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Section A: Natural Products & Metabolomics

Myricetin: Unleashing Nature's Anticancer Warrior for A Healthier Tomorrow

Muhammad Raza Naqvi[1], Basim. S. A. Al-Sulivany[2]*, Muhammad Yasir Naeem[3], Zeliha Selamoglu[4,5,6], Hamdia Yousif Issa[2]

[1]*Medical Director (Hematologist/Oncologist) and Cancer Liason Physician Intermountain Health Care, Denver, CO, USA*
[2]*Department of Biology, College of Science, University of Zakho, Zakho, Duhok, Iraq*
[3]*Department of Agronomy, Animals, Food, Natural Resources and the Environment (DAFNAE), University of Padova, Italy*
[4]*Department of Medical Biology, Medicine Faculty, Nigde Omer Halisdemir University, Nigde, Türkiye*
[5]*Western Caspian University, Baku, Azerbaijan*
[6]*Khoja Akhmet Yassawi International Kazakh-Turkish University, Faculty of Sciences, Department of Biology, Central Campus, Turkestan, Kazakhstan*

***Corresponding author:** *Basim. S. A. Al-Sulivany, Department of Biology, College of Science, University of Zakho, Zakho, Duhok, Iraq. Tel. + 009647504509701*
E-mail address: basim.ahmed@uoz.edu.krd

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ABSTRACT

Good nutrition is essential for a balanced lifestyle and effectively addresses nutritional disturbances. Nutraceuticals, particularly flavonoids found in plant-derived foods, show diverse medical advantages, particularly anti-inflammatory and antimicrobial, and anti-cancer properties. Myricetin, a flavonol found in fruits and vegetables, is notable due the prospect of nutraceutical benefit and cancer-protective properties. Cancer is a major worldwide health problem, and existing treatments can lead to difficulties. Although breakthroughs in cancer therapy, it's still an important factor of mortality worldwide, with high expenditures and unpleasant consequences. Antioxidant-rich medicinal plants and biologically active compounds have the potential to avoid and treat diseases. To overcome these challenges, the search foralternative anticancer drugs is crucial. Herbal medicines, particularly flavonoids, significantly influence cellular and molecular mechanisms and channels associated with Cancer initiation and metastasis. Myricetin, a dietary flavanol, exhibits notable pharmacological activities, especially in the realm of anticancer effects against various human cancers. Myricetin, a polyhydroxy flavonol, emerges as a promising candidate, in cancer prevention is evident through various mechanisms, making it a subject of extensive research. This review provides a most recent record of research findings on myricetin's anticancer potential,emphasizing its multifaceted contributions, safety considerations, optimal doses for various types of cancer, and implications in experimental studies. Additionally, difficulties such as bioavailability improvement through nano-formulations and the synthesis of derivatives for further exploration are discussed. The evidence gathered underscores myricetin's potential as asynergistic agent with existing anticancer drugs and offers valuable insights for researchers inthe field.

Keywords*: Anticancer, Apoptosis, flavonoids, Myricetin, Tumor, Nutraceuticals.*

INTRODUCTION

Cancer, an intricate and multifaceted health challenge, has risen as a significant global concern, contributing significantly to annual fatalities worldwide[1]. Recent data underscores the staggering number of over 19.3 million newly diagnosed cancer cases,resulting in nearly 10 million reported deaths[2]. Despite the advancement of diverse treatment strategies, cancer persists as a major cause of global mortality[3]. However, the current essential treatments for cancer patients remain crucial, they often bring about severe side effects, including intense nausea and vomiting[4].

Within this context, about half of recognized cancer therapeutic agents are sourced from natural products, with medicinal plant metabolites acknowledged for their valuable role in anti-cancerand chemo-preventive compounds[5,6]. Holistic medicines, particularly those produced from medicinal plants, have gained popularity in cancer treatment because of their cellular susceptibility and low cytotoxicity to normal cells[7]. Consuming organic foods or bioactive chemicals may reduce the risk of cancer and other pathogenic disorders. It is reported that natural chemicals and bioactive substitutes can inhibit cancer progression by regulating biological processes[8].

