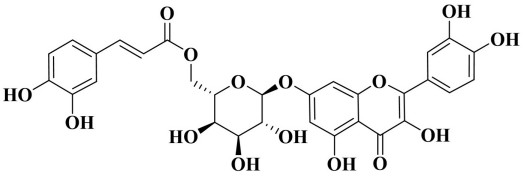

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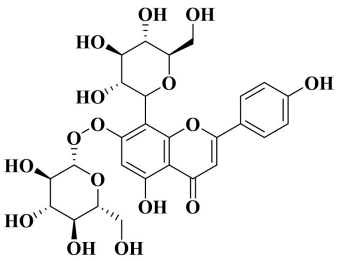

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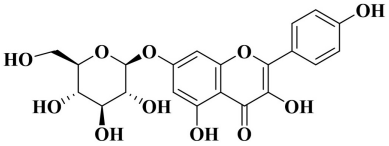

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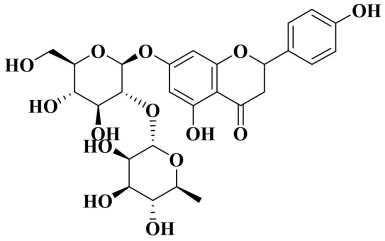

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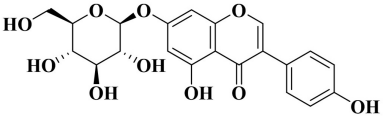

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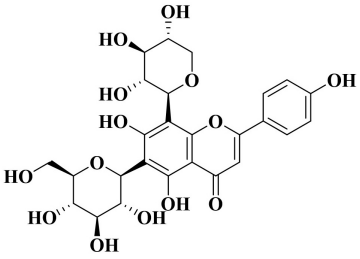

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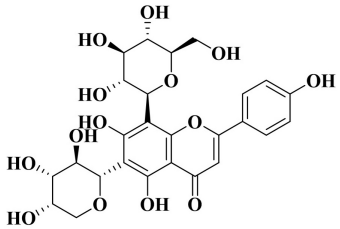

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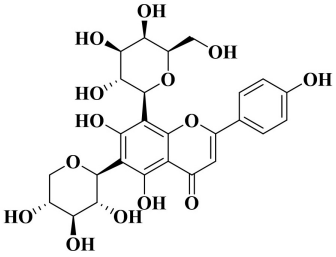

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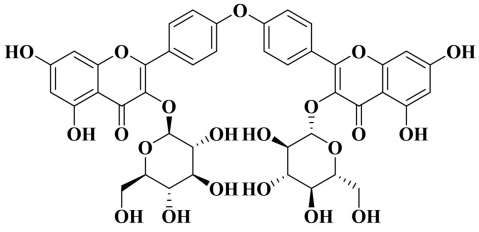

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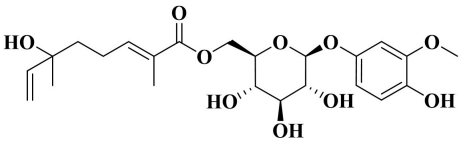

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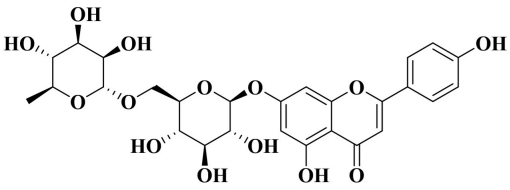

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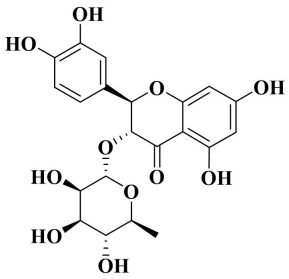

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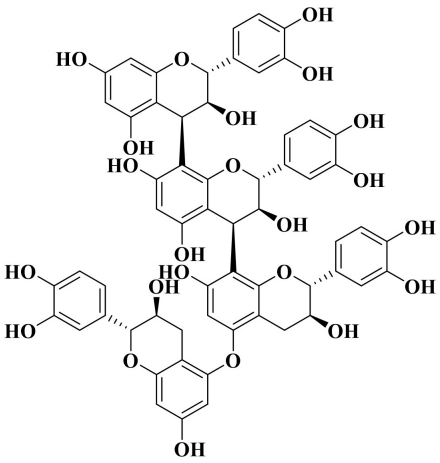

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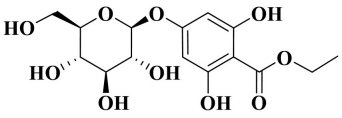

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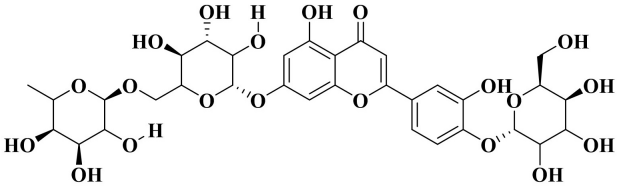

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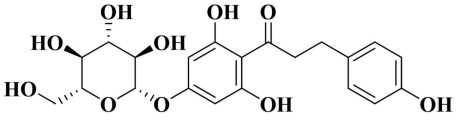

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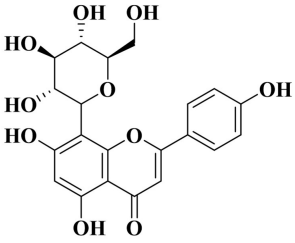

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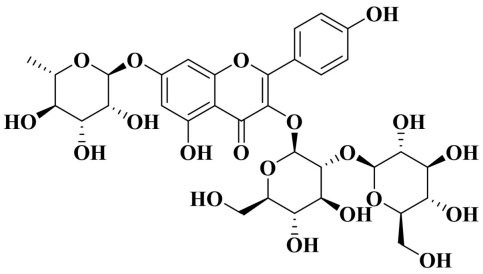

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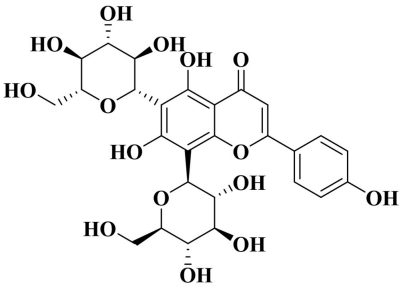

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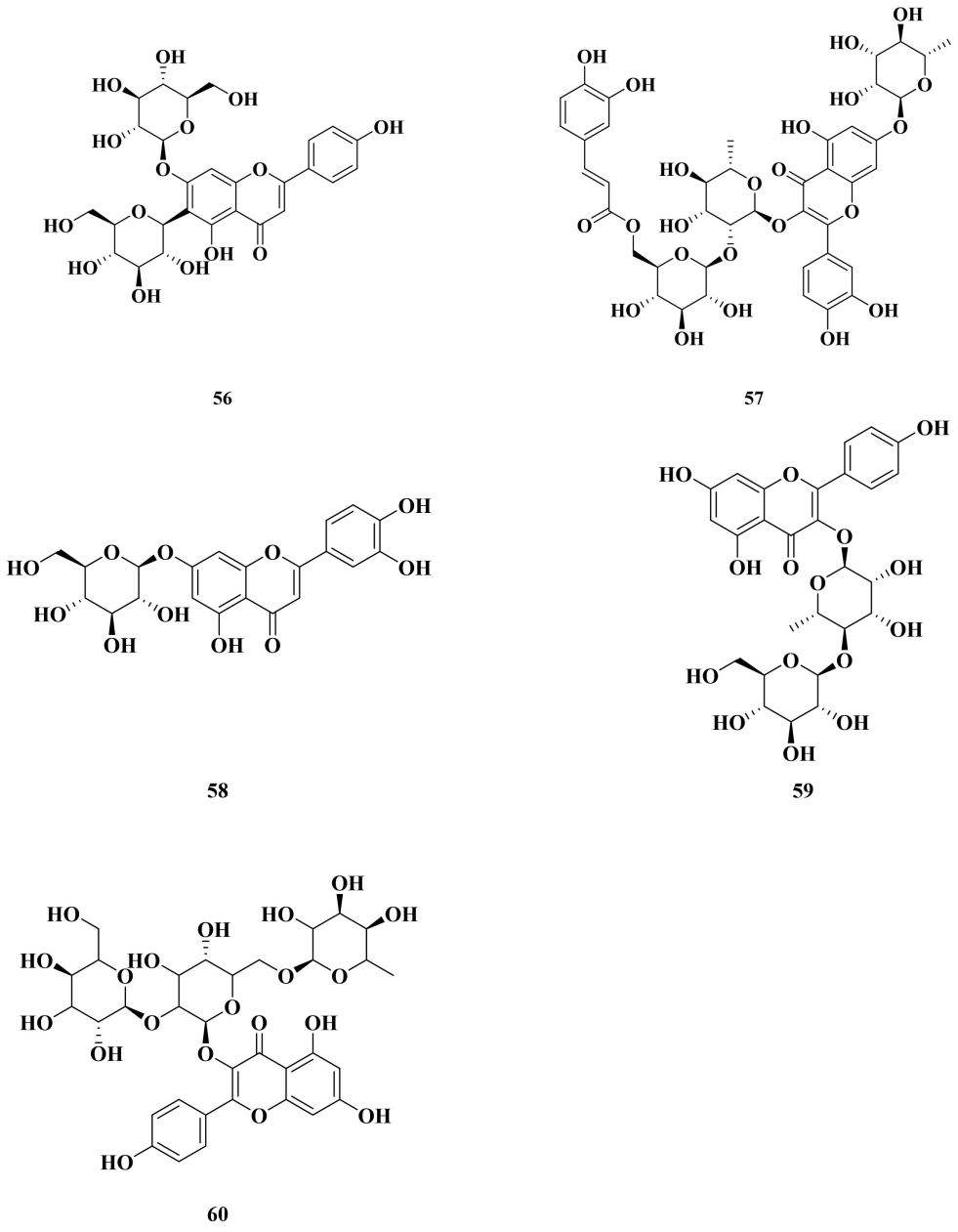


Fig. 5. Flavone glycosides in JHC.

and lignan. Their molecular characteristics show systematic chemical diversity. The phenylpropionic acid class is based on the C6-C3 type phenylpropane skeleton, and its core compounds include cinnamic acid and its ester derivatives (**137–139**). Coumarins have the benzo α-pyranone parent nucleus as the core (**140–141**, **145**, **147**), with some molecules undergoing isoprene formation at the C5/C8 position. Lignans form a dibenzylbutane-type (**142–143**) skeleton through the connection of phenylpropyl units, and some contain lactone rings or glycoside modifications (**145–149**) (Fig. 10). This type of structural system has led to a unique chemical map of the secondary metabolites of JHC through multi-dimensional modifications such as hydroxylation, methoxylation, cyclization, and glycosylation.

