

## MINI REVIEW

### Flavonoid: A Mini Review on Galangin

A. PRASAD<sup>1,\*</sup> and S. KUMARI<sup>2</sup>

<sup>1</sup>Department of Chemistry, Magadh Mahila College (Patna University), Patna-800001, India

<sup>2</sup>Department of Zoology, Magadh Mahila College (Patna University), Patna-800001, India

\*Corresponding author: E-mail: amritapd2407@gmail.com

Received: 29 September 2021;

Accepted: 23 October 2021;

Published online: 16 December 2021;

AJC-20614

Studies on flavonoids from plant sources has gained a momentum due to their versatile health benefits. The main sources of flavonoids in humans are fruits, vegetables, red wine and tea. They are small molecular weight secondary metabolites produced in different parts of the plant such as seeds, bark, root, fruits and flowers. Galangin (3,5,7-trihydroxyflavone) a naturally occurring flavonoid is present in roots of *Alpinia officinarum*, honey and propolis. Its extraction and detection has been reported using various methods such as chromatography, spectrophotometric and electrochemical methods associated with other sophisticated techniques. Galangin showed various pharmacological activities such as anti-inflammatory, antioxidative, radical scavenging, anticancer activity and hypolipidemic activity. The present review discusses the pharmacological activities, pharmacokinetics and bioanalytical aspects of galangin, which can be beneficial for researchers working in the field of galangin.

**Keywords:** Galangin, *Alpinia officinarum*, Flavonoids, Anti-inflammatory activity, Antimicrobial activity, Pharmacokinetics.

## INTRODUCTION

Since antiquity plants play important role in human lives as they are taken by us in the form of food as well as medicine. A number of drugs used in today's time are from natural sources and almost more than 25% are plant derivatives. These active phytoconstituents derived from plants are used for curing different diseases [1,2]. Flavonoids are plant-derived polyphenolic compounds which are responsible for different colors (yellow, orange and red) of plant parts like fruits, flowers and leaves. Being phytochemicals, they can only be synthesized by plants and not by human beings or animals [3]. The flavonoid term is taken from the Latin word "flavus", meaning yellow. Flavonoid are secondary metabolites of plants, though not considered as nutrients they make important components of human diet. A large variety of compounds such as flavonols, flavones, flavanols, flavanonols, flavanones, and isoflavones are categorized as Flavonoid; with a basic flavan nucleus, of two aromatic rings (ring A and B) of 6 carbon atoms interconnected by a heterocycle (ring C) of 3 carbon atoms [4-6].

Almost about 4,000 flavonoids from edible plants which we intake through fruits and vegetables are beneficial to health [7]. Flavonoid are synthesized biologically through a metabolic pathway of combination of shikimic acid and acyl polymalonate [8]. *In vitro* studies suggest that flavonoids are protective against reactive oxygen species (ROS) such as alkoxy, hydroxyl, or peroxy radicals. It defends human body from cancer, aging and cardiovascular diseases. A major group of flavonoids called flavonols occurs in the form of glycosides in plants. The most common flavonols quercetin, myricetin, galangin and kaempferol are found attached to glucose, galactose and other sugar moieties.

**Structure, properties and sources of galangin:** Galangin (3,5,7-trihydroxyflavone) bears 3-OH groups and therefore, it is placed in flavonol group of flavonoids (Fig. 1). It is present in significant level in honey, *Alpinia officinarum* Hance (Zingiberaceae family), *Helichrysum aureonitens* and in propolis. Galangin is involved in various pharmacological activity and it acts as a potential antioxidant [8], antimicrobial [9], antidiabetic [10,11], antitumor [12], antiobesity [13], anti-

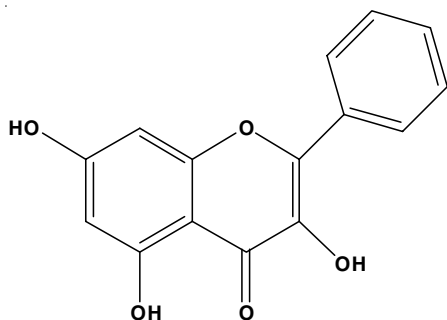


Fig. 1. Structure of galangin

inflammatory [14], antiosteoporosis [15] and lipid regulating effects [16].

**Isolation, extraction and detection techniques for galangin:** Isolation of galangin from *Alpinia officinarum* was achieved using high speed counter current chromatography coupled with 1,1-diphenyl-2-picrylhydrazyl-high performance liquid chromatography (DPPH-HPLC) assay and their antioxidant activities evaluation were carried out [17]. In this work, separation of galangin and kaempferide were carried out with purities 99.3% and 98.5%, respectively. Response surface methodology (RSM) technique was used for extraction of galangin from Galangal [18] and other experimental variables were also optimized. The conditions optimized for extraction were 90% ethanol in the ratio of 25 mL g<sup>-1</sup> to the material, extraction temperature 80 °C and extraction time 3 h. Under these optimized conditions, the yield of galangin experimentally was in close agreement with the predicted value. In another work, two major bioactive flavonoids *viz.* galangin and its derivative 3-O-methyl galangin were assessed using HPLC, with a simple, sensitive and accurate method, to evaluate the quality of *Alpinia officinarum* [19]. The HPLC column consisted an octadecyl silyl silica along with methanol water and phosphoric acid (60:38:2) ratio as the mobile phase and wavelength for detection was used as 254 nm. A good linear relationship was obtained for both the molecules with correlation coefficients higher than 0.999 for the analytes. In another work, a precise, simple and rapid HPLC coupled with photodiode array detection method [20] was adopted for the simultaneous determination of six antioxidant phenolic compounds *e.g.* galangin, luteolin, rutin, quercetin, genistein and curcumin in propolis. For isolating them from propolis sample ultrasound-assisted extraction was applied. The method showed good sensitivity, linearity, repeatability and accuracy. Spectrophotometric detection of galangin was also carried out by Dezmirean *et al.* [21]. Flavonoid content was investigated in propolis and poplar buds using zirconium oxychloride reagent. The results showed the lower concentration of galangin in poplar buds than that of propolis and also the concentration varied depending upon the origin of propolis. The electrochemical behaviours of galangin and 10 structurally different flavonoids were studied by cyclic voltammetry on a glassy carbon electrode (GCE) [22]. The electrode was modified using 1 mM nitrophenyl diazonium salt prepared in acetonitrile. Since nitrophenyl-modified GCE surface was electroinactive, it is activated by reducing the nitro group into the amine group. Then these electrodes were used for the determination of