Nutraceuticals are used to control cancer, diabetes, cardiovascular disease, and other developmental disorders[9]. Nutraceuticals, a modern kind of complementary and alternative medicine (CAM), which have shown promising results in the 21st century, these therapies are typically advised as a supplement to established chemotherapy treatments for cancer management[9]. Due to challenges like “cancer cell susceptibility”, toxicity, and adverse effects linked to synthetic chemotherapeutic agents, concerted efforts are underway to explore whether nutraceuticals have a detrimental impact on cancer cell growth[10,11]. From a chemical perspective, a nutritional supplement is categorized into diverse categories, encompassing derivatives of isoprenoids, phenolic compounds, carbohydrate derivatives, fatty acids and structural lipids, and amino acid derivatives[12].

Flavonoids, crucial plant secondary metabolites, contribute to the distinct characteristics of plant-derived foods and beverages, including color and taste. Synthesized in specific plant locations, they influence flower color, and aroma, and play roles in seed germination and fruit dispersion. Found in all plant components, flavonoids are diverse, with over 6,000 identified variants, classified into subgroups like anthocyanins, flavonols, flavones, and isoflavones. Structurally, they consist of 15 carbon atoms and three rings (A, B, C). Their functions in plants include UV protection, defence against phytopathogens, signal modulation, male fertility, auxin transport,and serving as visual signals for pollinators[12,13].

Myricetin is a naturally occurring hexahydroxy flavone distinguished by hydroxy groups at positions 3, 3′, 4′, 5, 5′, and 7. This compound is highly prevalent in edible products, showcasing antioxidant, anticancer, antidiabetic, and anti-inflammatory properties. Myricetin has a variety of functions, namely the capacity to cause apoptosis in cancer cells, operate as a Cox-1 inhibitor, antineoplastic and antioxidant agent, and a hypoglycemic agent. Myricetin, which typically occurs in the glycoside form (O-glycosides), is abundant in fruits, vegetables, berries, nuts, herbs, plants, drinks, and medicinal substances, indicating its importance as a vital flavonoid[14].

Chemistry of Myricetin

Myricetin ($C_{15}H_{10}O_8$) is a hexahydroxy flavone with hydroxy groups at positions 3, 3′, 4′, 5, 5′, and 7. Its molecular structure is comparable to other flavonoids such as fisetin [$C_{15}H_{10}O_6$], luteolin [$C_{15}H_{10}O_6$], and quercetin [$C_{15}H_{10}O_7$] (Park et al., 2016). Myricetin appears as a yellow powder with a molecular weight of 318.23 g/mol. Myricetin in ethanol absorbs UV at λ_{max} 255 and at λ_{max} nm 375[15].

Myricetin’s Ability to Combat Cancer

Breast Cancer

The current investigation aimed to examine how myricetin affects breast cancer cells (MCF-7) and its apoptotic processes. Myricetin dramatically enhanced the ratio of BAX to Bcl-2 and the proliferation of BRCA, p53, and GADD45 genes. Apoptosis genes caspase-3, caspase-8, and caspase-9 were shown to be significantly upregulated. Myricetin triggered apoptosis in breast cancer cells by activating both intrinsic and extrinsic pathways, including the BRCA1-GADD45 pathway[22]. Another study found that myricetin increased the generation of hydrogen peroxide (H_2O_2) in triple-negative breast cancer cells, resulting in substantial effects, myricetin's cytotoxicity was reduced in triple-negative breast cancer cells by inhibiting both intracellular and extracellular ROS production and accumulation in addition myricetin's lethal effect on those cells were linked to oxidative stress caused by extracellular H_2O_2 generation[23]

Myricetin improves antioxidant concentrations in plasma, breast tissue, and erythrocyte lysate, reducing oxidative harm that results from 12-dimethylbenzanthracene (DMBA). Ingestion of myricetin at dosages of 50, 100, and 200 mg/kg yielded equivalent outcomes to conventional vincristine and control groups, moreover its performance was found that similarly or better than vincristine in all tested parameters[24].

