REVIEW ARTICLES

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Review of the Composition and Antimicrobial Roles of Extracts of Vine Tea (*Ampelopsis grossedentata*)

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Vine tea (*Ampelopsis grossedentata*) is a traditional medicinal and edible plant widely consumed in China. Its bioactivity is attributed to flavonoids, particularly dihydromyricetin, which exhibits broad-spectrum antimicrobial activities. This review provides a comprehensive narrative synthesis of the antimicrobial properties of dihydromyricetin from vine tea against pathogenic bacteria and fungi, underlying mechanisms of action, and potential applications. Current evidence indicates that dihydromyricetin exerts antimicrobial effects through multiple mechanisms, including disruption of cell wall integrity, alteration of membrane permeability, interference with lipid and energy metabolism, inhibition of protein synthesis, and suppression of virulence-associated processes. Notably, its antibacterial activity is generally more pronounced against gram-positive bacteria than gram-negative species, while antifungal efficacy is comparatively weaker but can be significantly enhanced through advanced formulation strategies, including co-crystallization and nanoparticle-based delivery systems. Moreover, dihydromyricetin demonstrates a multi-target, multi-pathway mode of action, involving key metabolic and signaling pathways related to oxidative stress, inflammation, and cellular homeostasis. Despite its promising antimicrobial potential, current research is limited by methodological inconsistencies, including reliance on inhibition-zone assays, insufficient reporting of minimum inhibitory concentrations, and lack of standardized experimental frameworks. Furthermore, data on toxicity, bioavailability, and real-world application remain inadequate. Overall, dihydromyricetin represents a promising natural antimicrobial agent with potential applications in food preservation, agriculture, and pharmaceuticals. Future research should prioritize standardized evaluation methods, comprehensive toxicological assessment, and the development of effective delivery systems to facilitate its translation from laboratory studies to practical applications. This article aims to review the composition and antimicrobial roles of extracts of vine tea (*A. grossedentata*).

Keywords: Flavonoids • Pharmaceutical Research • Antimicrobial Activity

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Introduction

Vine tea (*Ampelopsis grossedentata*) primarily grows in hillside shrubs and forested regions of southern, southwestern, and northeastern China, as shown in **Figure 1** [1,2]. Taxonomically, vine tea belongs to the family Vitaceae (Hand.-Mazz.) [3,4]. Morphologically, vine tea is a perennial plant characterized by climbing stems with longitudinal ribs, glabrous surfaces, and swollen nodes. Its tendrils are bifurcated and positioned 2 internodes apart, opposite the leaves. The plant possesses thin, fibrous, slightly curved roots; bipinnate leaves with petioles measuring 1.5 to 3.0 cm; and caducous stipules. The apical leaflets have petioles, whereas the lateral leaflets are sessile [5,6]. Both sides of the leaves are smooth and glabrous. Cymes arise from the leaf axils or branch apices opposite the leaves. The calyx is discoid, approximately 2.2 mm in diameter, and the flowers contain 5 oblong petals, 5 stamens, and a shallow, cup-shaped floral disc (**Figure 2**) [7]. The fruit is a nearly spherical, purple-black berry measuring 3 to 6 mm in diameter. Flowering occurs from June to September, and fruiting continues from July to November [8].

The concept of "food and medicine homology," rooted in traditional Chinese medicine, emphasizes the intrinsic link between food and medicine, proposing that certain foods possess nutritional and therapeutic functions. This concept recognizes that some dietary components contribute not only to nourishment but also to health promotion, disease prevention, and treatment

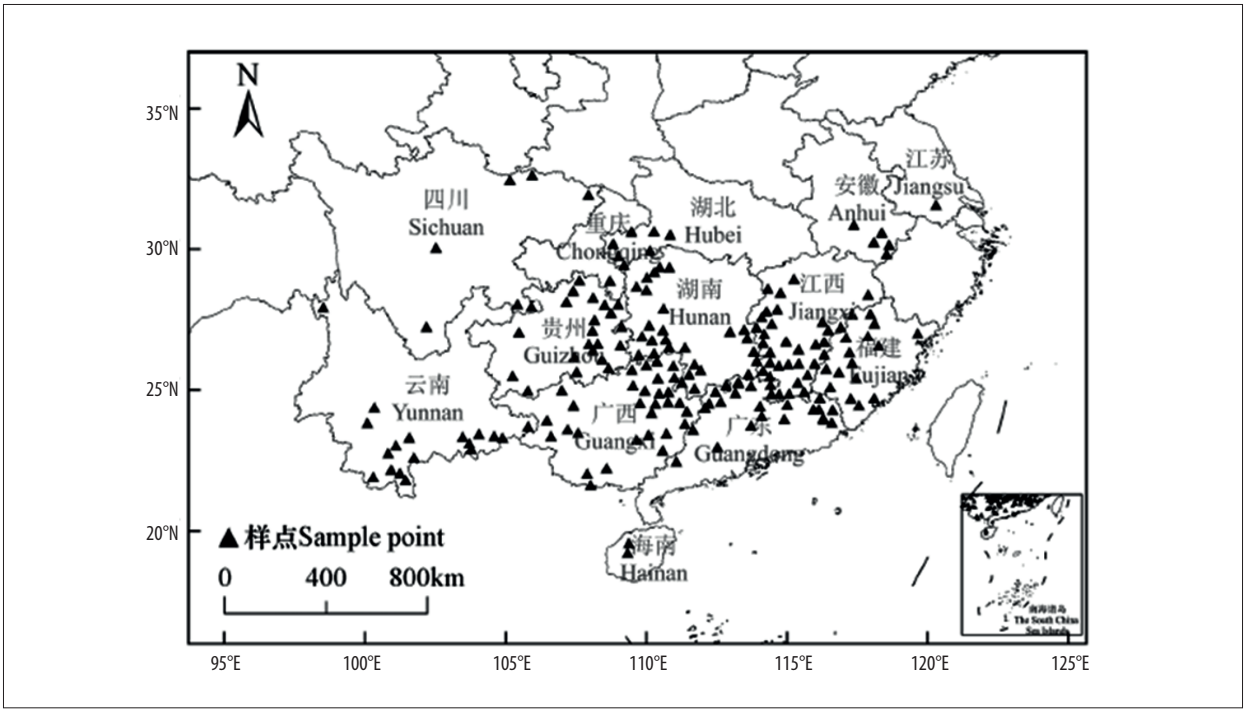

Figure 1. Distribution of vine tea (*A. grossedentata*) in China.

Figure 2. Morphological identification of vine tea (*A. grossedentata*): (**A**) leaves and stems; (**B**) flowers; and (**C**) root.