antioxidant activities of 10 flavonoid derivatives with cyclic voltammetry technique. In another work, a new GC sensor electrode using 2-amino-3-hydroxypyridine was developed for the quantitative determination of quercetin, galangin, 3-hydroxyflavone and chrysin separately and simultaneously by cyclic voltammetry [23]. Along with cyclic voltammetry, electrochemical impedance spectroscopy and scanning electron microscopy techniques were used for surface characterization of the modified electrodes. Modification with 2-amino-3-hydroxypyridine onto the GC electrode surface was carried out in a potential window -150 to +600 mV and scan rate with (100 mV s<sup>-1</sup>, 30 cycles). The binding of flavonoid derivatives was performed in a different potential window (+300 to +1,700 mV) and different scan rate (100 mV s<sup>-1</sup>, 10 cycles). Experimental results showed the successful application of these modified GC electrode for the separate determination of these molecules. Chromatographic analysis and antioxidant activity of Greek propolis were also reported [24]. In this work, they developed a novel method based on HPLC-electrospray photo diode array mass spectrometry (HPLC-PDA-ESI/MS) for identification and quantification of different phenolic compounds from hydroalcoholic extracts of Greek propolis. The results obtained were verified by UV. Galangin was among the six most abundant phenolic components and gave two peaks at  $\lambda_{\max}$  265 and 356 nm.

### Pharmacological activities of galangin

**(I) Antioxidant activity:** The antioxidant activity (radical scavenging and metal ion chelation ability) and its mechanisms of action in a flavonoid depends upon the functional group around the nuclear structure and are highly influenced by its configuration, substitution and total number of hydroxyl groups [25,26]. During scavenging of reactive nitrogen species (RNS) and reactive oxygen species (ROS) the significant factor is the configuration of hydroxyl B ring as it produces more stable flavonoid radicals by donating electron and hydrogen to other radicals (peroxyl, hydroxyl) and stabilizing them [27]. Tremil & Šmejkal [28] also confirmed that the important factor for hydroxyl radical scavenging are hydroxylation of ring B, a C2-C3 double bond connected with a C-3 hydroxyl group and a C-4 carbonyl group.

Firuzi *et al.* [29] evaluated the structure-antioxidant activity relationships (SAR) of 18 different flavonoids using ferric reducing antioxidant power (FRAP) assay and cyclic voltammetry, which directly determined the reducing capacity of a compound. Galangin showed a well-defined irreversible anodic peak ( $E_{ap} \pm 0.59$  V). Galangin with 3-OH is much more active than other flavonoids which lack 3-OH group. The antioxidant activities of galangin and kaempferide has also been evaluated in a different work by measuring their scavenging percentages with the help of different luminol chemiluminescence (CL) systems in various radicals such as hydrogen peroxide, superoxide anion and hydroxyl [17]. Galangin and kaempferide presented potent antioxidant activities. Aloud *et al.* [30] investigated that galangin, improves antioxidant status and reduces hyperglycemia-mediated oxidative stress in streptozotocin-induced diabetic rats. Enhanced levels of lipid

hydro peroxides, plasma glucose, conjugated dienes and thio-barbituric acid reactive substances were observed in diabetic rats. In diabetic control rats, the activity of enzymatic oxidants was found to decline significantly. Decrease in the levels of insulin and non-enzymatic antioxidants was observed. Introduction of galangin and glibenclamide leads to reversal of the altered non-enzymatic antioxidants ions, plasma glucose and insulin to normal level. Sulaiman [31] studied the antioxidant activity of galangin by using quantum chemical calculations and density functional theory (DFT) method. Scavenging capacity of free radicals were estimated by using DPPH and it was compared with vitamin C as a control. Galangin exhibited the concentration and time dependent response in HCT-116 where it reduces the cell proliferation rate. To explore the role of B ring, a comparative study was done between null B-ring galangin and 3,5,7-trihydroxychromone without a B-ring using spectro-photometric assays by scavenging of O<sub>2</sub>, DPPH, 2-phenyl-4,4,5,5-tetramethylimidazole-1-oxyl-3-oxide radical, 2,2'-azinobis(3-ethylbenzothiazoline-6-sulfonic acid) radical (ABTS<sup>•</sup>) and Fe<sup>3+</sup>-reducing activity [32]. The results indicated that galangin and 3,5,7-trihydroxychromone follows similar antioxidant pathways which includes the redox-dependent pathways and a non-redox-dependent radical adduct formation pathway. It is concluded that null B-ring has little or no effect in changing their antioxidant pathways. However, it improved the level of antioxidants in these pathways which can be attributed to B-ring and its conjugation, resulting in more resonance forms and bonding sites. Flavonoids isolated from *Alpinia galanga* L. (Zingiberaceae) galangin and kaempferide were studied for their antioxidant capacity and radio-protective properties against radiation induced cellular DNA damage by Divakaran *et al.* [33]. They concluded that the rhizome extracts and the isolated flavonoids scavenged superoxide and hydroxyl radicals effectively in a dose dependent manner. These radicals were generated inside the body during metabolism and in the presence of xenobiotics. Antioxidant property of honey [34] and various components of propolis [35-37] have been investigated and found that galangin possessed marked antioxidant activity. Several other antioxidant activity of galangin has already been reported [38,39].