Table 1. Reported Myricetin Origins and Quantities in Various Fruits and Vegetables

Plant Source	Myricetin Concentration(mg/kg or mg/g)	References
Purslane	3 – 58	16
Bambara groundnut	1800 (mg/g)	17
Lycium barbarum L. fruits	57.2 (mg/g)	18
Blueberry	25	19
Crowberry	44	19
Green chili	11.5	20
Red chili	29.5	20
Garlic	693	20
Guava	549.5	20
Carrot	525.3	21
Spinach	1660.9	21
Turnip	457.0	21
Cauliflower	1586.9	21
Peas	146.2	21

The levels are provided in milligrams per kilogramme (mg.kg-1), even though otherwise noted

Myricetin's anticancer properties were proven in human breast cancer cells (SK-BR-3). Raising myricetin dosages led to decreased survival of cells, higher levels of apoptosis, and production of apoptotic bodies. Furthermore, Bcl-2 levels decreased, whereas cleaved PARP and Bax proteins rose. The investigation explored the correlation between cell viability and autophagy in myricetin-treated cells. The results showed that treating cells with 3-methyladenine (3-MA) and myricetin at the same time increased apoptosis. Inhibition of JNK reduced cell viability, increased Bax expression, and reduced the levels of p-JNK, Bcl-2, and LC3 II/I.

Myricetin promotes mortality in T47D breast cancer cells by activating each internal and external pathway[17]. An *in vivo* investigation found that myricetin effectively inhibits metastasis by reducing ST6GALNAC5 mRNA levels and MMP2/9 activity in MDAMB-231BR cells[18]. Myricetin is known for its cytotoxic effect on cancer cells, indicating that it might be a feasible choice for breast cancer therapy[25].

Lung Cancer

A new study found that myricetin inhibits IFN-induced PD-L1 expression in human lung cancer cells. This resulted in reduced IDO1 expression and kynurenine production. Myricetin prevented lung cancer cells exposed to IFN from negatively impacting Jurkat-PD-1 T cells' survival, proliferation, CD69 expression, and interleukin-2 production. It was found[26] that targeting and suppressing the JAK-STAT-IRF1 pathway disrupted the mechanism responsible for IFN-induced upregulation of PD-L1 and IDO1. Microscopic study of A549 and NCI-H446 cells treated with myricetin revealed cellular enlargement and vacuolated membranes. Transmission electron microscopy detected many holes in the cell membranes of myricetin-treated A549 and NCI-H446 cells, signifying pyroptosis. It was also found that myricetin causes pyro-ptosis in NCI-H446 and A549 cells via GSDME cleavage, not GSDMD[27].

Myricetin's cytotoxic consequences were tested in A549 and A549-IR cells. A549-IR cells showed low cytotoxicity after 48 hours of myricetin administration, however cell migration was inhibited in a dose-dependent manner. Myricetin administration did not significantly affect E-cadherin expression in A549-IR cells. At a dosage of 100 µM, myricetin reduced slug, MMP2, MMP9, and vimentin levels while enhancing E-cadherin expression[28]. *In vitro*, myricetin combined with radiation reduced cell survival and proliferation, increased Caspase-3 protein expression, and promoted apoptosis in lung cancer A549 and H1299 cells. Also, myricetin treatment reduced tumour xenografts development in radiation-treated mice[29]. Total flavonoids from pine needles of *Cedrus deodara*, which contains myricetin, has been employed in cancer therapy due to their ability to induce apoptosis in lung cancer cells. *Ardisia insular* leaves, also containing myricetin, have demonstrated efficacy against A549[30]. Furthermore, a novel myricetin derivative with alkyl, methyl, and hydroxyalkyl substitutions isolated from *Mimosa pudica* exhibited anticancer activity against the

A549 cell line[31]. In radiated mice, myricetin dramatically slowed the development process of lung tumour xenografts[32].