3.5 Others

Other compounds have also been found in JHC, including essential oils (**150–163**) (Fig. 11), organic acid (**164–168**) (Fig. 12), long-chain fatty acids and their glycosides (**169–177**) (Figs. 13,14), as well as alkaloids.

3.5.1 Essential Oils

3.5.2 Organic Acid

3.5.3 Long-Chain Fatty Acids

3.5.4 Alkaloids

4. Pharmacological Effects

Several recent studies have reported a variety of definite biological activities for JHC. These included enzyme

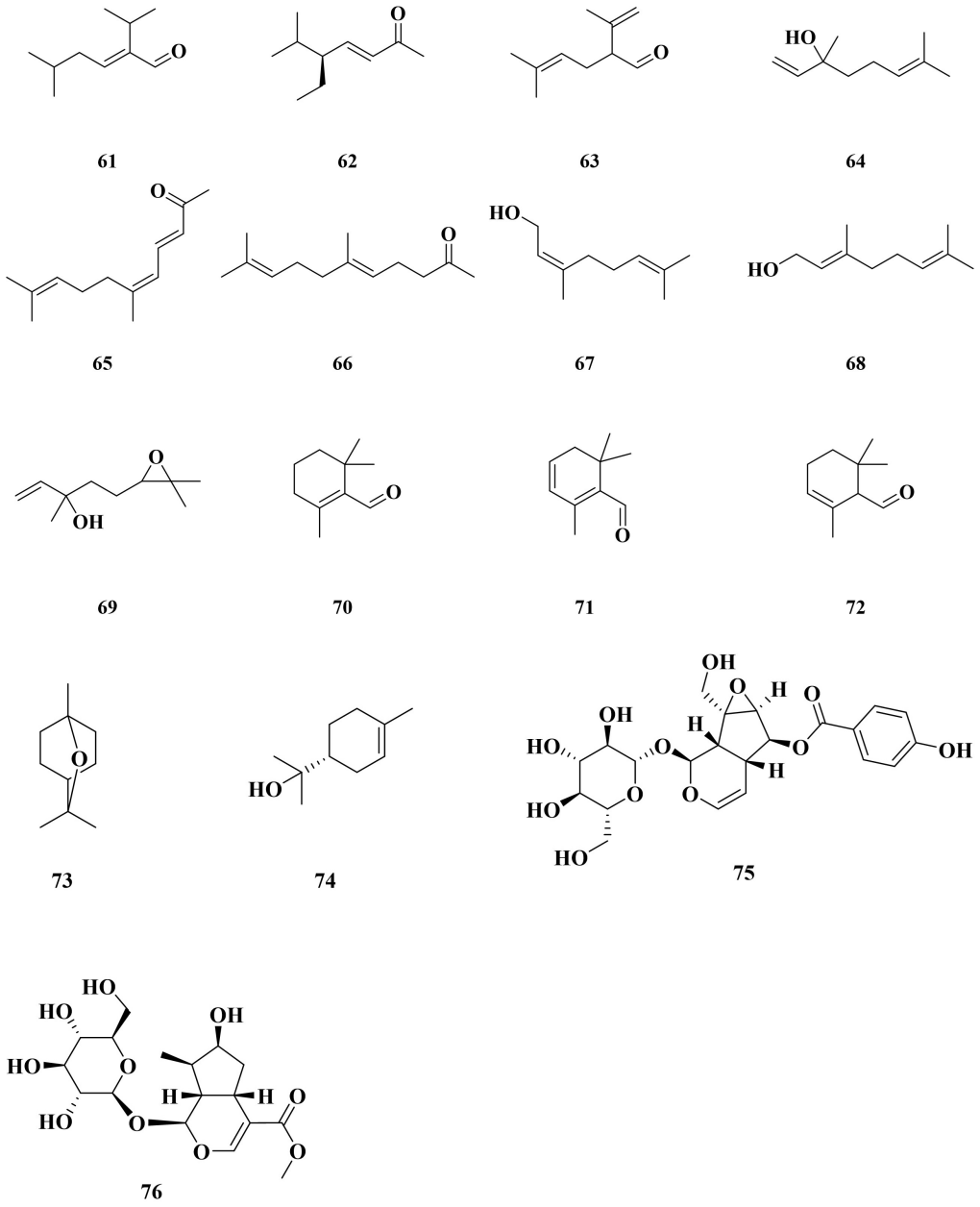


Fig. 6. Monoterpenoids in JHC.

activity assays, *in vitro* cell experiments, animal models, and some preliminary clinical data. They confirmed the extensive pharmacological effects exhibited by the chemical components, mainly flavonoids, terpenoids, and saponins. Specifically, the core biological activities of JHC include lipid reduction and hypoglycemic, anti-inflammatory, anti-tumor, and anti-oxidant effects (Fig. 15). These findings indicate that JHC has multiple health benefits and holds significant potential for disease prevention and treatment.

4.1 Anti-Tumor Effects

Multiple studies have confirmed that JHC exerts inhibitory effects on various human cancers such as lymphoma, esophageal squamous cell carcinoma, gastric cancer, lung cancer, colon cancer, and liver cancer. This function is related to various components in JHC, such as flavonoids, terpenoids, polyphenols, saponins, and polysaccharides (Table 2, Ref. [16–45]).

A new flavonoid glycoside extracted from JHC (quercetin 7-O-[600-O-E-caffeoyl]-B-D-glucopyranoside) could enhance the activity of caspase-3 in human lymphoma U937 cells and induce their apoptosis [46]. The water extract of JHC can inhibit the growth of human esophageal squamous cell carcinoma cells (Eca109) in the

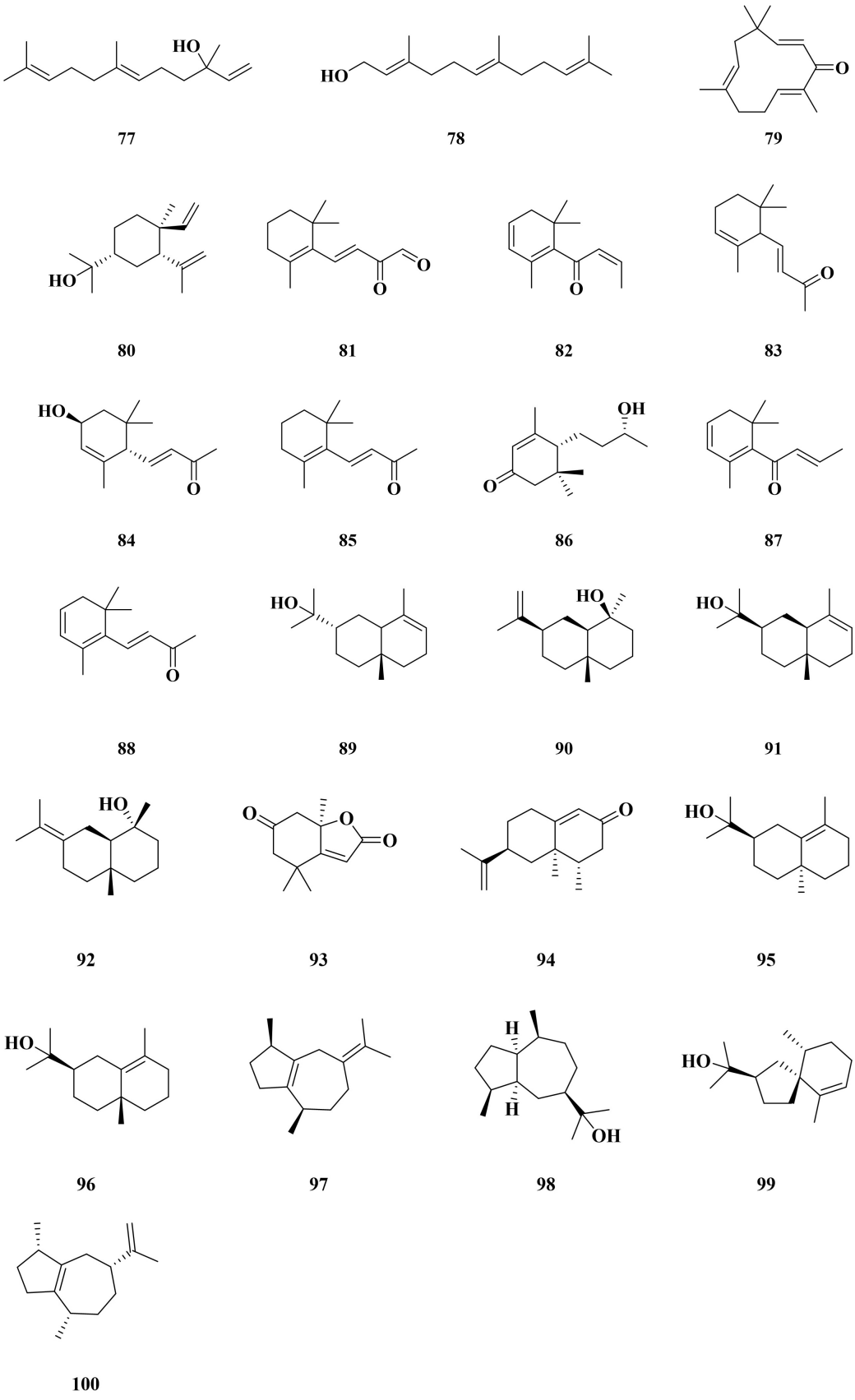
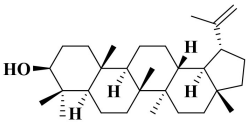


Fig. 7. Sesquiterpenes in JHC.