**(II) Anticancerous activity:** Galangin showed antitumor activities but because of poor solubility its use in pharmaceutical formulation for oral uses is difficult, which results in bio-availability variation. Li *et al.* [40] reported that galangin causes the apoptosis and autophagy as well as it suppresses the proliferation in the hepatocellular carcinoma cells [40]. The results demonstrated that galangin induced autophagy, decreases the level of acetylation of endogenous LC3. Galangin induces increase in binding of SIRT1-LC3 in HepG2 cells. In SIRT1 knocked-down cells, autophagy was subdued which resulted in reduction of the acetylation of endogenous LC3. In total, the above findings given an insight into a new mechanism of galangin induced autophagy with SIRT1 linked deacetylation of endogenous LC3. Chien *et al.* [41] showed that galangin has widespread applications in clinical therapy as anti-metastatic medicament. They investigated that galangin has an ability to inhibit 12-O-tetradecanoylphorbol-13-acetate (TPA), which

causes invasion and metastasis in HepG2 liver cancer cells. Nishita *et al.* [42] reported that *Alpinia officinarum* Hance extract inhibited the proliferation of vascular smooth muscle cell by increasing the expression of p27KIP1 and thus arresting the cells in G0/G1 phase. It was suggested that galangin has the same anti-proliferative activity and found to be a significant component of this extract as the raw extract. Yao *et al.* [43] studied the pharmacokinetics and antitumor activity of free galangin and PEGylated liposomes loaded with galangin. The galangin-liposomes and galangin-PEGylated liposomes were prepared using thin-film dispersion method. Experimental results of apoptosis showed that galangin-PEGylated liposomes demonstrated the higher level of cytotoxicity to hepatoma cells than the other two forms of drug carrier. In a recent study, nanosized galangin loaded niosomes was also evaluated for their antitumor activity on chemically induced HCC in rats [44]. The main hindrance during galangin use in clinical application is its low solubility, which limits the bioavailability of galangin. This problem was resolved by preparing niosomal vesicles using cholesterol and non-ionic surfactant in different molar ratios with the help of reverse-phase evaporation technique. Drug loading capacity, drug entrapment efficiency and percentage drug released were evaluated for the prepared niosomal formulations. Galangin loaded niosomes showed the significant improvement for liver cancer biomarkers. Immunohistochemical examination and histopathological study showed that gal-niosomes exhibited marked decline in minichromosome maintenance 3 (MCM3) immunostaining hepatocytes. Galangin loaded niosomes were found to be promising in improving the antitumor activity against liver cancer.

**(III) Hypolipidemic activity:** Potential role of galangin has been shown in obesity control as administration of galangin decreases the level of serum lipid, peroxidation of lipids and liver weight [45]. Galangin metabolites (galangin-3-O- $\beta$ -D-glucuronic acid and galangin-7-O- $\beta$ -D-glucuronic acid) separated from urine samples of rat and purified using HPLC are found to be effective *in vivo* components [46]. Further, spectroscopic analysis was done to identify the structure of metabolites. Treatment of HepG2 cell line with galangin had showed the down or up regulation in the level of lipogenic gene as assessed by real-time quantitative polymerase chain reaction (RT-qPCR). The changes in the level of lipogenic gene were regulated by inhibition of genes or proteins associated with biosynthesis of cholesterol and triglycerides. Antiobesity effects of galangin was studied in cafeteria diet (CD) fed female rats where galangin inhibited the enzyme pancreatic lipase [45]. In *in vitro* conditions, the inhibitory activity of galangin was assessed by quantifying the release of oleic acid from triolein. Inhibition of energy intake, increased body weight and parametrial adipose tissue weight induced by cafeteria diet was also effected by galangin. Reduction of the fatty acid degeneration of liver tissue caused by eating of high fats diet at both the prevention and treatment levels was seen as an effect galangin and this may be related to an enhancement of hepatocyte autophagy [16]. Galangin decreases the lipid accumulation and increases the level of hepatocyte autophagy at cellular level. This protective effect of galangin was blocked through inhibition

of autophagy by 3-methyladenine. Aloud *et al.* [47] elucidated galangin effect on complication related to hyperglycemia and lipid changes in rats. The result demonstrated that the administration of galangin reduced hyperlipidemia and diabetic complications. These finding suggested that galangin could be used in treatment of diabetic hyperlipidemia patients.