Prostate Cancer

Worldwide, prostate cancer is the next most prevalent cancer among males with over 1.2 millionnew cases and nearly 359,000 deaths reported annually, following lung cancer[33]. Myricetin has demonstrated its ability to impede the growth of prostate cancer by influencing different pathways of cell signalling. Flow cytometry assessment revealed a significant induction of apoptosis by myricetin in the cell of prostatic cancer, while reduced rates of apoptosis were observed in RWPE-1 cells, as indicated by cytotoxicity assays. Western-Blotting analysis further confirmed the upregulation of apoptosis-related proteins, including cleaved caspase-3 and cleaved caspase-9, in PC3 and DU145 cells following this naturally plant active constituent treatment. Notably, this plant-derived compound also inhibited the phosphorylation of ERK1/2 and AKT in these prostate cancer cells[34]. A critical recent study[34], highlighted myricetin as a potent α-ketoglutarate-type inhibitor, significantly reducing the growth of androgen-dependent and independent castration-resistant prostate cancer (CRPC) by inhibiting demethylation activity through KDM4s (C4-2B and CWR22Rv1). Combining myricetin and enzalutamide exhibited a synergistic cytotoxic effect on C4-2B cells. In a C4-2B xenograft model, the co-administration of PLGA- myricetin, enzalutamide, and the combination treatment showed superior antitumor potential compared to the control group.

Myricetin at 25 mg/kg reduced PC3 subcutaneous xenografts in nude mice, showing in a threefold decrease in the average tumor volume compared to the control group by day 48. Myricetin was reported[35] to inhibit prostate cancer development and epithelial-mesenchymal transition in a PC3-injected xenograft nude mouse model. Research of 58,279 males investigated the link between flavonoid intake (catechin, epicatechin, kaempferol, and myricetin), black tea use, and prostate cancer risk, where those who consume more flavonoids and black tea had a lower chance of developing stage III/IV or stage IV prostate cancer[36].

The comprehensive update in Appendix 1 encompasses recent studies on myricetin in the context of prostate cancer[36]. These encouraging results offer preliminary evidence supporting the prospective use of this plant-derived substance as a potential chemo-prophylactic and therapeutic agent, highlighting its role in precision medicine for targeting prostate cancer[37].

Liver Cancer

Hepatic cancer, specifically hepatocellular carcinoma (HCC), ranks as the 6th most prevalent and the 4th causes of cancer-related deaths globally[33]. Myricetin, a natural compound, is acknowledged for its potential role in preventing and treating liver cancer by influencing various cell-signaling molecules. The study aimed to understand the mechanisms by which myricetin hinders cell growth, and proliferation, and induces apoptosis in HCC cells.The findings indicated that myricetin effectively lowered the growth of Huh-7 and HepG2 cells while significantly increasing apoptosis rates, particularly in a time-dependent manner. Cleaved caspase3 levels were notably increased in myricetin- administered HCC cells. The compound inhibited YAP expression by promoting its phosphorylation and subsequent degradation, activating LATS1/2 kinase. Myricetin also hindered the migration and invasion of MHCC97H cells in a concentration-dependent manner, altering cell morphology andmodulating N-cadherin and E-cadherin expression. Furthermore, myricetin treatment significantly reduced the viability of HCC cells in a dose-dependent manner. Investigation into the involvement of MARCH 1 in the anti-HCC effect revealed a reduction in MARCH 1 expression in Hep3B and HepG2 cells, with MARCH 1 over expression partially offsetting myricetin-induced down-regulation and diminishing the antitumor effect. Interestingly, myricetin increased MARCH 1 mRNA levels in Hep3B cells whereas, reduced them in HepG2cells[22,38].