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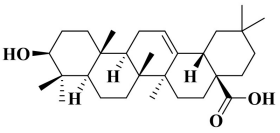

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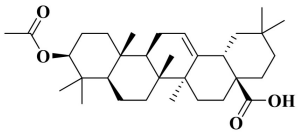

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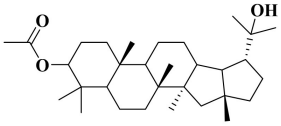

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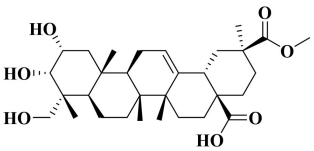

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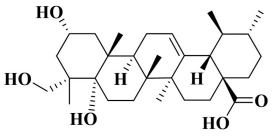

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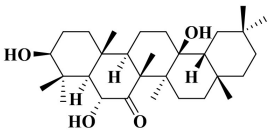

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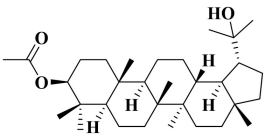

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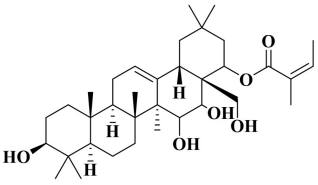

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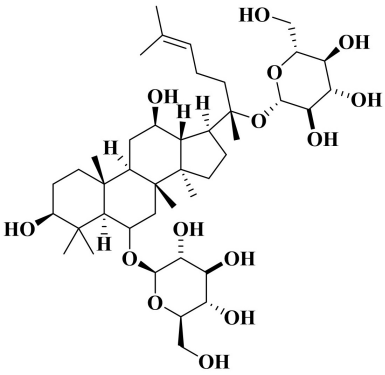

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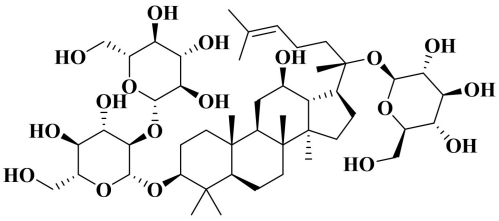

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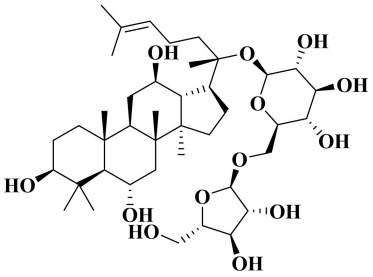

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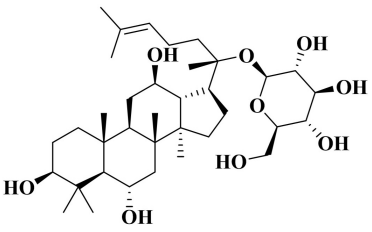

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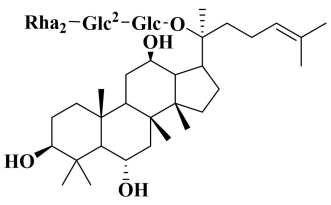

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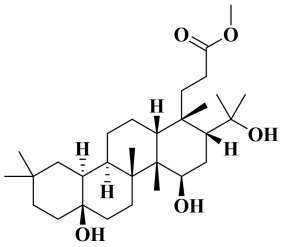

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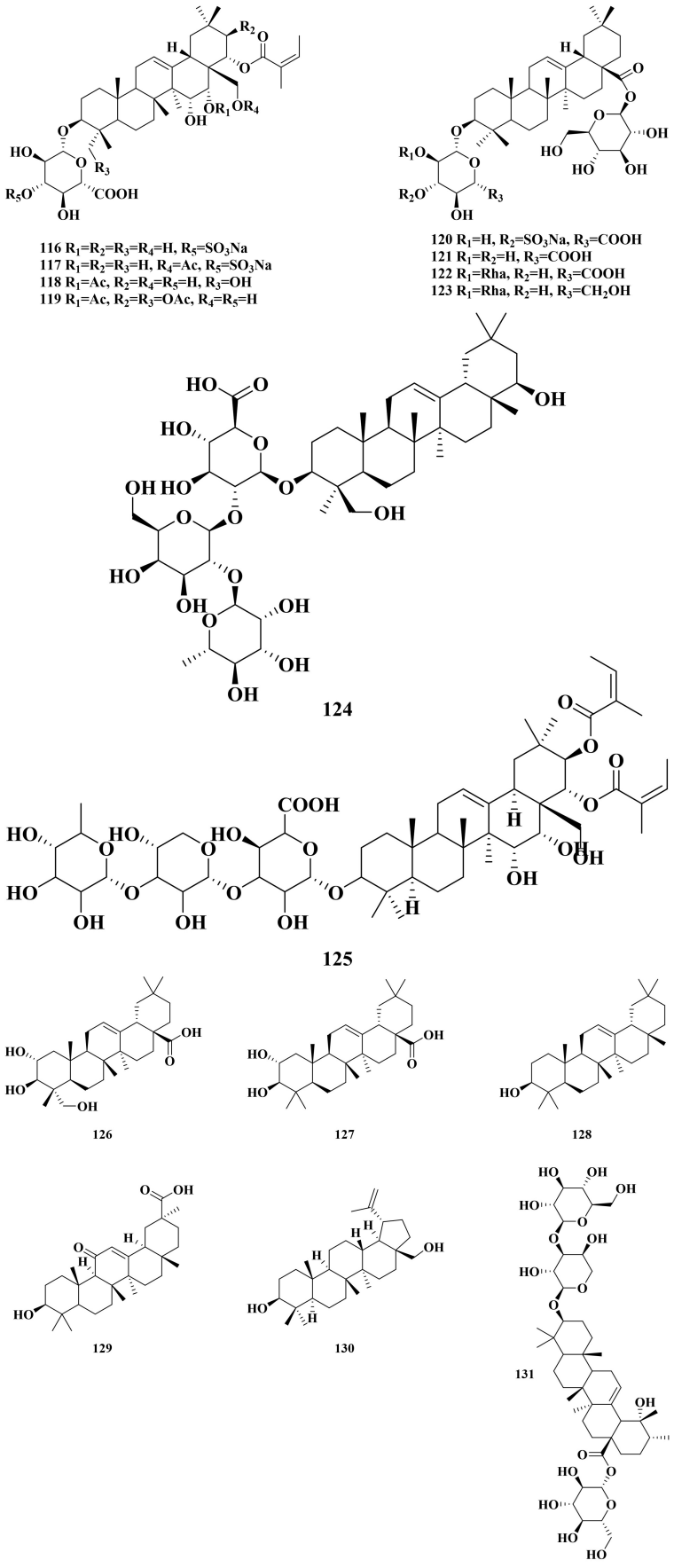

Fig. 8. Triterpenoids in JHC.

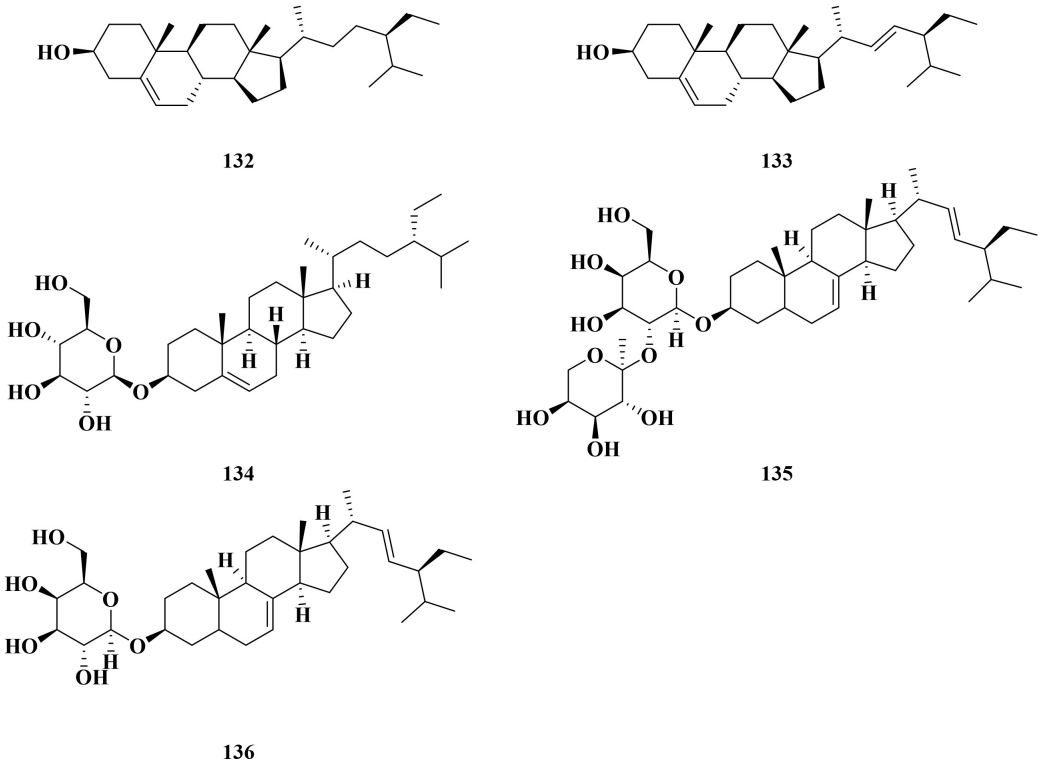

Fig. 9. Steroids in JHC.