**(IV) Antimicrobial activity:** Galangin and other antibacterial agents (novobiocin and penicillin G) caused the disruption of cytoplasmic membrane of *Staphylococcus aureus*, which resulted into release of potassium from bacteria [48]. Minimum inhibitory concentrations (MICs) of galangin, penicillin G and novobiocin against *S. aureus* was found to be 50 µg/mL, 31.3 ng/mL and 62.5 ng/mL, respectively by agar dilution assay. After 12 h incubation in 50 µg/mL galangin, *S. aureus* lost 21% more potassium compared to the untreated control population. Bacterial population incubated with 31.3 ng/mL penicillin G exhibited a 6% more potassium loss whereas novobiocin had no effect on potassium loss. These results demonstrate galangin significantly enhanced the loss of potassium from *S. aureus* cells, which is caused either due to direct cytoplasmic membrane damage or by weakening of the cell wall and consequent osmolysis. In another work, galangin antibacterial activity was assessed with *S. aureus* strains resistant of 4-quinolone. It was done using an agar dilution assay [49]. MIC of approximately 50 mg/mL was determined against 16 strains; among them were those with 250 and 500 fold increase in norfloxacin resistance. Cushnie *et al.* [50] studied the aggregatory effect of galangin in bacterial cells. Bacterial growth was inhibited after aggregation. The antibacterial activity of galangin and ceftazidime; their synergy and primary mechanism of action against *S. aureus* were investigated by determining MIC, killing curve determinations, electron microscopy and enzyme assay method [51]. Galangin showed the significant inhibition against penicillinase and β-lactamase. Combination of galangin and ceftazidime damaged the ultra-structures of the cell as clearly shown by electron microscopy. The result indicated that baicalein, quercetin and galangin reversed bacterial resistance significantly for β-lactam antibiotics against penicillin-resistant strain of *S. aureus*. Eumkeb *et al.* [52] showed that the galangin, kaempferide and kaempferide-3-O-glucoside exhibited antibacterial activity against amoxicillin-resistant *E. coli* and *via* inhibition of peptidoglycan and ribosome-synthesis they can even reverse the resistance. In a recent work, galangin antimicrobial activity was also investigated against murein hydrolases of vancomycin-intermediate *S. aureus* strain Mu50 [53]. The results showed that galangin with 32 µg/mL of MIC against N315, ATCC25293, and Mu50 inhibited the bacterial growth. Using TEM, it was demonstrated that after galangin treatment, Mu50 daughter cells division was inhibited. Other antimicrobial activity of galangin has also been reported against another bacterial strains *Campylobacter jejuni* [54] and parasite *Leishmania amazonensis* [55]. Anti-bacterial properties was studied for eight flavonoids against four Gram-positive and four Gram-negative bacteria [56]. Galangin was found to be the most active flavonoid, due to presence of two hydroxyl groups on ring A and the absence of polar groups on ring B in its structural feature. Consecutive methylation of each hydroxyl

group decreases the activity in 3-O-methylgalangin and resulted in inactivation of 3,7-O-dimethylgalangin. It was concluded that the amphipathic features of flavonoids are crucial for the antibacterial activity.

**(V) Anti-inflammatory activity:** Galangin anti-inflammatory effect has been reported in asthma, acute lung injury, arthritis, paw edema and acute kidney injury. In murine macrophage cell line RAW 264.7 the anti-inflammatory effects of galangin is shown [57]. Cytotoxic effect of galangin is not observed in RAW 264.7. Nitric oxide (NO) production in lipopolysaccharide (LPS) stimulated RAW 264.7 was significantly decreased in presence of 50 mM galangin. Interlukin-1β (IL-1β) production in LPS activated macrophages was inhibited by galangin. Anti-inflammatory effect of galangin is exerted by inhibition of ERK and NF-κB phosphorylation. These result indicated that anti-inflammatory effect of galangin on LPS activated macrophages is done by inhibition of NF-κB-p65, ERK and pro-inflammatory gene expression. Galangin suppressed the expression of pro-inflammatory cytokines and inducible nitric oxide synthase (iNOS) meanwhile, IL-10 expression was enhanced microglia cell line BV2 after LPS stimulation [58]. Pro-inflammatory cytokine expression and microglial cells activation were inhibited by galangin after LPS administration in mouse brain. In another study by Huang *et al.* [59], galangin was found to enhance cisplatin-induced acute kidney injury *via* ERK and NF-κB signaling pathways. It also inhibited the oxidative stress, inflammatory response, apoptosis and necroptosis [59]. Galangin anti-inflammatory effects during *in vitro* and *in vivo* neuro inflammatory conditions initiated by polyinosinic-polycytidylic acid (poly(I:C)), a viral mimic dsRNA analog were investigated [58]. Decrease in the production of NO, ROS and pro-inflammatory cytokines in poly(I:C) stimulated BV2 microglia was observed. The anti-inflammatory effects and mechanism of action of quercetin and galangin in LPS stimulated RAW264.7 macrophages were investigated [60]. The results showed that quercetin and galangin subdued the production of iNOS, IL-6, NO. It also deceases the nuclear translocation of NF-κB. In another study, the molecular mechanism behind galangin role in attenuation of uric acid induced renal inflammation in normal rat kidney epithelial cells NRK-52E was described [61]. The findings indicated that galangin treatment protected NRK-52E cells against uric acid induced renal inflammation by suppressing tumor necrosis factor (TNF)-α, IL-1β, IL-18, prostaglandin E2 (PGE2), and NO production. Galangin also inhibited the iNOS, prostaglandin endoperoxide synthase 2 (PTGS2), TNF-α, IL-1β and IL-18 mRNA expression. Inhibitory effect of galangin on LPS-induced matrix metalloproteinases (MMP-9) expression in rat brain astrocytes (RBA-1 cells) was investigated [62]. Results showed that the galangin treatment diminished the LPS-mediated production of MMP-9 protein, mRNA expression and activity of MMP-9 promoter. Two flavonols, galangin and kaempferol were investigated for their effect on indomethacin damaged rat intestine epithelial (IEC-6) cells [63]. Treatment of cells with 300 µmol/L indomethacin for 24 h caused cell toxicity, which resulted in the loss of cell viability, increased lactate dehydrogenase (LDH) release and ROS production.