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[9,10]. Vine tea has been officially recognized in China as a functional herbal beverage [11,12]. It is typically processed using methods similar to those employed in green tea production, including withering, steaming, and drying [13], and its medicinal use can be traced back over a century [14,15]. This species exhibits notable medicinal properties and has been widely utilized in traditional Chinese medicine [16,17]. Traditionally, vine tea has been used to treat various conditions, including toothache, aphonia, and aphtha [18]. It has also been applied to clear heat and toxins, reduce inflammation, relieve sore throat, prevent heatstroke, lower blood pressure and lipid levels, and alleviate fatigue, functions recognized by the National Health and Family Planning Commission of China [19]. These therapeutic effects are largely attributed to its diverse secondary metabolites, including phenols, flavonoids, terpenes, steroids, and volatile compounds [20]. Among these constituents, dihydromyricetin (also known as ampelopsin) is the predominant flavonoid, accounting for approximately 25% to 45% of total flavonoids (approximately 326.8 mg/g dry weight) [21-23]. This high flavonoid content contributes significantly to the plant's antimicrobial properties [24]. Xiao et al [25] demonstrated that dihydromyricetin effectively inhibits several foodborne pathogens, including *Staphylococcus aureus, Bacillus subtilis*, *Pseudomonas aeruginosa*, *Escherichia coli,* and *Salmonella paratyphi*, at concentrations ranging from 0.3 to 2.5 mg/mL.

The antimicrobial mechanisms of flavonoids involve multiple cellular targets, including inhibition synthesis of nucleic acid, disruption of cytoplasmic membrane function, suppression of energy metabolism, prevention of microbial adhesion and biofilm formation, blockage of membrane porins, alteration of membrane permeability, and attenuation of pathogenicity [26,27]. Despite growing interest in natural product research, comprehensive reviews focusing on the biological control potential of vine tea, particularly its antimicrobial mechanisms and secondary metabolite profiles, remain limited. With increasing concerns regarding antimicrobial resistance and the environmental impact of synthetic chemicals, plant-derived antimicrobial agents are receiving heightened attention. Therefore, this review synthesizes current evidence demonstrating that dihydromyricetin is a promising natural antimicrobial flavonoid, with emphasis on its chemical composition, mechanisms of action, and potential applications in agriculture, pharmaceuticals, and food preservation. This article aims to review the composition and antimicrobial roles of extracts of vine tea.

Chemical Compositions of Vine Tea

Vine tea (*A. grossedentata*) has been traditionally consumed in China as both an herbal tea and medicinal plant due to its pleasant flavor and health-promoting properties [28]. It contains a diverse range of bioactive secondary metabolites, including flavonoids, phenolic acids, polysaccharides, terpenoids, steroids, and volatile compounds [29]. Flavonoids represent the dominant class and are primarily responsible for the plant's biological activities, particularly its antimicrobial effects, which can inhibit or eliminate pathogenic microorganisms at relatively low concentrations [30-32]. More than 30 flavonoid compounds have been identified, with dihydromyricetin being the most abundant and extensively studied constituent [33]. The compounds isolated from vine tea and their chemical structures are listed in **Table 1 and Figure 3** [26,34,35].

Toxicological Profile of Vine Tea

Vine tea is often regarded as non-toxic, largely based on its long-standing and widespread use in plant-derived herbal medicines and beverages [36]. However, this perception is primarily supported by traditional consumption rather than comprehensive toxicological evaluation. Therefore, systematic toxicological evaluation using standardized experimental and regulatory frameworks is essential to substantiate the non-toxic profile of vine tea beyond empirical evidence derived from traditional use. Available studies generally suggest that vine tea is well tolerated under conventional dietary or experimental conditions; however, the current toxicological evidence remains fragmented and methodologically limited. Most evaluations report an absence of overt physiological toxicity associated with flavonoid-rich vine tea preparations, yet these conclusions are largely derived from short-term or narrowly scoped studies. For instance, Carneiro et al [37] reported no adverse sensory or physiological effects following consumption of vine tea infusions at intake-relevant concentrations, while Zhang et al [38] observed no significant hepatic or renal toxicity after oral administration of vine tea formulations in experimental models. Notably, these investigations do not systematically assess dose-response relationships, cumulative exposure, or potential toxic effects at pharmacologically relevant doses. Similarly Wu RR et al [5] reported that rats administered total flavonoids extracted from vine tea for 12 weeks at doses of 0.3 and 1.5 g/kg exhibited no significant alterations in general appearance, body weight, behavior, organ coefficients, or blood biochemical parameters, either during treatment or following a 2-week recovery period. Histopathological examination revealed no apparent lesions attributable to treatment, and no delayed toxicity was observed after cessation. While these findings support the absence of acute or sub-chronic toxicity in rodent models, the reliance on limited endpoints and a single species constrains the broader extrapolation of safety, particularly with respect to long-term human consumption, vulnerable populations, and non-oral exposure routes. Consequently, comprehensive chronic toxicity, reproductive toxicity, and human-relevant exposure studies are still required to substantiate the safety profile of vine tea.

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Table 1. The flavonoid compound isolated and identified from vine tea (*A. grossedentata*).