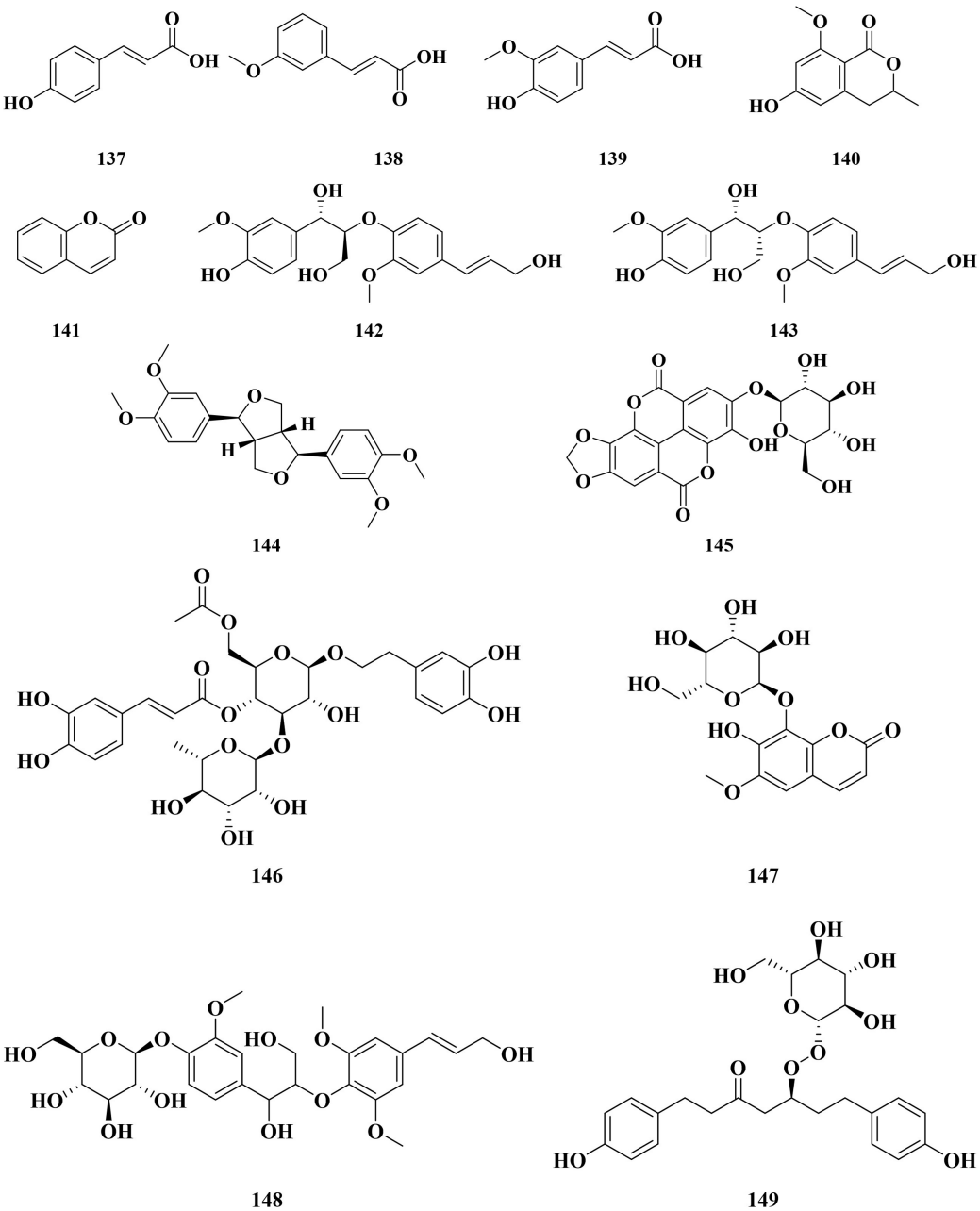

Fig. 10. Phenylpropanoids in JHC.

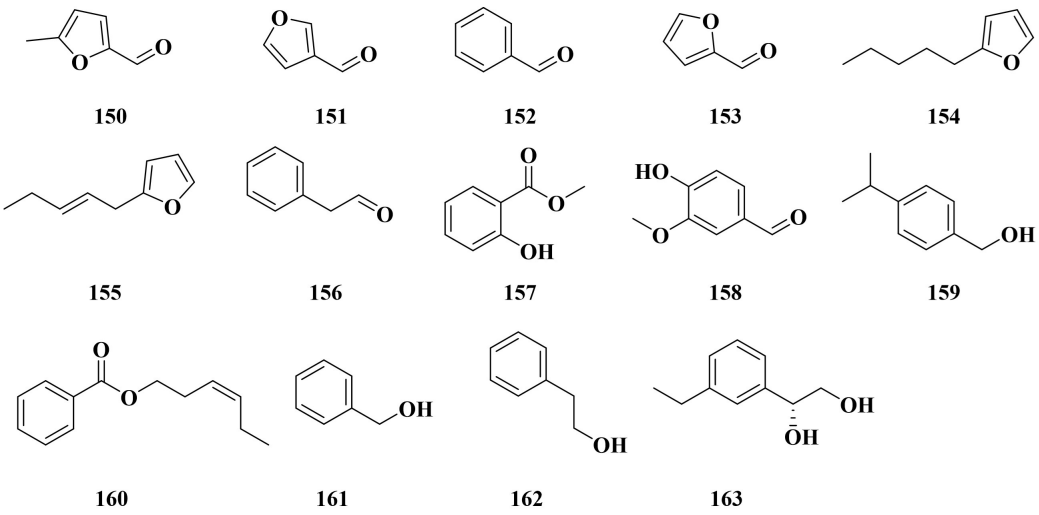

Fig. 11. Essential oils in JHC.

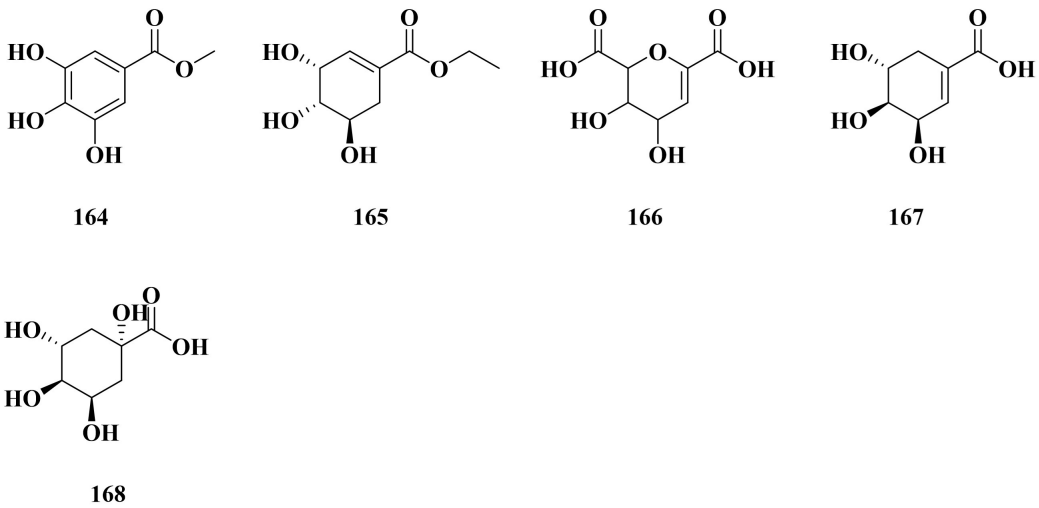

Fig. 12. Organic acid in JHC.