Gastric Cancer

To assess the effects of this natural drugs on AGS gastric cancer cell viability, the MTT assaywas conducted, revealing a gradual lowering in cell survival with gradual increasing the dosesof this drug (95.8% at 5 µM, 90.3% at 10 µM, 80.6% at 15 µM, and 64.6%, 52.7%, and 36.3% at 20 µM, 25 µM, and 30 µM, respectively). This decline showed a dose-dependent reduction in the viability of gastric cancer cells compared to the control group. Myricetin exhibited its inhibitory effects on the PI3K/Akt/mTOR pathway, inducing apoptosis and autophagy and thereby reducing the survival rate of gastric cancer cells. Cancer proliferation was suppressed in an *in vivo* investigation[39]. Flow cytometry analysis in another study demonstrated that this natural drug induced the program cell death in the cells of gastric cancer.Western blotting confirmed the influence of myricetin on apoptosis and cell cycle arrest by regulating associated proteins[40].

Skin Cancer

In JB6 P+ cells from mouse skin epidermis, myricetin inhibits the expression of Cox-2 inducedby UVB. The treatment with myricetin not only prevents UVB-induced NF-κB activation andactivator protein-1 activation in a dose-dependent manner but also inhibits

Fyn kinase activity, reducing UVB-induced MAPK phosphorylation. *In vivo* studies on mouse skin confirm that myricetin directly inhibits Fyn kinase activity, subsequently reducing UVB-induced expression of Cox-2. Findings from a mouse skin tumor study show that pre-treatment with myricetin notably and dose-dependently decreases the occurrence of UVB-induced skin tumors. This indicates that myricetin has strong chemo preventive properties, primarily by targeting Fyn in skin cancer development[41].

Brain Cancer

This medication inhibited the development of glioma cells (U251) in humans while also affecting ROS generation. Myricetin therapy inhibited the development of human glioma cells (U251) in a dosage and time-dependent manner. This medication caused cells to detach and form clusters that migrated within the media. Myricetin therapy led to increased apoptotic cell death, including both early and late apoptotic cells. It was found that[42] myricetin inhibited the cell cycle during the G2/M phase. In another investigation, myricetin, TRAIL (tumor necrosis factor-related apoptosis-inducing ligand), or a combination of these drugs were used to treat human astrocytes and glioblastoma cells. Subtoxic dosages of myricetin coupled with TRAIL induced rapid death in glioma cells. The combination of medicines did not alter human astrocytes. Co-treatment with myricetin and TRAIL increased activation of effector caspases-3/-7 and initiator caspases-8/-9[43].

Myricetin's anticancer ability was tested in A431 skin cancer cell lines and found effective against them. Myricetin's anticancer activities were linked to ROS-induced mitochondrial membrane potential and apoptosis changes. Myricetin administration altered Bcl-2 and Bax expressions, produced cell cycle arrest in A431 cells, and inhibited migration and invasion. It was suggested that[44] myricetin might be a viable lead chemical for developing a successful skin cancer treatment approach. Micelles containing myricetin can trigger apoptosis in mice by regulating the expression of apoptotic proteins such as Bcl-2, BAD, and BAX[45].

Bone Cancer

Myricetin enhances osteogenic stem cells by stimulating the mitogen-activated protein kinase signalling pathway[46]. Myricetin has been shown to boost both innate and adaptive immune responses, making immunomodulatory medicines more effective for bone cancer patients receiving chemotherapy. Myricetin has been shown to promote osteogenic development in human periodontal ligament stem cells via the BMP-2/Smad and ERK/JNK/p38 signalling pathways, indicating therapeutic potential for bone cancer[47].

Esophageal Cancer

The study found that combining myricetin with 5-fluorouracil (5-FU) substantially decreased clonogenic survival in EC9706 cells compared to 5-FU alone. Both 5-FU and this medication increased the percentage of cells in the G0/G1 phase, preventing entrance into the S phase. When coupled, myricetin dramatically increases the proportion of cells in the G0/G1 phase, indicating that it improves 5-FU chemosensitivity through cell cycle arrest[47,48].