**(VI) Pharmacokinetics:** Ye *et al.* [64] studied the pharmacokinetics of galangin in plasma and tissue of rat by a sensitive and reliable ultra-high performance liquid chromatography-tandem mass spectrometry (UHPLC-MS/MS) technique. The method was precised upto more than 12.1% and the accuracy range occurs from -4.8% to 8.1%. Rapid galangin absorption ( $t_{\max}$  0.25 h) and fast elimination ( $t_{1/2} > 1.1$  h) was demonstrated after three different dosages and ~7.6% of absolute bioavailability was observed from pharmacokinetics results. Galangin was abundantly present in liver, lung, spleen, kidney, and smaller amounts in brain as revealed from the tissue distribution analysis. Yao *et al.* [43] compared the pharmacokinetics of free galangin and galangin-loaded PEGylated liposomes. Experiments performed *in vivo* demonstrated that  $t_{1/2}$  of galangin in modified liposomes was 4 h more as compared to the free galangin in the plasma of rats. Results suggested that pharmacokinetic parameters of galangin gets amended due to PEG modification resulting in an increase in solubility and thereby its effectiveness in liver cancer treatment. Jia *et al.* [65] worked on simultaneous determination of galangin and other four flavonoids namely kaempferol, kaempferide, galangin-3-methyl-ether, and quercetin in rat plasma through oral administration of Mongolian medicine, Shudage-4 extracts. The metabolism of Mongolian medicine and its transport were found to be faster in gastric ulcer rats than that in normal rats possibly due to flavonoids in Shudage-4 gets concentrated and consumed faster at the lesion site in gastric ulcer rats than the normal group [65]. Ma *et al.* [66] studied the pharmacokinetic parameters and evaluated the effects of galangin administration on the activities and expression of mRNA in seven Cytochrome P450 (CYP) systems using a "cocktail-probes" approach. The mRNA-expression levels of CYP were investigated in RT-qPCR experiments. The galangin treated group showed a marked decrease in area under concentration time curve extrapolated to infinity ( $AUC_{0-\infty}$ ) and  $C_{\max}$  (maximum concentration) values for CYP1A2 and CYP2B3. The results of mRNA-expression and pharmacokinetic were found to be consistent. They concluded that the long-term galangin administration can alter the CYP450 enzyme activities, establishing galangin as a potential candidate for enhancement of oral drug bioavailability and reversal of multi-drug resistance. Galangin gets converted rapidly into its glucuronidated metabolites *in vivo* [67]. In a recent work, an analytical method UFLC-MS/MS was developed for simultaneous determination of galangin-3-O- $\beta$ -D-glucuronic acid and galangin-7-O- $\beta$ -D-glucuronic acid in rat plasma. Blood was obtained from orbital sinus after administrating galangin extract (0.3 g/kg) orally, then methanol precipitation treatment and gradient elution with Phenomenex Kinetex 2.6 mm XB-C18 column was carried out. Pharmacokinetic parameters of both the metabolites were also studied. Chen *et al.* [64] found that systemic exposure to oral and intravenous administration of galangin and its metabolites in rat plasma was different. Major findings of their experiment can be looked upon as bioavailability of free galangin was very low, its oral dosing resulted in glucuronidation whereas intravenous medication resulted in sulphation, oxidation reaction can be predicted as conjugates of kaempferol were detected,

further both glucuronidation and sulfation were found to be efficient.

## Conclusion

Flavonoids, secondary metabolites of plants are a broad class of low molecular weight phenolics having basic flavan nucleus. It is widely distributed in plant parts like leaves, bark, seeds and flowers of plants. Flavonoids protect plants against UV radiation, herbivores and pathogens. Galangin (a phytoconstituent) is a flavonol class of flavonoid, significantly present in honey, *Alpinia officinarum*, *Helichrysum aureonitens* and propolis and is being used in traditional medicine for a long time. The present review brings together the various aspects and works done on galangin including the techniques involved for its isolation, extraction, detection and its pharmacological activities including its antimicrobial, anti-inflammatory, anti-tumor, antioxidant and lipid regulating effects. Galangin attracted much attention to the researchers, but further investigation is needed due to its medicinal importance, which can be helpful for its development as a molecular agent to combat different type of diseases.

## ACKNOWLEDGEMENTS

The authors thank the University Grant Commission, New Delhi for granting UGC-BSR Start-UP Project (No.F.30-477/2019 (BSR)).

## CONFLICT OF INTEREST

The authors declare that there is no conflict of interests regarding the publication of this article.