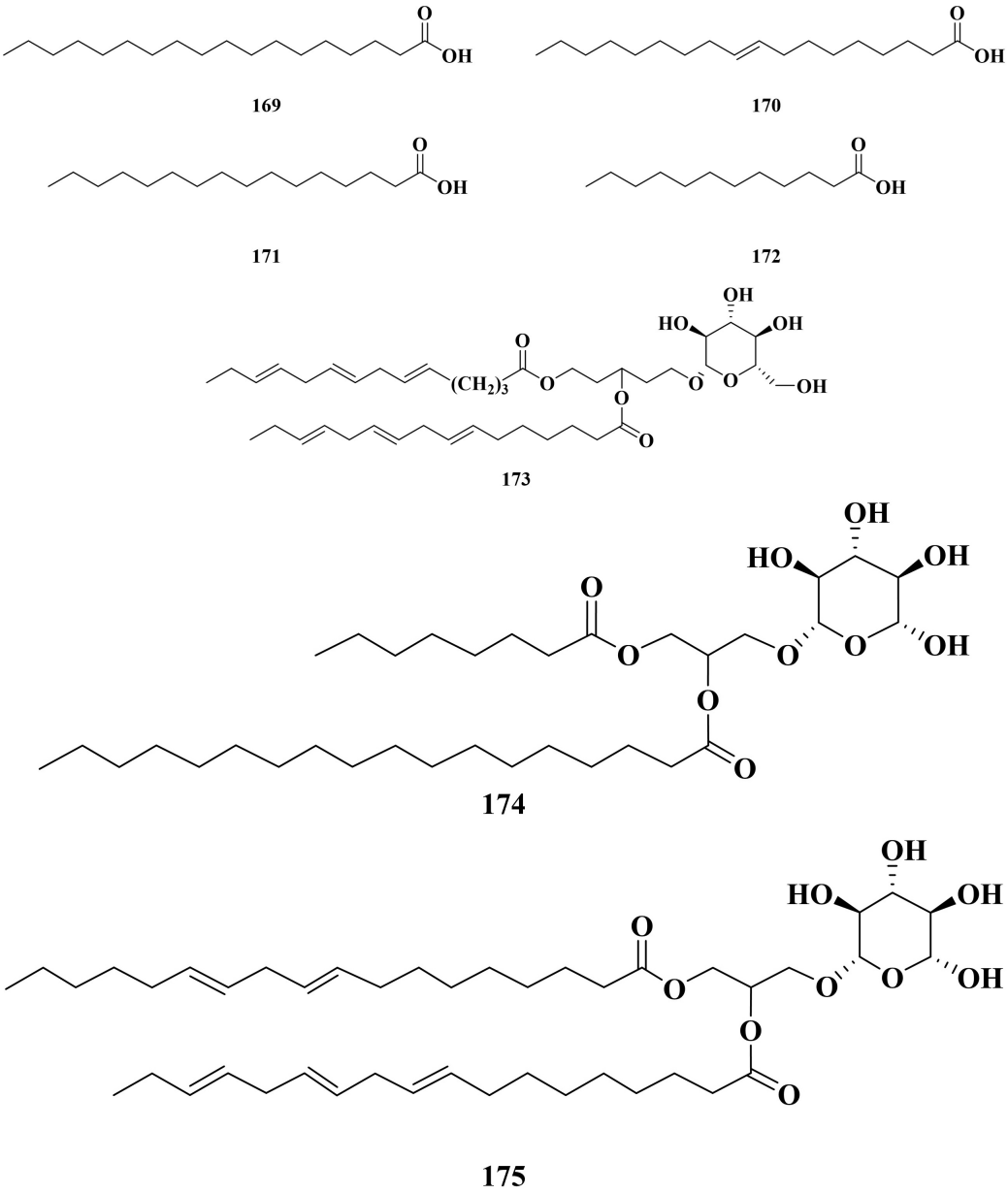

Fig. 13. Long-chain fatty acids in JHC.

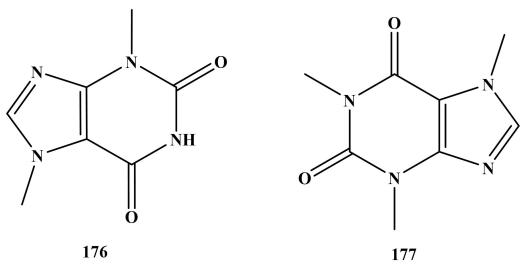

Fig. 14. Alkaloids in JHC.

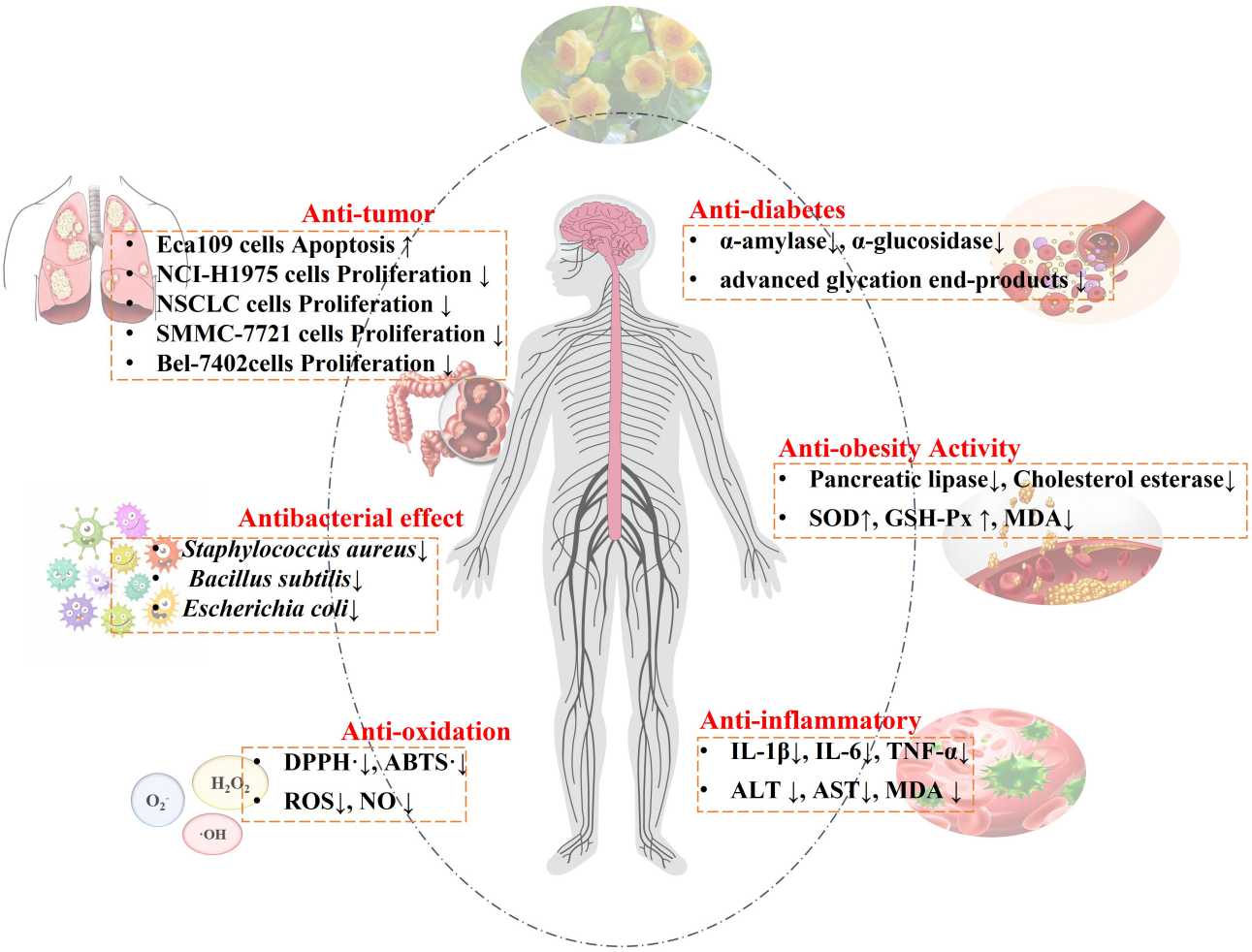

Fig. 15. The functions and index changes of JHC in the human body. It mainly works in the lungs, gastrointestinal tract and blood vessels.

Table 2. The summary of JHC active components - pharmacological effects - mechanisms - models.