No	Flavonoid compounds	Molecular formula	References
1	3-Dihydroxyquercetin	$C_{15}H_{12}O_8$	[85]
2	Dihydromyricetin	$C_{15}H_{12}O_8$	[35,86,87]
3	Isodihydromyricetin	$C_{15}H_{12}O_8$	[86]
4	Myricitrin	$C_{21}H_{20}O_{12}$	[86]
5	Taxifolin	$C_{15}H_{12}O_7$	[86,88]
6	Myricetin	$C_{21}H_{20}O_{12}$	[89]
7	Quercetin-3-O-β-D-xyloside	$C_{20}H_{18}O_{11}$	[85]
8	Kaempferol	$C_{15}H_{10}O_6$	[85]
9	Luteolin	$C_{15}H_{10}O_6$	[35]
10	Phloretin	$C_{15}H_{14}O_5$	[85]
11	Apigenin	$C_{15}H_{10}O_5$	[35]
12	Hesperitin	$C_{16}H_{14}O_6$	[85]
13	Quercetin	$C_{15}H_{10}O_7$	[85]
14	Rutin	$C_{27}H_{30}O_{16}$	[35]
15	Apiin	$C_{26}H_{28}O_{14}$	[35]
16	Dihydroquercetin	$C_{15}H_{12}O_7$	[26,85]
17	Phloridzin	$C_{21}H_{24}O_{10}$	[85]
18	Kaempferol-3-O-α-l-rhamnoside	$C_{21}H_{20}O_{10}$	[85]
19	Kaempferol 3-O-sophoroside	$C_{27}H_{30}O_{16}$	[90]
20	Kaempferol 7-O-β-D-glucoside	$C_{21}H_{20}O_{11}$	[90]
21	Kaempferol-3-O-β-D glucuronide	$C_{21}H_{18}O_{12}$	[90]
22	Myricetin 3-O-rhamnoside	$C_{21}H_{20}O_{12}$	[43]
23	6,8-dihydroxy kaempferol	$C_{15}H_{10}O_8$	[26]
24	Epigallocatechin	$C_{22}H_{18}O_{11}$	[88]
25	Vitexin	$C_{21}H_{20}O_{10}$	[91]
26	Vitexin-2-O-rhamnoside	$C_{27}H_{30}O_{14}$	[91]
27	Quercetin-3-galactoside	$C_{21}H_{20}O_{12}$	[91]
28	Isoquercitrin	$C_{21}H_{20}O_{12}$	[87]
29	Quercetin-3-O-α-L-rhamnoside	$C_{21}H_{20}O_{11}$	[91]
30	Myricetin-3-O-β-D-glucoside	$C_{21}H_{20}O_{13}$	[92]
31	6,7-dihydroxy-3'-methoxy-4',5'-methylenedioxy isoflavone	$C_{28}H_{30}O_{16}$	[93]
32	6,7-dihydroxy-3'-methoxy-4',5'-methylenedioxy isoflavone 6-O-β-D-glucopyranoside	$C_{28}H_{30}O_{16}$	[93]
33	6,7-dihydroxy-3'-methoxy-4',5'-methylenedioxy isoflavone 6-O-β-D-xylopyranosyl-(1-6)-β-Dglucopyranoside	$C_{28}H_{30}O_{16}$	[93,94]
34	6,7-dihydroxy-3'-methoxy-4',5'-methylenedioxy isoflavone 6-O-α-L-rhamnopyranoside	$C_{28}H_{30}O_{16}$	[93]
35	5,7-dihydroxy-3',4'-trihydroxyflavone-3-O-6''-rhamnose	$C_{21}H_{20}O_{12}$	[95,96]
36	5,7-dihydroxy-3',4'-dihydroxyflavone-3-O-6''-rhamnose	$C_{15}H_9O_6$	[95,96]

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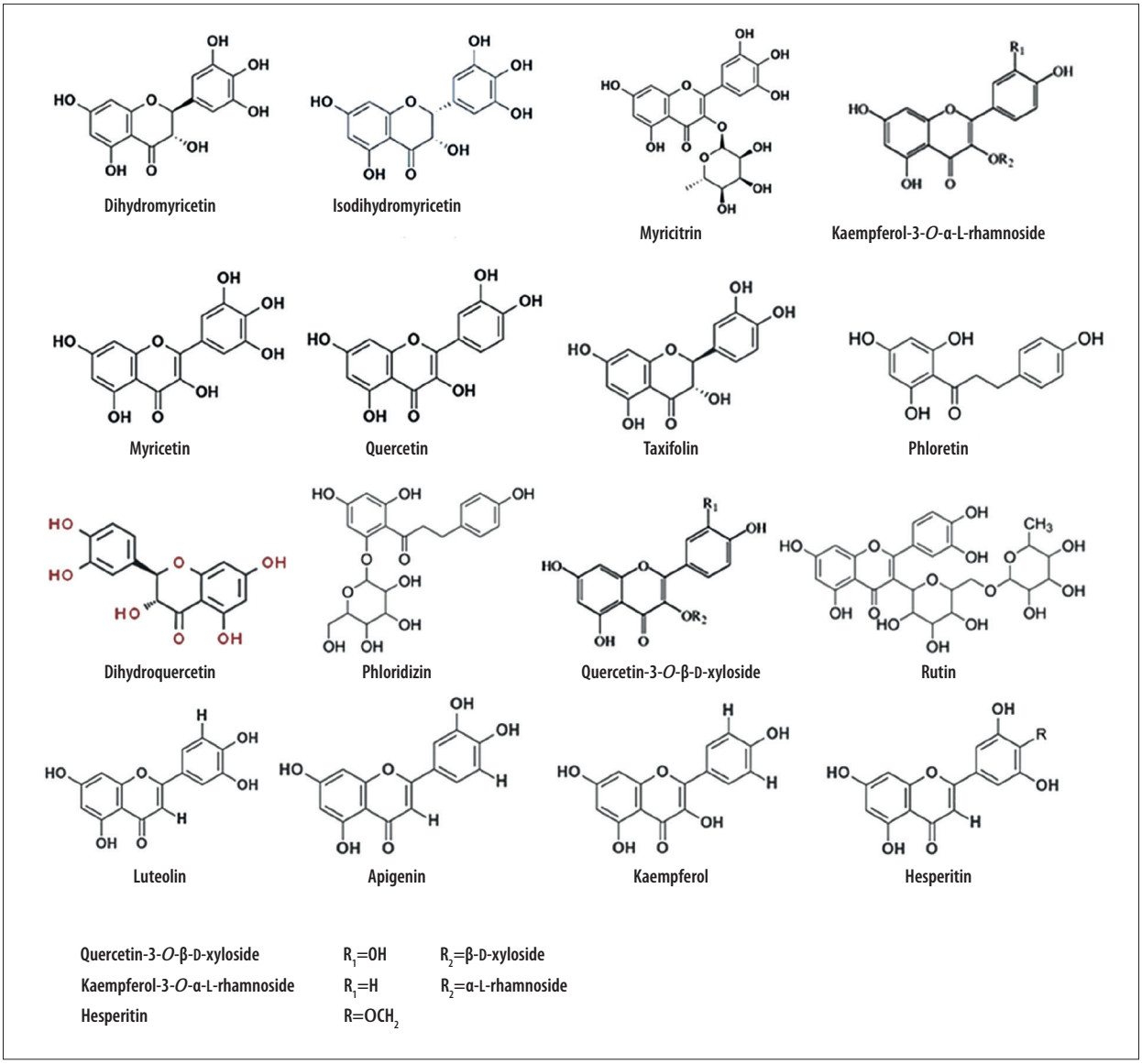

Figure 3. Chemical structures of major flavonoid compounds isolated from vine tea (*A. grossedentata*).

Biological Functional and Pathway Enrichment of Dihydromyricetin

Dihydromyricetin, a major flavonoid derived from vine tea, exhibits broad-spectrum antimicrobial activity and diverse biological functions associated with multiple metabolic and signaling pathways [39,40]. Li et al [41] reported that transcriptomic and pathway enrichment analyses indicate that dihydromyricetin biosynthesis is primarily regulated through the phenylpropanoid and flavonoid pathways and is associated with key enzymes, including chalcone synthase, phenylalanine ammonia-lyase, flavanone 3-hydroxylase, and flavonoid 3',5'-hydroxylase. These enzymes may function sequentially in precursor conversion, flavonoid skeleton formation, hydroxylation, and accumulation of dihydromyricetin. The process may also be regulated by transcription factors, including members of the MYB and bHLH families, which respond to environmental and metabolic cues. However, a closer examination of these analyses suggests that the precise regulatory mechanisms governing these pathways remain incompletely understood. In particular, the specific roles and interactions of these enzymes in driving dihydromyricetin accumulation in vine tea require further clarification and experimental validation. The functional enrichment analyses conducted by Ma et al [42] suggest that dihydromyricetin-responsive genes and proteins are involved in essential cellular processes, including metabolic regulation, ribosome assembly, lipid and nucleotide metabolism, and fatty acid and glutamine metabolism, indicating dihydromyricetin does not exert its antimicrobial effects through a single target; rather, it influences an interconnected network of biosynthetic and energy-related pathways.