## REFERENCES

1. D.K. Patel, D. Laloo, R. Kumar and S. Hemalatha, *Asian Pac. J. Trop. Med.*, **4**, 748 (2011); [https://doi.org/10.1016/S1995-7645\(11\)60186-7](https://doi.org/10.1016/S1995-7645(11)60186-7)
2. D.K. Patel, R. Kumar, S.K. Prasad and S. Hemalatha, *Asian Pac. J. Trop. Biomed.*, **1**, S131 (2011); [https://doi.org/10.1016/S2221-1691\(11\)60140-8](https://doi.org/10.1016/S2221-1691(11)60140-8)
3. R. Koes, W. Verweij and F. Quattrocchio, *Trends Plant Sci.*, **10**, 236 (2005); <https://doi.org/10.1016/j.tplants.2005.03.002>
4. L. Peng, B. Wang and P. Ren, *Colloids Surf. B Biointerfaces*, **45**, 108 (2005); <https://doi.org/10.1016/j.colsurfb.2005.07.014>
5. D. Procházková, I. Bouřová and N. Wilhelmová, *Fitoterapia*, **82**, 513 (2011); <https://doi.org/10.1016/j.fitote.2011.01.018>
6. J.S. Kim, O.J. Kang and O.C. Gweon, *J. Funct. Foods*, **5**, 80 (2013); <https://doi.org/10.1016/j.jff.2012.08.006>
7. R.J. Nijveldt, E. van Nood, D.E. van Hoorn, P.G. Boelens, K. van Norren and P.A. van Leeuwen, *Am. J. Clin. Nutr.*, **74**, 418 (2001); <https://doi.org/10.1093/ajcn/74.4.418>
8. P.P. Venkateswara Rao, S. Kiran, P. Rohini and P. Bhagyasree, *J. Pharmacogn. Phytochem.*, **6**, 2778 (2017).
9. I. Górniak, R. Bartoszewski and J. Króliczewski, *Phytochem. Rev.*, **18**, 241 (2019); <https://doi.org/10.1007/s11101-018-9591-z>
10. M.H. Abukhalil, O.Y. Althunibat, S.H. Aladaileh, W. Al-Amarat, H.M. Obeidat, A.A.A. Al-khawalde, O.E. Hussein, M.A. Alfwuaires, A.I. Algefere, K.M. Alanazi, F.K. Al-Swailmi, H.H. Arab and A.M. Mahmoud, *Biomed. Pharmacother.*, **138**, 111410 (2021); <https://doi.org/10.1016/j.biopha.2021.111410>