Active ingredient	Pharmacological effects	Mechanism of action	Research model	References
Quercetin-7-O-(6″-O-E-caffeoyl)-β-D-glucopyranoside	Anti-Lymphoma	Enhances caspase-3 activity in human lymphoma U937 cells and induces apoptosis	Cell model (human lymphoma cell U937)	[16]
Camellia nitidissima Flower Water Extract	Anti-esophageal squamous cell carcinoma	Arrests Eca109 cells at G0/G1 phase and induces apoptosis	Cell model (human esophageal squamous cell carcinoma cell Eca109)	[17]
Camellia nitidissima Leaf n-Butanol Extract	Anti-Gastric Cancer	Induces cell autophagy and apoptosis; regulates the PI3K/Akt/mTOR signaling pathway; enhances the sensitivity of gastric cancer cells to paclitaxel	Cell model (human gastric cancer cells BGC-823, SGC-7901)	[18]
A1-barrieron-22a-angelate	Anti-EGFR-Mutant Lung Cancer	Induces apoptosis and inhibits cell proliferation	Cell model (National Cancer Institute-H1975 human lung adenocarcinoma cell line (NCI-H1975))	[19]
Polyphenols, Saponins	Anti-Non-Small Cell Lung Cancer (NSCLC)	Inhibits cytokines such as TGFB2 and INHBB, downregulates key genes such as PIK3R3 and ITGB8, downregulates NTRK and CACNA1D, and inhibits tumor proliferation and metastasis	Cell model (EGFR-mutant non-small cell lung cancer (NSCLC) cell lines NCI-H1975, HCC827 and EGFR wild-type NSCLC cell line A549), nude mouse subcutaneous xenograft model	[20]
Camellia nitidissima Leaf Water Extract/Ethanol Extract	Anti-Lung Cancer	Arrests the cell cycle at G0/G1 and S phases; increases intracellular ROS levels; upregulates the expression of pro-apoptotic proteins Bax and Caspase-3; regulates the expression of core proteins EGFR, SRC, and AKT	Cell model (lung cancer cells A549 (human lung adenocarcinoma) and SK-MES-1 (human lung squamous cell carcinoma))	[21]
Camellia nitidissima Leaf n-Butanol Extract/Water Extract	Anti-Colitis-Associated Colorectal Cancer	Enhances the activity of serum antioxidant enzymes (CAT, SOD); reduces the level of lipid peroxidation product MDA; regulates pathways such as glycolysis, amino acid metabolism, oxidative stress, and nucleic acid metabolism	Animal model (AOM/DSS-induced mouse colon cancer model)	[22]
Camellia nitidissima Ethanol Extract	Anti-Colorectal Cancer	Regulates apoptosis, cell cycle, and ferroptosis pathways; targets proteins GPX4, HMOX1, SLC7A11, FTH1, p53, and ACSL4	Cell model (human colorectal cancer cells HCT116, SW480, HCT15), nude mouse subcutaneous xenograft model	[23]
(3β,6α,12β)-3,6,12-Trihydroxydammar-24-en-20-yl 2-O-β-D-glucopyranosyl-(2$\rightarrow$1)-O-β-D-glucopyranosyl-(2$\rightarrow$1)-O-α-L-rhamnopyranoside	Anti-Hepatocellular Carcinoma	Inhibits the proliferation and migration of hepatocellular carcinoma cells	Cell model (human hepatocellular carcinoma cells Bel-7402, SMMC-7721)	[24]
Polysaccharides	Anti-Liver Cancer	Induces cell apoptosis (mainly early apoptosis); increases ROS levels; upregulates the expression of Bax and caspase-3; downregulates EGFR expression; inhibits cell proliferation and migration	Cell model (MHCC-97H, SMMC-7721 human hepatocellular carcinoma cell line)	[25]

Table 2. Continued.

Active ingredient	Pharmacological effects	Mechanism of action	Research model	References
Camellia nitidissima Flower Ethanol Extract	Hypolipidemic, Anti-obesity	Inhibits the activity of pancreatic lipase (PL) and cholesterol esterase (CEase); reduces cholesterol micelle solubility and cholesterol absorption; changes the conformation of pancreatic enzymes	Enzymatic experiment, animal model (high-fat diet rat model)	[26]
Camellia nitidissima Flower Water Extract, Ethanol Extract	Hypolipidemic, Anti-obesity	Increases the activity of SOD and GSH-Px, reduces serum MDA content; regulates lipid metabolism-related genes (fatty acid synthase, HMG-CoA reductase, etc.)	Cell model (HepG2 human hepatoblastoma cells), animal model (high-fat diet mouse model)	[27]
Leaf Essential Oil (mainly containing phytol, geraniol, n-hexanal)	Antioxidation	Scavenges DPPH and ABTS free radicals	*In vitro* antioxidant experiment	[28]
Gallic Acid, Catechin, Salicylic Acid, okicamelliaside	Antioxidation	Free radical scavenging and enzyme protection activities	*In vitro* activity screening (HPLC-UV-FLD post-column derivatization system)	[29]
3-cinnamoyltribuloside	Anti-inflammatory	Inhibits NO production and iNOS mRNA expression in LPS-activated RAW264.7 cells; downregulates inflammatory factors such as TNF-α, IL-1β, and IL-6	Cell model (RAW264.7 murine macrophage cell line)	[30]
Camellia nitidissima Leaf 10% Ethanol Extract	Liver protection via antioxidative stress and anti-inflammatory effects	Reduces the levels of ALT, AST, and MDA in serum and liver tissue; inhibits ROS production; blocks p65 protein phosphorylation; regulates the Nrf2 pathway and increases the levels of HO1, SOD, and GSH	Animal model (CCl_4-induced acute liver injury model in rats)	[31]
Camellia nitidissima Water Extract	Alcohol detoxification and liver protection	Enhances the activity of alcohol dehydrogenase and acetaldehyde dehydrogenase; improves liver oxidative stress and inflammatory status; regulates intestinal flora	Animal model (acute alcohol exposure model)	[32]
Camellia nitidissima Ethanol Extract	Anti-ulcerative colitis	Downregulates the TLR4/NF-κB signaling pathway (inhibits the expression of TLR4, p-NF-κB p65, and p-IκBα); reduces MPO activity and MDA levels; increases CAT and T-AOC activity; inhibits pro-inflammatory factors (NO, PGE2, IL-1β, etc.) and upregulates the anti-inflammatory factor IL-10	Animal model (DSS-induced acute ulcerative colitis model in mice)	[33]
Camellia nitidissima Leaf Essential Oil	Antibacterial activity (against Staphylococcus aureus, Bacillus subtilis and Escherichia coli)	Directly inhibits bacterial growth	*In vitro* antibacterial experiment	[28]

Table 2. Continued.

Active ingredient	Pharmacological effects	Mechanism of action	Research model	References
Gallic Acid, Catechin, Ellagic Acid, Chlorogenic Acid, Quercetin, Kaempferol	Anti-Pseudomonas aeruginosa	Inhibits the production of bacterial virulence factors and motility	*In vitro* antibacterial experiment	[34]
Methyl gallate	Anti-Aeromonas hydrophila	Inhibits the activity of bacterial hemolysin, protease, and lipase; weakens swimming ability and biofilm formation; regulates virulence-related genes such as ahyR and fleQ	*In vitro* antibacterial experiment	[35]
1,2,6-tri-O-galloyl-beta-d-glucose	Anti-Proteus penneri	Inhibits virulence factors such as protease and extracellular polysaccharide (EPS); regulates genes such as hfq and flhD and destroys the cell membrane structure	*In vitro* antibacterial experiment	[36]
Camellia nitidissima Leaf Water Extract	Antidepressant	Regulates the hypothalamic-pituitary-adrenal (HPA) axis and monoaminergic nervous system; inhibits the mitochondrial-mediated apoptotic pathway; activates the PKA-CREB-BDNF signaling pathway	Cell model (corticosterone-induced neuronal injury model)	[37]
Camellia nitidissima Leaf Ethanol Extract	Antidepressant	Promotes hippocampal neurogenesis; regulates HPA axis function; promotes serotonin reuptake; activates the Akt/GSK3β/CREB signaling pathway	Animal model (depression mouse model)	[38]
Phenolic Substances/Phenolic Compounds	Antidiabetic (improving complications)	Scavenges methylglyoxal and inhibits the formation of advanced glycation end products (AGEs)	*In vitro* biochemical experiment	[39]
3β-O-α-L-rhamnopyranosyl-(1$\rightarrow$3)-α-D-xylopyranosyl-(1$\rightarrow$3)-β-D-glucopyranoside (**124**), CampetelosidesA-E (**116–120**)	Antidiabetic	Inhibits α-glucosidase activity	*In vitro* enzyme activity experiment	[40]
Camellia nitidissima Flower Ethanol Extract	Antidiabetic	Inhibits α-amylase and α-glucosidase activity	*In vitro* enzyme activity experiment	[41,42]
Luteolin	Improving autism	Alleviates endoplasmic reticulum stress and mitochondrial dysfunction	Animal model (rodent autism model)	[43]
Ellagic Acid	Liver protection	Improves diabetes-induced liver injury and enhances liver function	Animal model (diabetic rat model)	[44]
Rutin	Skeletal muscle protection	Alleviates cisplatin-induced oxidative damage to skeletal muscle	Animal model (cisplatin-treated rat model)	[45]

G0/G1 phase and induce apoptosis of these cells [18]. The n-Butanol extract of JHC (JHC-4) was found to significantly inhibit the proliferation of human gastric cancer cells and induce autophagy. As an autophagy agonist, JHC-4 could synergically increase the sensitivity of gastric cancer cells to paclitaxel, while significantly enhancing the growth inhibitory effect of paclitaxel by inducing autophagy and apoptosis. The synergistic anti-proliferative effect of JHC-4 and paclitaxel was related to the PI3K/Akt/mTOR signaling pathway [21]. Hou *et al*. [47] analyzed the components of JHC and screened their anti-tumor activity. They identified a new oleane-type triterpene, A1-barrigenol22a-angelate, that has strong anti-tumor activity and can induce apoptosis and significantly inhibit the proliferation of the EGFR-mutant lung cancer cell line NCI-H1975. Wang *et al*. [48] found that polyphenols and saponins present in JHC have a significant inhibitory effect on non-small cell lung cancer (NSCLC) cells through cytokine-cytokine receptor interaction and the PI3K-Akt and MAPK signaling pathways. The key targets through which JHC exerted its anti-tumor effects *in vitro* were TGFB2 (Transforming Growth Factor Beta 2), INHBB (Inhibin Subunit Beta B), PIK3R3 (Phosphatidylinositol-3-Kinase Regulatory Subunit 3), ITGB8 (Integrin Subunit Beta 8), TrkB (Tropomyosin Receptor Kinase B), and CACNA1D (Calcium Voltage-Gated Channel Subunit Alpha1 D).