Eye Cancer

Luteolin, apigenin, myricetin, and quercetin are successful treatments for retinal illnesses, according to reports. Research examined how flavonoids such as epigallocatechin gallate, luteolin, apigenin, myricetin, quercetin, and cyanidin affect the physiological characteristics and vitality of human retinal pigment epithelial (RPE) cells. The study found that flavonoids, excluding epigallocatechin gallate, reduced RPE cell proliferation, migration, and vascular endothelial growth factor production in a dose-dependent manner. Lutein, apigenin, myricetin, and quercetin may be effective cancer treatments for retinal illnesses[33,49].

The Intriguing Mechanism of Myricetin's Action

Myricetin has anti-cancer properties via affecting many cell signaling molecules and pathways. This covers the control of inflammation, apoptosis, cell cycle, PI3K/Akt, angiogenesis, transcription factors/components, and other substances or molecules. The next sections go into detail on the putative processes by which myricetin prevents cancer[49].

Inflammatory Response

It was found[29] a strong link between viral and bacterial infections, chronic inflammation, and a significant number of human malignancies. Lipopolysaccharide (LPS) is commonly employed in experiments to cause inflammation and play a role in chronic intestinal inflammation, which can contribute to cancer progression. The LPS/TLR4 signal transduction pathway interacts with several metabolic processes, including bile acid production and secretion, the renin-angiotensin system, arachidonic acid pathways, and glutathione metabolism. These connections significantly contribute to chronic intestinal inflammation and carcinogenesis[50].

Macrophages and other leukocytes generate RNS and ROS to fight infections[51]. Persistent tissue breakdown and cell proliferation can extend the existence of infection-fighting chemicals, causing negative consequences. Mutagenic agents, such as peroxy-nitrite, cause DNA mutations in growing epithelial and stromal cells. The release of TNF-α and macrophage migration inhibitory factor by macrophages

and T-lymphocytes can worsen DNA damage and accelerate cancer progression[32,52,53].

Apoptosis

Apoptosis, a planned cell death process, is vital for maintaining physiological homeostasis. Disrupting this pathway can lead to pathological changes and the development of cancers. Controlling and inducing apoptosis is a potential technique for treating cancer. Natural substances have been shown to trigger apoptosis in cancer cells, potentially helping to cancer prevention[54].

Myricetin, a natural compound, has shown apoptotic potential in numerous cancer types. The study found that exposure to myricetin caused the development of apoptotic structures and sacs on the cell membrane. As myricetin concentrations increased, so did the fraction of cells undergoing apoptosis, reaching 28.5% and 67.4% at 50 and 100 µmol/L. Flow cytometry revealed a favorable connection between apoptotic rates and myricetin concentrations (50 and 100 µmol/L). Myricetin-induced apoptosis decreased the Bcl-2/Bax ratio, indicating a dose-dependent effect[3,54].

Pure contents have been shown to effectively regulate the cell cycle, which is crucial for tumor therapy and management. Myricetin was studied for its effect on cell cycle progression in hepatocellular carcinoma (HCC). Myricetin treatment of HepG2 and Hep3B cells (0, 25, 50 µM) led to a reduction in cell population in the G0/G1 stage and stoppage of the cell cycle in the G2/M stage. The concentration-dependent decrease in proliferative nuclei revealed that myricetin inhibits cancer cell proliferation, possibly by stopping the cell cycle during the G2/M phase[38,54]. Moreover, myricetin-exposed cells (100 µM) showed a higher percentage of S-phase cells (17.70 ± 7.66%) compared to untreated cells (10.42 ± 2.95%)[34,37,40,55].

Angiogenesis is critical for the onset and advancement of numerous pathogenic diseases. Natural substances have anticancer characteristics because they impede angiogenesis. Myricetin significantly inhibited UVB-induced VEGF production in SKH-1 hairless mouse skin[56,57.]