11. A.A. Aloud, C. Veeramani, C. Govindasamy, M.A. Alsaif and K.S. Al-Numair, *Redox Rep.*, **23**, 29 (2018); <https://doi.org/10.1080/13510002.2017.1365224>
12. H. Huang, A.Y. Chen, X. Ye, R. Guan, G.O. Rankin and Y.C. Chen, *Molecules*, **25**, 1579 (2020); <https://doi.org/10.3390/molecules25071579>
13. P. Prasathong, S. Meeapat, S. Rattanakanokchai, J. Khamseekaew, S. Bunbupha, P. Prachaney, P. Maneesai and P. Pakdeecheote, *Antioxidants*, **10**, 769 (2021); <https://doi.org/10.3390/antiox10050769>
14. S.H. Aladaileh, M.H. Abukhalil, S.A.M. Saghir, H. Hanieh, M.A. Alfwuaires, A.A. Almaiman, M. Bin-Jumah and A.M. Mahmoud, *Biomolecules*, **9**, 346 (2019); <https://doi.org/10.3390/biom9080346>
15. Y. Su, L. Shen, J. Xue, J. Zou, D. Wan and Z. Shi, *Electron. J. Biotechnol.*, **53**, 8 (2021); <https://doi.org/10.1016/j.ejbt.2021.05.005>
16. X. Zhang, Y. Deng, J. Xiang, H. Liu, J. Zhang, J. Liao, K. Chen, B. Liu, J. Liu and Y. Pu, *Drug Des. Devel. Ther.*, **14**, 3393 (2020); <https://doi.org/10.2147/DDDT.S258187>
17. L. Fang, H. Zhang, J. Zhou, Y. Geng and X. Wang, *J. Anal. Methods Chem.*, **2018**, 3158293 (2018); <https://doi.org/10.1155/2018/3158293>
18. M. Zheng, Z. Peng, J. Li, L. Lin, S. Peng and X. Huang, Optimization of Extraction of Galangin from Galangal by Response Surface Method, Proceedings of the 2016 6th International Conference on Machinery, Materials, Environment, Biotechnology and Computer (MMEBC), p. 663 (2016).
19. L. Tao, Z.T. Wang, E.Y. Zhu, Y.H. Lu and D.Z. Wei, *S. Afr. J. Bot.*, **72**, 163 (2006); <https://doi.org/10.1016/j.sajb.2005.06.007>
20. L. Yang, Q.H. Yan, J.Y. Ma, Q. Wang, J.W. Zhang and G.X. Xi, *Trop. J. Pharm. Res.*, **12**, 771 (2013); <https://doi.org/10.4314/tjpr.v12i5.17>
21. O. Bobis, I.A. Marghitas, D. Dezmiorean, O. Morar, V. Bonta and F. Chirila, *Bull. Univ. Agric. Sci. Vet. Med. Cluj-Napoca Anim. Sci. Biotechnol.*, **67**, 85 (2010).
22. J. Dobes, O. Zitka, J. Sochor, B. Ruttkay-Nedecky, P. Babula, M. Beklova, J. Kynicky, J. Hubalek, B. Klejdus, R. Kizek and V. Adam, *Int. J. Electrochem. Sci.*, **8**, 4520 (2013).
23. A.D. Mülazimoglu and I.E. Mülazimoglu, *Food Anal. Methods*, **6**, 845 (2013); <https://doi.org/10.1007/s12161-012-9492-5>
24. K.M. Kasiotis, P. Anastasiadou, A. Papadopoulos and K. Machera, *PLoS One*, **12**, e0170077 (2017); <https://doi.org/10.1371/journal.pone.0170077>
25. A.K. Pandey, A.K. Mishra and A. Mishra, *Cell. Mol. Biol.*, **58**, 142 (2012).
26. K.E. Heim, A.R. Tagliaferro and D.J. Bobilya, *J. Nutr. Biochem.*, **13**, 572 (2002); [https://doi.org/10.1016/S0955-2863\(02\)00208-5](https://doi.org/10.1016/S0955-2863(02)00208-5)
27. G. Cao, E. Sofic and R.L. Prior, *Free Radic. Biol. Med.*, **22**, 749 (1997); [https://doi.org/10.1016/S0891-5849\(96\)00351-6](https://doi.org/10.1016/S0891-5849(96)00351-6)
28. J. Treml and K. Šmejkal, *Compr. Rev. Food Sci. Food Saf.*, **15**, 720 (2016); <https://doi.org/10.1111/1541-4337.12204>
29. O. Firuzi, A. Lacanna, R. Petrucci, G. Marrosu and L. Saso, *Biochim. Biophys. Acta, Gen. Subj.*, **1721**, 174 (2005); <https://doi.org/10.1016/j.bbagen.2004.11.001>
30. A.A. Aloud, C. Veeramani, C. Govindasamy, M.A. Alsaif, A.S. El Newehy and K.S. Al-Numair, *Redox Rep.*, **22**, 290 (2017); <https://doi.org/10.1080/13510002.2016.1273437>
31. G.M. Sulaiman, *Food Sci. Biotechnol.*, **25**, 247 (2016); <https://doi.org/10.1007/s10068-016-0036-4>
32. Q. Liu, X. Li, X. Ouyang and D. Chen, *Molecules*, **23**, 3225 (2018); <https://doi.org/10.3390/molecules23123225>
33. S.A. Divakaran, P.S. Hema, M.S. Nair and C.K.K. Nair, *Int. J. Radiat. Res.*, **11**, 81 (2013).
34. A. Mandas, E.L. Iorio, M.G. Congiu, C. Balestrieri, A. Mereu, D. Cau, S. Dessì and N. Curreli, *J. Biomed. Biotechnol.*, **2009**, 1 (2009); <https://doi.org/10.1155/2009/749575>
35. M.R. Ahn, K. Kunimasa, S. Kumazawa, T. Nakayama, K. Kaji, Y. Uto, H. Hori, H. Nagasawa and T. Ohta, *Mol. Nutr. Food Res.*, **53**, 643 (2009); <https://doi.org/10.1002/mnfr.200800021>
36. V. Curti, V. Zaccaria, A. Tsetegho Sokeng, M. Dacrema, I. Masiello, A. Mascaro, G. D'Antona and M. Daglia, *Int. J. Mol. Sci.*, **20**, 1250 (2019); <https://doi.org/10.3390/ijms20051250>
37. M.S. Hernández Zarate M. del R. Abraham Juárez, A. Cerón García, C. Ozuna López, A. J. Gutiérrez Chávez, J. de J. N. Segoviano Garfias, F. Avila Ramos, *Food Sci. Technol.*, **38**, 210 (2018); <https://doi.org/10.1590/fst.29916>
38. D.K. Patel, K. Patel, M. Gadewar and V. Tahilyani, *Asian Pac. J. Trop. Biomed.*, **2**, 449 (2012); [https://doi.org/10.1016/S2221-1691\(12\)60205-6](https://doi.org/10.1016/S2221-1691(12)60205-6)
39. A.S. Sivakumar and C.V. Anuradha, *Chem. Biol. Interact.*, **193**, 141 (2011); <https://doi.org/10.1016/j.cbi.2011.06.003>
40. X. Li, Y. Wang, Y. Xiong, J. Wu, H. Ding, X. Chen, L. Lan and H. Zhang, *Sci. Rep.*, **6**, 30496 (2016); <https://doi.org/10.1038/srep30496>
41. S.-T. Chien, M.-D. Shi, Y.-C. Lee, C.-C. Te and Y.-W. Shih, *Cancer Cell Int.*, **15**, 15 (2015); <https://doi.org/10.1186/s12935-015-0168-2>
42. M. Nishita, S.-Y. Park, T. Nishio, K. Kamizaki, Z.C. Wang, K. Tamada, T. Takumi, R. Hashimoto, H. Otani, G.J. Pazour, V.W. Hsu and Y. Minami, *Sci. Rep.*, **7**, 1 (2017); <https://doi.org/10.1038/s41598-016-0028-x>
43. H. Yao, H. Lu, J. Zhang, X. Xue, C. Yin, J. Hu, R. Zou, L. Wang and H. Xu, *J. Nanomater.*, **2019**, 7236895 (2019); <https://doi.org/10.1155/2019/7236895>
44. S. Sabry, A. El hakim Ramadan, M. Abd elghany, T. Okda and A. Hasan, *J. Drug Deliv. Sci. Technol.*, **61**, 102163 (2021); <https://doi.org/10.1016/j.jddst.2020.102163>
45. S. Kumar and K.R. Alagawadi, *Pharm. Biol.*, **51**, 607 (2013); <https://doi.org/10.3109/13880209.2012.757327>
46. M.B. Hursthouse, D.S. Hughes, T. Gelbrich and T.L. Threlfall, *Chem. Cent. J.*, **9**, 1 (2015); <https://doi.org/10.1186/s13065-014-0076-x>
47. A.A. Aloud, V. Chinnadurai, C. Govindasamy, M.A. Alsaif and K.S. Al-Numair, *Pharm. Biol.*, **56**, 302 (2018); <https://doi.org/10.1080/13880209.2018.1474931>
48. T.P.T. Cushnie and A.J. Lamb, *J. Ethnopharmacol.*, **101**, 243 (2005); <https://doi.org/10.1016/j.jep.2005.04.014>
49. T.P.T. Cushnie and A.J. Lamb, *Phytomedicine*, **13**, 187 (2006); <https://doi.org/10.1016/j.phymed.2004.07.003>
50. T.P.T. Cushnie, V.E.S. Hamilton, D.G. Chapman, P.W. Taylor and A.J. Lamb, *J. Appl. Microbiol.*, **103**, 1562 (2007); <https://doi.org/10.1111/j.1365-2672.2007.03393.x>
51. G. Eumkeb, S. Sakdarat and S. Siriwong, *Phytomedicine*, **18**, 40 (2010); <https://doi.org/10.1016/j.phymed.2010.09.003>
52. G. Eumkeb, S. Siriwong, S. Phitaktim, N. Rojtinakorn and S. Sakdarat, *J. Appl. Microbiol.*, **112**, 55 (2012); <https://doi.org/10.1111/j.1365-2672.2011.05190.x>
53. J. Ouyang, F. Sun, W. Feng, Y. Xie, L. Ren and Y. Chen, *Chemotherapy*, **63**, 20 (2018); <https://doi.org/10.1159/000481658>
54. R. Campana, V. Patrone, I.T.M. Franzini, G. Diamantini, E. Vittoria and W. Baffone, *J. Med. Food*, **12**, 1050 (2009); <https://doi.org/10.1089/jmf.2008.0173>
55. M.J. Salvador, F.T. Sartori, A.C.B.C. Sacilotto, E.M.F. Pral, S.C. Alfieri and W. Vichniewski, *Z. Naturforsch.*, **64C**, 509 (2018); <https://doi.org/10.1515/znc-2009-7-807>
56. J. Echeverría, J. Opazo, L. Mendoza, A. Urzúa and M. Wilkens, *Molecules*, **22**, 608 (2017); <https://doi.org/10.3390/molecules22040608>
57. Y.C. Jung, M.E. Kim, J.H. Yoon, P.R. Park, H.Y. Youn, H.W. Lee and J.S. Lee, *Immunopharmacol. Immunotoxicol.*, **36**, 426 (2014); <https://doi.org/10.3109/08923973.2014.968257>
58. M.J. Choi, J.S. Park, J.E. Park, H.S. Kim and H.S. Kim, *Biomol. Ther.*, **25**, 641 (2017); <https://doi.org/10.4062/biomolther.2017.173>