Zhou *et al*. [22] reported that JHC can cause G0/G1 and S phase cell cycle arrest, increase intracellular ROS levels, promote the expression of apoptotic proteins Bax and Caspase-3, and simultaneously regulate the expression of core proteins EGFR (Epidermal Growth Factor Receptor), SRC (Proto-Oncogene Tyrosine-Protein Kinase Src), and AKT, thereby inhibiting lung cancer cells by inducing apoptosis. Li *et al*. [23] confirmed that the n-butanol and water extracts of JHC could significantly reduce the incidence of colon cancer in a mouse model of colitis generated by the injection of azomethane oxide (AOM) and sodium dextrin sulfate (DSS). Studies have confirmed that JHC mainly inhibits the development of colon cancer by increasing the activities of antioxidant enzymes (CAT (Catalase), SOD (Superoxide Dismutase)) in serum, reducing the level of lipid peroxidation product (MDA (Malondialdehyde)), and regulating multiple pathways such as glycolysis, amino acid metabolism, oxidative stress, and nucleic acid metabolism.

Chen *et al*. [25] found that a JHC extract regulates multiple pathways in colon cancer cells, including apoptosis, cell cycle, and ferroptosis. This extract exerts its anti-colon cancer effect by regulating the expression of GPX4 (Glutathione Peroxidase 4), HO1 (Heme Oxygenase 1), SLC7A11 (Solute Carrier Family 7 Member 11), FTH1 (Ferritin Heavy Chain 1), p53, and ACSL4 (Acyl-CoA Synthetase Long Chain Family Member 4) proteins. Xu *et al*. [41] reported that (3β,6α,12β)-3,6,12-trihydroxydammar-24-en-20-yl-2-O-β-D-glucopyranosyl-(2$\rightarrow$1)-O-β-D-glucopyranosyl-(2$\rightarrow$1)-O-α-L-rhamnopyranoside from JHC could inhibit the proliferation and migration of the human hepatoma cell lines Bel-7402 and SMMC-7721. Zhou *et al*. [26] found that JHC polysaccharides can promote apoptosis (especially early apoptosis), increase the ROS level in hepatoma cells (MHCC-97H and SMMC-7721), up-regulate the expression of Bax and caspase-3, and down-regulate EGFR expression to inhibit the proliferation and migration of these cells.

At present, only a minority of research on the anti-tumor activity of JHC has employed animal models, with most experimental studies being at the *in vitro* cellular stage. Further animal experiments are needed to confirm the reliability of JHC anti-tumor activity.

4.2 Anti-Obesity

Published studies have shown that a JHC extract (CNEF) can lower blood lipids by inhibiting the activities of pancreatic lipase (PL) and cholesterol esterase (CEase). Moreover, water extract (AEC) and ethanol extract (EEC) of the flower can reduce blood lipids by increasing the activities of SOD and GSH-Px(Glutathione Peroxidase), and reducing the level of serum MDA. CNEF inhibited the activities of PL and CEase by simultaneously binding to free enzymes and enzyme-substrate complexes, with the inhibition of PL and CEase being dose-dependent. The median inhibitory concentrations (IC_{50}) of CNEF for PL and CEase were 320 µg/mL and 200 µg/mL, respectively, with the inhibitory effect of CEase being stronger than that of PL. Moreover, CNEF reduced cholesterol absorption by lowering the solubility of artificially prepared cholesterol micelles in a dose-dependent fashion, thereby reducing blood lipids and improving obesity [27]. After treatment of PL with CNEF, the content of α-helix decreased, β-folding and β-turning increased, and the fluorescence intensity decreased, indicating changes in the conformation of pancreatic enzymes [49].

Animal experiments showed that adding CNEF to a high-fat feed can significantly reduce the food intake of rats in a dose-dependent manner. This indicates that CNEF delays the digestive process by inhibiting the activities of PL and CEase, thereby reducing the body's food requirements. Compared with the high-fat feed group, fecal excretion of total triglycerides (TG) and total cholesterol (TC) in the high-dose CNEF group increased significantly by 37 $\pm$ 6% and 92 $\pm$ 12%, respectively ($p \leq 0.01$). The increase in TC excretion was significantly higher than that of TG, supporting the conclusion that the inhibitory effect of CNEF was stronger on CEase than on PL [27]. A high-fat diet has been shown to induce the production of free radicals, leading to hypercholesterolemia and oxidative stress. SOD and GSH-Px are two important antioxidant enzymes in the human body. These help to reduce the production of ROS and prevent lipid peroxidation and metabolic intermediates from disrupting normal functions of the body. The occurrence of

hyperlipidemia is related to the production of MDA. Low- and high-doses of AEC, as well as high-dose EEC, were found to significantly increase SOD activity in mice fed a high-fat diet. Low-dose AEC, and low- and high-doses of EEC were found to significantly increase GSH-Px activity. High-dose AEC and low- and high-dose EEC significantly reduced the accumulation of serum MDA. Moreover, compared with the lovastatin group, AEC and EEC treatment in mice fed a high-fat diet had no significant effects on SOD and GSH-Px activities, nor on the level of serum MDA [50].

AEC and EEC showed significant differences in lipid-lowering capabilities in cell and animal experiments. Cell experiments showed that AEC has greater potential to prevent fatty liver than EEC. AEC and EEC (both 100 µg/mL) significantly reduced lipid accumulation and TG levels in oleic acid-induced HepG2 cells (Human Hepatocellular Carcinoma G2 cell line), with a stronger effect by AEC. AEC (100 µg/mL) was found to down-regulate the mRNA levels of fatty acid synthase, 3-hydroxy-3-methylglutaryl-coenzyme A reductase, and glycerol-3-phosphoacyltransferase in HepG2 cells, whereas EEC (150 µg/mL) only down-regulated the mRNA level of 3-hydroxy-3-methylglutaryl-coenzyme A reductase [29]. Animal experiments showed that high-dose AEC, low- and high-dose EEC, and lovastatin significantly reduced the serum levels of TC, TG, and LDL-C, while increasing serum HDL-C. At the same dose, EEC had a stronger lipid-lowering effect than AEC [50]. The above results indicate that further studies are needed to determine which polar parts or chemical components in JHC have greater lipid-lowering capabilities.

4.3 Anti-Inflammatory and Anti-Oxidant Effects

The antioxidant activity of essential oil extracted from JHC leaves was evaluated by Ge *et al*. [24] through DPPH and ABTS free-radical scavenging experiments. The antioxidant capacity of JHC essential oil, mainly composed of phytolol (58%), geraniol (5.6%), and n-hexanal (3.3%), was significantly stronger than the essential oil, which is mainly composed of linalool (35.8%), phytolol (7.9%), geraniol (7.3%), and methyl salicylate (6.8%). This was the first report on the antioxidant activity of JHC. Wang *et al*. [51] found that the ethanol extract exhibited strong antioxidant activity close to that of vitamin C due to its rich polyphenol content, which was significantly superior to that of the essential oils. Tian *et al*. [16] found that the JHC ethanol extract exhibited DPPH and ABTS radical scavenging rates of 84.61% and 95.51%. Currently, the screening of active ingredients in natural products is often time-consuming and labor-intensive. Cheng *et al*. [30] established an efficient screening method for active ingredients that integrates molecular network multi-data processing technology, chromatographic fingerprinting, and the evaluation of efficacy. The core antioxidant components of JHC (gallic acid, catechins, and salicylic acid) were screened using an HPLC-UV-FLD post-column derivatization system. Based on their structural similarity, seven potentially active components related to active markers were rapidly identified by Molecular Networking analysis. The free-radical scavenging and enzyme protective activities of the novel antioxidant component okicamelliaside were discovered and confirmed for the first time, demonstrating a successful example of the discovery of trace components and offering new ideas for the research and development of active natural products. Wang *et al*. [31] discovered that 3-cinnamoyltribuloside (3-CT) extracted from JHC could inhibit the production of nitric oxide, as well as the mRNA expression of its key synthetase (iNOS) in LPS-activated RAW 264.7 cells. In addition, through mRNA detection and ELISA analysis, these authors confirmed that 3-CT could inhibit the production of inflammatory factors such as TNF-α, IL-1β, and IL-6.

JHC extract can protect the liver and treat colitis through its antioxidant and anti-inflammatory effects. Zhang *et al*. [32] found that an extract from JHC leaves could reduce the levels of ALT, AST, and MDA in serum and liver tissue, reduce histopathological damage, inhibit the generation of ROS, and reduce levels of the inflammatory factors TNF-α, IL-1β, and IL-6. Moreover, it could dose-dependently block the phosphorylation of p65 protein, regulate the Nrf2 pathway, and increase the levels of HO1, SOD, and GSH in rats with acute liver injury induced by CCl_4, thereby exerting a protective effect on the liver. Cheng *et al*. [33] confirmed that JHC extract could significantly increase the activity of alcohol dehydrogenase or aldehyde dehydrogenase, improve the oxidative stress and inflammatory states of the liver, regulate intestinal flora, and protect against acute alcohol exposure. Li *et al*. [34] found that JHC extract could significantly reduce weight loss in mice with DSS-induced acute ulcerative colitis, maintain colon length, and lower the DAI(Disease Activity Index) score. The effect of high-dose JHC extract was comparable to that of mesalazine. Moreover, the JHC extract could reduce inflammatory cell infiltration and colonic mucosal damage, improve the histopathological score, reduce MPO (Myeloperoxidase) activity and the level of MDA, increase the activities of CAT and T-AOC (Total Antioxidant Capacity), alleviate oxidative damage, inhibit pro-inflammatory factors (NO, PGE2 (Prostaglandin E2), IL-1β, IL-6, TNF-α), and increase the level of the anti-inflammatory factor IL-10. The responsible mechanism involved down-regulating the protein expression of TLR4, p-NF-κB p65, and p-IκBα, and inhibiting the TLR4/NF-κB signaling pathway.