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The molecular mechanisms associated with these differentially expressed proteins involved anion binding, cation binding, ligase, and phospholipase activities. Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway analysis indicated that the differentially expressed proteins were significantly enriched in D-alanine metabolism, pyrimidine metabolism, and aminoacyl–transfer RNA biosynthesis. Disruption of pyrimidine metabolism affected synthesis of RNA and DNA, whereas interference with aminoacyl–transfer RNA biosynthesis impaired protein synthesis [43].

At the cellular levels, dihydromyricetin is associated with functions such as ribosomal RNA binding, glutaminase activity, protein binding, and antioxidant enzyme activity, and is localized to key cellular components, including ribosomal subunits and membrane-associated structures [44]. KEGG pathway enrichment analyses reveal that dihydromyricetin is closely linked to ribosomal pathways, pyrimidine and nucleotide metabolism, ubiquinone and terpenoid quinone biosynthesis, and cationic antimicrobial peptide resistance. In addition, it modulates major signaling cascades, including NF-κB, FOXO, HIF-1, JAK-STAT, and Toll-like receptor pathways, which are implicated in oxidative stress, inflammation, apoptosis, and immune regulation [45].

Similarly, Hui et al [43] reported that the antimicrobial activity of dihydromyricetin is associated with multiple biological processes, including macromolecule metabolism, protein metabolism, and the biosynthesis of nitrogen-containing cellular compounds. At the molecular function level, differentially expressed proteins were primarily related to anion binding and kinase activity. KEGG pathway enrichment analysis further indicated significant involvement in pathways related to fatty acid degradation, butyrate metabolism, amino acid metabolism, and glyceride metabolism. Moreover, dihydromyricetin has been shown to influence gut microbiota-associated metabolism and bile acid signaling via the FXR/TGR5 axis, further supporting its role in systemic metabolic regulation [46].

Collectively, these findings suggest that dihydromyricetin exerts its biological effects through a multi-target and multi-pathway regulatory network. In this network, metabolic regulation, ribosomal activity, nucleotide biosynthesis, lipid metabolism, oxidative-stress responses, and membrane-associated processes may interact to reduce microbial viability. However, most existing evidence is derived from transcriptomic, proteomic, pathway-enrichment, and molecular docking analyses. Direct experimental validation of the causal relationships among these pathways remains limited. Therefore, future studies should integrate omics approaches with enzyme activity assays, targeted gene knockdown, metabolomic profiling, and membrane-function tests to clarify how these interconnected pathways collectively contribute to antimicrobial activity.

Antibacterial Activities of Dihydromyricetin

The antimicrobial activity of dihydromyricetin varies across different pathogenic bacteria, with notable differences observed between bacterial species and drug-resistant strains. Current evidence suggests that dihydromyricetin generally exhibits stronger antibacterial activity against gram-positive bacteria than gram-negative bacteria. The antibacterial activity of dihydromyricetin has been evaluated using the poisoned food method against 5 foodborne pathogens, including 3 gram-negative bacteria, *Escherichia coli*, *Pseudomonas aeruginosa*, and *Salmonella paratyphi,* and 2 gram-positive bacteria, *Bacillus subtilis* and *Staphylococcus aureus*. This study indicated that dihydromyricetin significantly inhibited the growth of all tested bacteria at concentration of 3.78 mg/mL [25]. Among these, *S. aureus* appears particularly sensitive, as evidenced by significant disruption of cell morphology, growth kinetics, and membrane integrity, even at relatively low concentrations [47]. Treatment of dihydromyricetin markedly reduced bacterial density and altered cellular morphology, disrupted the bacterial growth curve, prolonged the lag phase, and shortened the logarithmic growth phase. Scanning electron microscopy observed that untreated *S. aureus* cells exhibited smooth surfaces with normal spore formation, whereas cells treated for 12 hours showed extensive dissolution around the spores and severe damage to both the cell wall and membrane [48].

In contrast, gram-negative bacteria, especially *P. aeruginosa* and *Acinetobacter baumannii*, generally demonstrate greater tolerance [49]. The reduction of inhibitory effect may be partly attributed to the structural complexity of the gram-negative outer membrane, which can limit compound penetration and reduce the interaction of dihydromyricetin with intracellular or membrane-associated targets. Drug-resistant strains may further decrease susceptibility through adaptive resistance mechanisms, including altered membrane permeability, efflux pump activity, stress-response regulation, and biofilm formation [50]. Therefore, the variability in antimicrobial activity across bacterial species and resistant strains remains an important issue requiring further systematic investigation.

Recent studies suggest that formulation strategies may enhance the antibacterial effect of dihydromyricetin particularly against less susceptible gram-negative and drug-resistant strains of bacteria. An enhanced antibacterial effect has been observed when dihydromyricetin is formulated as co-crystals or a nano formulation or combined with other agents. For instance, dihydromyricetin–4,4'-bipyridine co-crystals exhibit strong activity against carbapenem-resistant *A. baumannii*, producing a 15-mm inhibition zone at 128 μg/mL, indicating improved potency against drug-resistant pathogens [51]. Similarly, co-crystallization with ciprofloxacin hydrochloride further increases antibacterial activity against *S. aureus* and *E. coli*, highlighting

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the role of formulation in enhancing dihydromyricetin bioavailability and efficacy [52]. Another formulation, chitosan-derived nanoparticles loaded with dihydromyricetin, has been shown to enhance bioabsorption and antibacterial activity against *S. aureus* and *E. coli*. In this study, *S. aureus* was more sensitive, as evidenced by a significantly larger inhibition zone at a concentration of 0.51 mg/mL, whereas *E. coli* exhibited an inhibition zone at a higher concentration of 1.02 mg/mL [53]. Dalcin et al [54] further reported that dihydromyricetin formulated as nanocapsules exhibited a significantly stronger antimicrobial effect against *P. aeruginosa* than the free compound. Specifically, the nanocapsule formulation inhibited the *P. aeruginosa* population by 78.6%, whereas free dihydromyricetin achieved a reduction of 70.5% at a concentration of 1 mg/mL. A summary of the antibacterial activities of dihydromyricetin is presented in **Table 2 and Figure 4**.