59. Y.C. Huang, M.S. Tsai, P.C. Hsieh, J.H. Shih, T.S. Wang, Y.C. Wang, T.H. Lin and S.H. Wang, *Toxicol. Appl. Pharmacol.*, **329**, 128 (2017); <https://doi.org/10.1016/j.taap.2017.05.034>
60. H.N. Lee, S.A. Shin, G.S. Choo, H.J. Kim, Y.S. Park, B.S. Kim, S.K. Kim, S.D. Cho, J.S. Nam, C.S. Choi, J.H. Che, B.K. Park and J.Y. Jung, *Int. J. Mol. Med.*, **41**, 888 (2018); <https://doi.org/10.3892/ijmm.2017.3296>
61. H. Lu, H. Yao, R. Zou, X. Chen and H. Xu, *BioMed Res. Int.*, **2019**, 3018357 (2019); <https://doi.org/10.1155/2019/3018357>
62. C.-C. Yang, L.-D. Hsiao and C.-M. Yang, *J. Inflamm. Res.*, **13**, 945 (2020); <https://doi.org/10.2147/JIR.S276925>
63. J. Fan, X.H. Zhao, J.R. Zhao and B.R. Li, *ACS Omega*, **6**, 15046 (2021); <https://doi.org/10.1021/acsomega.1c01167>
64. W. Ye, W. Sun, R. Chen, Z. Wang, X. Cui, H. Zhang, S. Qian, Q. Zheng, Y. Zhou, J. Wan, J. Xu, X. Wang and Y. Zhou, *Acta Chromatogr.*, **31**, 120 (2019); <https://doi.org/10.1556/1326.2017.00389>
65. X. Jia, Y. Du, J. Xu and Y. Dong, *Trop. J. Pharm. Res.*, **19**, 651 (2020); <https://doi.org/10.4314/tjpr.v19i3.28>
66. Y.L. Ma, F. Zhao, J.T. Yin, C.J. Liang, X.L. Niu, Z.H. Qiu and L.T. Zhang, *Molecules*, **24**, 1171 (2019); <https://doi.org/10.3390/molecules24061171>
67. R. Liu, H. Li, N. Wei and Y. Tan, *PeerJ*, **9**, e11041 (2021); <https://doi.org/10.7717/peerj.11041>