4.4 Anti-Bacterial Effects

Studies have shown that JHC essential oil has inhibitory effects on *Staphylococcus aureus*, *Bacillus subtilis*, and *Escherichia coli*. The dichloromethane extract component (DF) of JH can also significantly inhibit Pseu-

domonas aeruginosa. Six active components detected by DF, namely gallic acid, catechin, ellagic acid, chlorogenic acid, quercetin, and kaempferol, could all inhibit the production and motility of Pseudomonas aeruginosa, with ellagic acid having the strongest effect [24,36]. Jiang *et al*. [37,52] isolated methyl gallate and 1,2,6-tri-O-galloyl-beta-d-glucose from JHC. Methyl gallate could effectively inhibit the hemolytic, protease, and lipase activities of *Aeromonas hydrophila* (SHAe 115), weaken its ability to swim and form biofilms, down-regulate positive regulatory genes (ahyR, fleQ), and up-regulate negative regulatory genes (litR, fleN). 1,2,6-tri-O-galloyl-beta-d-glucose inhibited the production of key virulence factors for *Proteus penneri* (ALK 1200), such as protease and EPS. The cell membrane structure was affected by down-regulating hfq and flhD, and up-regulating bssS, thereby reducing its pathogenicity.Notably, the compound isolated from JHC by Wang *et al*. [53] did not exhibit significant inhibitory effects on pyocyanin synthesis regulated by quorum sensing in Pseudomonas aeruginosa PAO1.

4.5 Anti-Depressant Effects

He *et al*. [54] reported that the antidepressant activity of the water extract of JHC might be due to the regulation of the hypothalamic-pituitary-adrenal axis and the monoaminergic nervous system. Moreover, they confirmed that JHC extract could effectively antagonize corticosterone-induced neuronal damage. The protective mechanism may involve inhibition of the mitochondrial-mediated apoptotic pathway and activation of the PKA-CREB-BDNF signaling pathway [38]. Tsoi *et al*. [39] confirmed that JHC extract can significantly improve depressive behaviors in mice by promoting hippocampal neurogenesis. The mechanism of action involves regulating the function of the hypothalamic-pituitary-adrenal axis, promoting serotonin reuptake, and activating the Akt/GSK3β/CREB signaling pathway.

4.6 Anti-Diabetes Effects

The therapeutic effect of JHC on diabetes is mainly achieved by inhibiting the formation of advanced glycation end products (AGE) and suppressing the activities of α-amylase and α-glucosidase. Phenolic substances in JHC can inhibit the formation of AGE by eliminating methylglyoxal. AGE is known to have significant effects on diabetic complications, Alzheimer's disease, aging, and atherosclerosis [40,55]. Six new saponins (**128**, **120–124**) were shown to inhibit α-glucosidase [42,56]. Zhang *et al*. [43] reported that JHC extract could inhibit the activities of α-amylase and α-glucosidase, with the best inhibitory effect achieved by administering before meals.

4.7 Other Effects

Recent studies have demonstrated that luteolin derived from JHC alleviates endoplasmic reticulum stress and mitochondrial dysfunction in rodent models of autism [44]. Ellagic acid has been shown to improve hepatic function in male rats and to mitigate diabetes-induced liver injury [45]. Additionally, Rutin exhibits protective effects against cisplatin-induced oxidative damage in the skeletal muscle tissue of rats [57].

5. Toxicity and Safety Evaluation

The safety of the water extract of JHC was systematically evaluated through a series of toxicological tests including acute toxicity, genotoxicity, and subchronic toxicity. The acute oral LD_{50} (Lethal Dose 50) values of both rats and mice were greater than 10.0 g/kg·bw, indicating that the water extract of JHC belongs to the actually non-toxic grade. Through the Ames test, the micronucleus test of polychromatic erythrocytes in mouse bone marrow and the *in vitro* chromosomal aberration test of mammalian cells, it was proved that this extract has no potential mutagenic effect. In the 90-day oral toxicity test of rats, the growth and development of animals in each group were good. The no-observed-adverse-effect level (NOAEL) of this extract for SD rats was 50 g/kg·bw, reaching 150 times the recommended daily intake for adults. Based on the actual tea consumption of the population (5 g per person per day), it is estimated that the maximum ineffective dose can reach 600 times the actual daily intake of the population [58].

6. Conclusions and Future Perspectives

This study systematically summarized the chemical components and pharmacological activities of JHC. The main chemical components of JHC are flavonoids, terpenoids (including monoterpenoids, sesquiterpenoids, and triterpenoids), steroidal saponins, and phenylpropanes. Among these, the flavonoids, triterpenoids, and saponins form the material basis for the main pharmacological activities. Flavonoid components mediate significant anti-tumor activity by activating the Caspase-3/Bax apoptotic pathway and inhibiting the PI3K/Akt/mTOR signaling axis. Triterpene saponins promote reverse cholesterol transport by activating liver X receptor α (LXRα), thereby reducing the levels of serumTC and TG. Steroidal saponin components effectively regulate glucose metabolism by promoting the translocation/transport of glucose transporter 4 (GLUT4) and activating peroxisome proliferator-activated receptor γ (PPARγ). The above-mentioned pharmacological activities are highly consistent with the efficacy of *Camellia petelotii* recorded in traditional Chinese medical classics for the prevention and treatment of pharyngitis, nephritis, dysentery, tumors, hematochezia, hypertension, and menstrual disorders, etc. This verifies its traditional application value from a modern pharmacological perspective.

However, developing JHC into a standardized herbal product still faces two core challenges: insufficient pharmacokinetic (ADME) research and unclear quality markers. Firstly, most of the existing research focuses on *in vitro* activity and mechanism, while *in vivo* pharmacokinetic (ab-

sorption, distribution, metabolism, excretion) data are seriously lacking. In the future, a combination of metabolomics and chemical analysis should be employed to systematically track the dynamic changes of multiple components within organisms. By using animal disease models (e.g., hyperlipidemia and diabetes models), the bioavailability, tissue distribution and metabolic pathways of their active ingredients are comprehensively evaluated. Computerized molecular docking and *in vitro* enzymatic experiments were adopted to predict and verify the interactions between key components and drug-metabolizing enzymes (e.g., CYP450), and to assess the potential risks of drug-drug interactions. Secondly, the chemical composition of JHC is complex and is significantly affected by factors such as the growth environment and harvesting and processing, making it difficult to control the uniformity of product quality. To this end, it is necessary to establish and integrate a multi-dimensional quality control system: apply omics technologies (e.g., metabolomics) to construct a digital fingerprint that comprehensively reflects the chemical composition spectrum of its small molecules. And by integrating chemometrics methods, identify the characteristic component groups related to key pharmacological activities and set reasonable quantitative standards to ensure the consistency between product batches and the reproducibility of clinical efficacy.

As a distinctive "Food and Medicine Homology" plant resource, JHC has great potential for further development. Future research should focus on the following directions to extend the industrial chain and promote sustainable utilization. (1) Targeted functional product development: based on the clearly identified active ingredients and their mechanisms, develop foods or drugs with specific functions, e.g., anti-tumor, lipid-regulating, and hypoglycemic. (2) Further explore the active components of macromolecules: focus on the relatively weak links in current research, such as the structural identification of macromolecular substances (e.g., polysaccharides and polypeptides), clarify their structure-activity relationships, and analyze in detail the mechanisms and key targets of their immune regulation and inhibition of angiotensin-converting enzyme (ACE). (3) Expand the pharmacological verification of traditional effects: systematically study the modern pharmacological basis of other effects recorded in ancient texts (e.g., lowering of blood pressure) and explore potential new pharmacological effects. (4) Evaluation of system safety: establish a complete toxicological assessment system, covering acute toxicity (LD_{50}), 30-day feeding (subchronic) toxicity with a focus on monitoring changes in liver and kidney function indicators, and interference assessment of drug-metabolizing enzymes (e.g., CYP450), to ensure the safety of clinical application. On this basis, a sustainable resource development and utilization model that integrates "targeted cultivation of active ingredients - development of high-quality functional products - ecological protection of rare resources" can be constructed by integrating and applying the quality control technology of chemical fingerprinting.

In conclusion, JHC possesses significant scientific, economic, medicinal, and ornamental value. However, due to its limited distribution, habitat degradation, and persistent illegal harvesting, the wild population of JHC has declined sharply. Therefore, its development and utilization must adhere to the principles of sustainable development. While promoting resource utilization, it is essential to strengthen relevant legal frameworks, enforce strict penalties against illegal collection, and enhance quality control systems to ensure the traceability and legality of raw materials. Furthermore, conservation efforts should be intensified through the expansion of protected areas and the improvement of nature reserve management to maintain the stability and integrity of wild populations. A transition from reliance on wild harvesting to large-scale artificial cultivation should be facilitated through technological innovation. The establishment of standardized cultivation bases and high-quality germplasm resource banks is critical to ensuring a sustainable and reliable supply of JHC, thereby enabling the long-term conservation and responsible use of this valuable medicinal resource.

Author Contributions

DL: Writing-original draft, Software, Investigation, Formal analysis. YLi: Writing-review & editing, Validation, Investigation, Conceptualization. YLiu: Data curation, Investigation, Software. XWu: Investigation, Formal analysis. YLu: Investigation, Formal analysis. RZ: Validation, Resources. JT: Validation, Resources. XWei: Validation, Investigation, Conceptualization, Funding acquisition. CZ: Writing-review & editing, Software, Resources, Funding acquisition. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/IJP47944.

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