Overall, these findings suggest that dihydromyricetin demonstrates broad-spectrum antibacterial activity. However, its inhibitory effects are species-dependent and generally more effective against gram-positive bacteria than gram-negative bacteria. Although co-crystallization, nano-formulation, and

Table 2. Antimicrobial activity of dihydromyricetin derived from vine tea (*A. grossedentata*) against pathogenic bacteria.

Pathogenic bacterial	Source of dihydromyricetin	Experimental designed	Antibacterial mechanism	References
B. subtilis, *E. coli*, *S. paratyphi*, *P. aeruginosa* A mix of gram+ and gram- causes food-borne disease	Vine tea (*A. grossedentata*) extract	Disc diffusion method	Inhibited zone (15-18 mm), broad-spectrum antimicrobial activity	[25]
S. aureus ATCC 29213 gram+ causes food-borne and clinical disease	2R,3R- dihydromyricetin	Planktonic growing and biofilm assays	Inhibition planktonic growth, decreased biofilm biomass, and disrupted cell functions	[97]
V. parahaemolyticus gram- causes marine/seafood-borne disease	Vine tea (*A. grossedentata*) extract	Growth inhibition, cell morphology, membrane permeability, test	Deformation cells, increased membrane permeability, and degradation of cellular components.	[98]
E. coli gram- causes food-borne disease	Vine tea (*A. grossedentata*) extract	Disc diffusion assay	Disrupted cell envelope, energy metabolism, and stress-response proteins	[99]
E. coli (ETEC K88) gram- causes diarrheagenic swine	Dihydromyricetin (oral/in vivo model)	Mouse infection model; virulence, quorum sensing tests	Reduced virulence via AI-2 quorum-sensing inhibition and downregulated virulence genes	[33]
S. aureus, *E. coli*, *S. paratyphi*, *L. monocytogenes* A mixture of gram+, gram- causes food-borne pathogens	Vine tea leaves extracts (*A. grossedentata*)	Agar diffusion method	Broad-spectrum antibacterial activity	[26]
S. aureus, *P. aeruginosa*, *C. albicans* gram- caused burns and trophic ulcers pathogens	Dihydromyricetin (ampelopsin)	Animals wound healing model	Wound area reduced by 66.87% (5.15 to 1.71 cm^2) with 14 days	[100]
Salmonella typhimurium gram-	Vine tea (*A. grossedentata*) extract	Weaned piglets' infection model	Inhibited the abundance of *S. typhimurium* in the colon and improved PANoptosis in both the colon tissues and ileum	[101]

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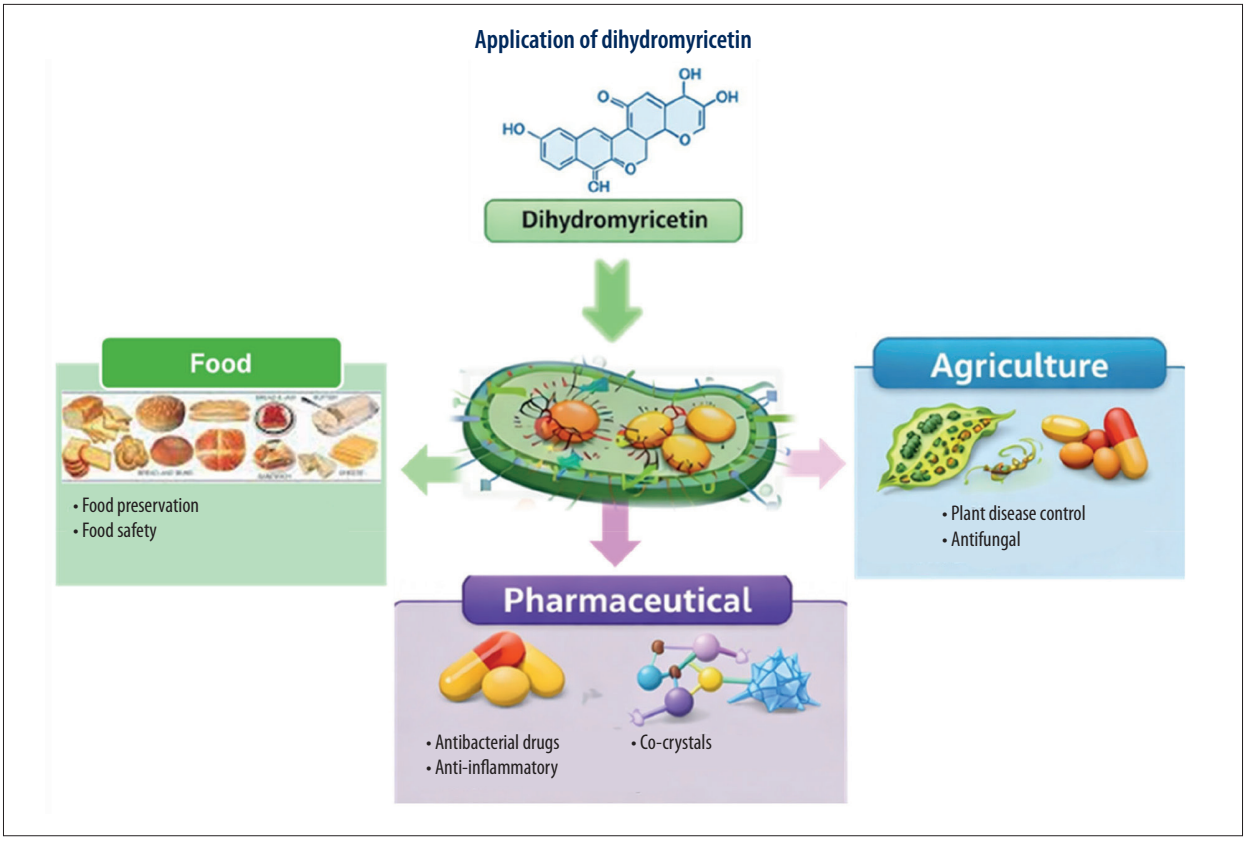

Figure 4. The application of dihydromyricetin derived from vine tea (*A. grossedentata*). Dihydromyricetin can be applied in food preservation, agriculture, and pharmaceutical development. Advanced formulation strategies, including nanoparticles and co-crystallization, improve its bioavailability and antimicrobial efficacy.

combination strategies may enhance the solubility, stability, penetration, and antimicrobial efficacy of dihydromyricetin, the optimal formulation strategy should be chosen according to the intended application. For biomedical or pharmaceutical applications, nanoparticle-base systems, including nanocapsules and chitosan-derived nanoparticles, appear particularly promising because they can improve biosorption, prolong retention, enhance penetration into microbial biofilms, and improve contact with fungal or bacterial cell surfaces. For food preservation, co-crystallization and edible biopolymer-based delivery systems may be more suitable because they can improve aqueous solubility, chemical stability, and controlled release while maintaining compatibility with food matrices. For agricultural applications, nano-formulations and encapsulated delivery systems may be advantageous for preservation of environmental stability, adhesion to plant surfaces, and improved antibacterial or antifungal activity for application in the field. However, biomedical or pharmaceutical optimization requires systematic evaluation using standardized checkerboard assays, fractional inhibitory concentration index analysis, time-kill curves, biofilm inhibition assays, and cytotoxicity testing. Future studies should also compare different drug ratios, delivery carriers, administration routes, and infection models to identify combinations that maximize antimicrobial efficacy while minimizing toxicity and resistance development. Therefore, dihydromyricetin should not only be considered as an independent antimicrobial compound but also as a potential adjuvant that may enhance the therapeutic performance of existing antimicrobial agents.

Antifungal Activities of Dihydromyricetin

Dihydromyricetin has also demonstrated antifungal activity against several pathogenic fungi, either alone or in combination with other agents. However, compared with its antibacterial effects, the antifungal activity of dihydromyricetin is generally less effective and requires relatively higher concentrations. Its antifungal activity also appears to be species-dependent, revealing that fungal cell wall composition, membrane structure, growth stage, and stress-response capacity may influence susceptibility to dihydromyricetin. For instance, dihydromyricetin inhibits *Aspergillus flavus* at 4 mg/mL by disrupting cell wall and membrane integrity, while also suppressing spore formation and mycelial growth [55]. It also shows effectiveness against *Penicillium italicum* by reducing lesion development

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Table 3. Antifungal activity of dihydromyricetin derived from vine tea (*A. grossedentata*) against pathogenic fungi.

Pathogenic fungi	Source of dihydromyricetin	Experimental designed	Antifungal mechanism	References
A. niger, *A. fumigatus*, *P. formosus*, *C. parapsilosis*, *C. albicans*	Vine tea extract	Agar diffusion method	Inhibited mycelial growth with zones of 17.6-22.2 mm at 0.73 μg/mL	[57]
A. flavus	Vine tea leaves extracts (*A. grossedentata*)	Disc diffusion method	Inhibited colony growth and sporulation at 4 mg/mL; damaged cell wall and membrane	[55]
P. italicum caused blue mold disease of citrus	Vine tea extracts	In vivo citrus inoculation; lesion measurement	Reduced lesion diameter, inhibited mycelial growth, induced plant defense enzymes	[56]

in infected fruits and enhancing host defense enzyme activity [56]. Importantly, nanoparticle formulations appear to overcome, at least partly, the limited antifungal potency of free dihydromyricetin. Dihydromyricetin-derived silver nanoparticles exhibit markedly stronger activity against multiple pathogenic fungi, including *Aspergillus niger*, *A. fumigatus*, *P. formosus*, *C. parapsilosis*, and *C. albicans*, with inhibition zones ranging from 17.6 to 22.2 mm at sub-microgram concentrations, indicating significantly higher potency compared with free dihydromyricetin. Among these, *P. formosus* is the most sensitive species [57]. The enhanced activity of dihydromyricetin-derived silver nanoparticles may be related to improved dispersion, increased contact with fungal surfaces, stronger penetration into fungal cells, silver ion release, oxidative stress induction, and combined damage to the cell wall and membrane. However, these mechanisms remain largely inferential and require direct verification through ROS assays, membrane-potential analysis, cell-wall integrity staining, transcriptomic profiling, and ultrastructural observation. Overall, these findings suggest that the antifungal activity of dihydromyricetin is relatively weak in its native form but can be substantially enhanced through formulation strategies, including co-crystallization and nanoparticle synthesis. Nanoparticle formulations appear to be particularly effective, as demonstrated by Phal et al [58] who reported that nanoparticles can readily penetrate pathogenic microbial cells, thereby enhancing the efficacy of bioactive compounds. This highlights the importance of delivery systems in optimizing the antimicrobial potential of dihydromyricetin across different pathogen groups. A summary of the antifungal activity of dihydromyricetin is presented in **Table 3**.

Mechanisms of Action: Cell Wall Disruption

The bacteria cell wall constitutes the first line response for antimicrobial agents [59]. Several mechanisms have been proposed to explain the antimicrobial effects of dihydromyricetin (**Figure 5**). One widely supported mechanism involves its interaction with microbial cell wall components, leading to structural disruption, impaired cell wall function, and disrupted bacterial membrane integrity [60]. This is consistent with the results of Wu Y et al [48], who observed pronounced morphological abnormalities in bacteria treated with dihydromyricetin, including cell wall degradation or loss and leakage of intracellular contents. These observations suggest that the antibacterial activity of dihydromyricetin treatment is partly attributable to its direct disruption of cell wall integrity, resulting in loss of cell viability. Another study confirmed that various proteins associated with cell wall metabolism were significantly upregulated after dihydromyricetin treatment, including LtaS, LytM, and SceD, which increased by 9-fold, 7.22-fold, and 2.68-fold, respectively. LytM is a zinc-dependent glycine-glycine endopeptidase produced by pathogenic bacteria [43]. LytM acts as a lytic enzyme involved in cell wall remodeling and autolysis through the hydrolysis of peptidoglycan. It specifically cleaves glycine-glycine peptide bonds within pathogenic bacterial peptidoglycan, thereby compromising cell wall integrity [61]. SceD is a glycosyltransferase that exhibits hydrolase activity of the cell wall [62]. In peptidoglycan, N-acetylmuramic acid and N-acetylglucosamine are linked by β-1,4-glycosidic bonds, which can be cleaved during cell wall hydrolysis [63]. This action influences peptidoglycan turnover and facilitates septum separation through cell division [64]. LtaS is a major enzyme involved in biosynthesis of lipoteichoic acid [65], which is a key component of gram-positive cell walls [66]. Lipoteichoic acid damage is typically associated with defects in cell division and reduced growth [67]. However, direct structural studies or molecular modeling experiments confirming the binding of dihydromyricetin to LtaS, LytM, or SceD remain limited. Therefore, the observed changes in these proteins should currently be interpreted as stress-response or compensatory remodeling events rather than definitive direct molecular targets.

Dihydromyricetin also affects the fungal cell wall, a structure essential for fungal growth and pathogenicity [68]. Previous

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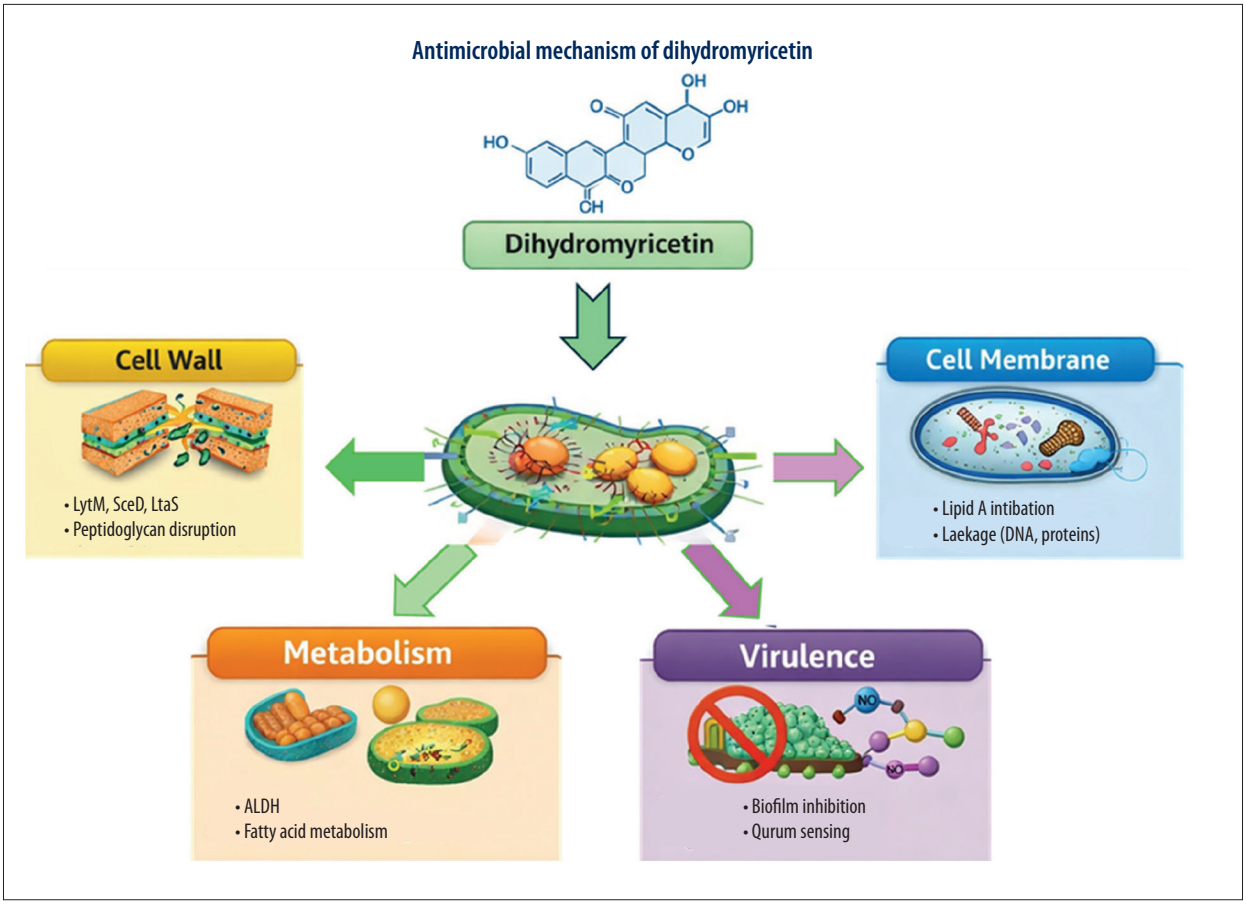

Figure 5. Schematic illustration of the antimicrobial mechanisms of dihydromyricetin. Dihydromyricetin exhibits antimicrobial activity through multiple pathways, including disruption of cell wall integrity via inhibition of peptidoglycan synthesis and hydrolysis enzymes, alteration of membrane permeability leading to leakage of intracellular components, interference with lipid and energy metabolism, and suppression of virulence factors and biofilm formation.

studies have shown that dihydromyricetin may disrupt the fungal cell wall by interfering with transmembrane electron transfer, inducing cell lysis, and promoting the oxidation of cellular components through the production of reactive oxygen species. In addition, dihydromyricetin may interact with DNA and proteins, ultimately affecting the respiratory chain and cell division processes [57]. Cell wall modification in fungal pathogens is regulated by key enzymes such as xyloglucan endo-transglucosylase/hydrolases (XTHs). Pathogenic fungi degrade host cell walls by inducing the expression of XTHs [69]. However, the inhibitory effect of dihydromyricetin on main fungal wall components, such as mannoproteins, beta-glucans, and chitin, have not been systematically quantified. Future studies should therefore evaluate XTH expression, chitin synthase activity, beta-1,3-glucan synthase activity, cell-wall integrity signaling, and fluorescence-based cell-wall staining to clarify how dihydromyricetin weakens fungal cell-wall structure and whether this contributes to its enhanced antifungal activity.

Mechanisms of Action: Cell Membrane Disruption

Membrane damage is also considered a major antimicrobial mechanism of dihydromyricetin (**Figure 5**). The compound interferes with proline dehydrogenase, a key regulatory and rate-limiting enzyme in proline metabolism. Dihydromyricetin binds to primary amino acid residues (Glu292, Arg288, Gly64, and Tyr285) within the hydrophobic pockets of proline dehydrogenase, resulting in decreased enzyme activity [70,71]. This interference affects the cationic peptide resistance system of pathogenic bacteria. Specifically, dihydromyricetin downregulates several proteins (ArnA, ArnB, AcrA, LpxA, PpiA, and NlpE), and reduced LpxA expression disrupts lipid A biosynthesis [72]. Lipid A is a crucial component of the outer membrane of gram-negative bacteria. It protects bacterial cells, enables adaptation to host environments, and prevents damage from chemotactic factors. Structurally, lipid A is composed of a phosphorylated glucosamine dimer substituted with fatty acyl chains and forms the hydrophobic anchor of lipopolysaccharides [73].

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Although docking analysis suggests potential interactions between dihydromyricetin and proline dehydrogenase, detailed kinetic parameters, including IC50 values and the type of inhibition, have not been fully established. Additional enzyme-kinetic assays are therefore required to determine whether the inhibition is competitive, non-competitive, or uncompetitive and whether other enzymes involved in proline metabolism are also affected.

Additionally, Liu W et al [74] demonstrated that dihydromyricetin disrupts bacterial membrane integrity, reduces membrane selectivity, and induces the leakage of intracellular components, including nucleic acids, polysaccharides, and metabolites. By altering membrane permeability and impairing normal membrane function, dihydromyricetin interferes with essential physiological processes, ultimately leading to bacterial cell death [75]. Similarly, dihydromyricetin treatment promotes the release of intracellular constituents, thereby damaging the bacterial cell wall and membrane. This process induces the leakage of alanine transaminase, alkaline phosphatase, and aspartate transaminase from bacterial cells, indicating a loss of cytoderm integrity and ultimately resulting in bacterial death [76]. At the mechanistic level, membrane injury may include direct and indirect activities. Directly, dihydromyricetin may alter membrane permeability and decrease membrane selectivity, leading in leakage of nucleic acids, metabolites, proteins, and polysaccharides. Indirectly, suppression of lipid A biosynthesis, disturbance of glycerophospholipid metabolism, and induction of lipid peroxidation may weaken membrane architecture and increase susceptibility to osmotic and oxidative stress. These effects may differ among microorganisms, due to gram-negative bacteria possessing an outer membrane enriched in lipopolysaccharides; however, gram-positive bacteria lack this barrier but have a thick peptidoglycan layer. Therefore, the broad-spectrum antimicrobial activity of dihydromyricetin should be understood as the combined outcome of species-specific cell-envelope vulnerability, membrane permeability changes, metabolic disruption, and stress-response failure.

The bacterial cell membrane is primarily composed of glycerophospholipids and proteins [77]. Glycerides are essential lipids in cells that play key physiological roles. They serve as the primary form of energy storage, such as triacylglycerols, and also provide the structural foundation for membrane lipids, such as phospholipids [78]. Wulf et al [79] reported that 3 glyceride-metabolism-related proteins were significantly upregulated following dihydromyricetin treatment, including aldehyde dehydrogenases (ALDH; EC 1.2.1.3), lipase 1, and lipase 2 (EC 3.1.1.3). The transformation of D-glyceraldehyde into D-glyceric acid is facilitated by the enzyme ADH [79]. This is supported by previous studies demonstrating that oxidative stress and membrane lipid peroxidation caused by antibacterial agents can generate reactive aldehydes that are toxic to cells. Bacteria can reduce this toxicity by transforming these aldehydes into less harmful substances through ALDH-driven oxidation ("acidification") [80]. Another study reported that dihydromyricetin treatment induces lipid peroxidation in pathogenic bacteria. In this context, glycerol oxidation generates glyceraldehyde, while increased ALDH activity enhances the detoxification of aldehyde-related compounds. Glyceraldehyde can be further transformed into D-glyceric acid, thereby reducing its intracellular accumulation. In addition, lipases hydrolyze triacylglycerols into diacylglycerols, which are subsequently converted into monoacylglycerols and ultimately yield free fatty acids and glycerol [81]. Phospholipids are the primary structural components of bacterial membranes, and fatty acids and glycerol serve as the main precursors for phospholipid biosynthesis [82]. However, the effects of dihydromyricetin treatment on specific lipid species remain unclear. Further lipidomic studies are needed to identify changes in phosphatidylethanolamine, phosphatidylglycerol, lipid A-related molecules, cardiolipin, and fatty-acid saturation patterns, together with assays evaluating membrane fluidity, permeability, and protein-lipid interactions.

Critical Evaluation of Current Evidence

Most studies evaluating the antimicrobial activity of dihydromyricetin rely on disk diffusion assays and inhibition zone diameters, with limited reporting of minimum inhibitory concentrations or concentration-response relationships. Comparative efficacy against established antimicrobial agents is rarely assessed, and effect sizes are generally not quantified. Additionally, variability in extraction methods, compound purity, and assay design introduces a risk of methodological bias. These limitations restrict quantitative synthesis and highlight the need for standardized experimental frameworks. The potential therapeutic use of dihydromyricetin is associated both with important benefits and unresolved risks. As a natural flavonoid, dihydromyricetin exhibits broad-spectrum antimicrobial activity against several pathogenic bacteria and fungi. It acts through multiple mechanisms, including disruption of cell wall integrity, alteration of membrane permeability, interference with lipid and energy metabolism, inhibition of protein synthesis, suppression of virulence-related processes, and modulation of oxidative-stress responses. These multi-target effects may reduce microbial viability and may also lower the likelihood of resistance development compared with single-target antimicrobial agents. However, several limitations must be addressed before dihydromyricetin can be translated into human therapeutic use. To optimize the safety and efficacy of dihydromyricetin for human use, future studies should adopt standardized and clinically relevant evaluation systems.

Developing more effective delivery systems, conducting long-term safety assessments, and validating efficacy under real-world

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conditions are essential steps toward translating the promising antimicrobial activity of dihydromyricetin into practical applications. In addition, the potential interactions between dihydromyricetin and other antimicrobial agents, together with its possible effects on the human microbiome, require careful consideration. Ultimately, a deeper understanding of the complex mechanisms underlying the antimicrobial activities of dihydromyricetin will be crucial for fully harnessing its therapeutic potential and addressing the growing threat of antimicrobial resistance.

Future Directions

Despite its promising antimicrobial activity, several challenges limit the practical application of dihydromyricetin. These include low bioavailability, chemical instability, variability in antimicrobial efficacy, and insufficient toxicological data. In addition, standardized evaluation methods and regulatory frameworks remain underdeveloped. The chemical stability and bioavailability of dihydromyricetin are affected by solubility, temperature, pH, gastrointestinal metabolism, oxidative degradation, and formulation type [83]. Xu et al [84] reported that inhalation delivery significantly improved dihydromyricetin bioavailability in pulmonary tissues and systemic circulation compared with oral administration. Therefore, application-specific delivery strategies are needed. Potential synergistic combinations with conventional antibiotics, antifungal agents, chitosan, silver nanoparticles, or membrane-disrupting adjuvants should be optimized using fractional inhibitory concentration index analysis, checkerboard assays, time-kill curves, resistance-development assays, and toxicity assessment. Long-term safety studies, microbiome-impact evaluation, and regulatory guidance are also needed to ensure safe and effective translation.

Conclusions

Dihydromyricetin, the major bioactive flavonoid isolated from vine tea (*A. grossedentata*), exhibits antimicrobial activity against a wide range of pathogenic bacteria and fungi in both in vitro and in vivo studies. Evidence demonstrates that dihydromyricetin inhibits mycelial growth and sporulation, reduces pathogenicity, and targets multiple cellular structures and processes, including disruption of cell wall integrity, alteration of membrane permeability, and interference with essential metabolic pathways. Its antimicrobial efficacy can be further enhanced through synergistic combinations, such as nanoparticle formulations and co-crystallization systems, which allow potent activity at lower concentrations. Although dihydromyricetin shows broad antimicrobial activity in vitro and supportive efficacy in selected in vivo models, its translation into agricultural, food, or pharmaceutical applications faces several challenges. These include variability in antimicrobial potency, formulation-dependent efficacy, limited toxicological data beyond dietary exposure, and the absence of regulatory guidelines for antimicrobial use. Future research should prioritize standardized minimum inhibitory concentration determination, comparative efficacy against existing treatments, long-term safety assessment, and application-specific delivery strategies.

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Availability of Data and Materials

The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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