In a choriocarcinoma investigation, myricetin administration reduced the pro-angiogenic and invasive activities of malignant JAR and JEG-3 trophoblast cells via the PI3K/AKT and MAPK signaling cascades. Myricetin therapy significantly reduced cell invasion by 90% in JAR and JEG-3 cells as it was found that[58] myricetin lowered the levels of VEGFA in JAR cells' media with conditioning by 40%.

PI3K/AKT Pathways

The PI3K/AKT/mTOR signaling system is linked to cancer growth and metastasis through genetic mutations, amplifications, RTK overactivation, and tumor suppressor loss. Natural chemicals have been identified as possible cancer prevention agents by targeting this pathway. Myricetin reduced Zn cell growth and triggered autophagy by inhibiting the PI3K/Akt/mTOR signaling pathway. Myricetin inhibits p-PI3K, p-Akt, and p-mTOR expression, leading to decreased cancer cell survival, improved autophagy, and apoptosis[59,60].

Myricetin inhibits the PI3K/Akt/mTOR pathway in gastric cancer cells, resulting in lower expression of p-PI3K, p-Akt, and p-mTOR in a concentration-dependent manner. This inhibition suppressed cancer cell proliferation, promoted autophagy, and induced apoptosis[61]. Myricetin has been shown in studies to reduce phosphorylated p-Akt, p-MAPK, and p-P38 levels, indicating its potential to modulate apoptosis through ROS induction, inhibit cell migration and tube formation, and impact the PI3K/Akt/mTOR signaling in human umbilical vascular endothelial cells[44,61,62].

Autophagy

Autophagy, a cellular mechanism, may protect cancer cells from nutrition deprivation while also destroying them, affecting energy balance. Myricetin, a natural substance, has been shown to modulate autophagy. Myricetin treatment in gastric cancer cells led to an increase in vacuoles and autolysosomes, indicating increased autophagic activity as it was found that[3] treatment groups had significantly higher levels of LC3-II/LC3-I and beclin1, an autophagy-related protein.

A similar study was undertaken to see if myricetin triggered autophagy in HCC cells. Myricetin appears to stimulate autophagy, as evidenced by a dose-dependent increase in the percentage of cells generating GFP-LC3 puncta Western blot analysis research has shown a dose-dependent decrease in p62 protein levels and an increase in the LC3-II/LC3-I ratio[45,62,63,64].

Limitations and Future Perspectives

Myricetin has been recognized for its health advantages and function in cancer care, although there is inadequate evidence to support its full therapeutic significance. Myricetin's fast digestion and elimination from the body, as well as its difficulty to dissolve in water, are crucial characteristics. There are limitations to its utility in treating illnesses, notably cancer. Due to its poor bioavailability, its potential utility in cancer therapy and prevention is still being researched. Limited translational research is needed to discover optimal delivery routes and doses for diverse cancers[11], exacerbating treatment challenges. Recent clinical trials seek a link the difference between laboratory research and direct clinical usage is now being investigated. Investigators are investigating the health benefits of flavonoids, specifically myricetin, as antineoplastic drugs. Several clinical studies. Nano-formulation-based

flavonoids are increasingly being used to target certain types of tumors, decreasing off-target effects and improving medication effectiveness. However, this research is still in the preclinical phase.

Further clinical trials are needed to completely understand myricetin's mechanism of action, safety profile, and effective dose. Addressing existing issues entails developing alternative nano-formulations of myricetin to address difficulties including inadequate bioavailability, limited loading capacity, targeted administration, and premature release. Understanding the production of myricetin derivatives is vital for evaluating their potential as anticancer medicines.

CONCLUSION

Our food choices and our susceptibility to infections and overall health are directly impacted by our food choices. Among the leading causes of death and disability, cancer stands out. While various cancer treatments exist, they often come with long-term adverse effects. The current demand is for targeted therapies to mitigate the drawbacks of existing treatments. Natural therapies, with their insignificant negative impact on cells in health, are being considered as alternative cancer treatments.

Plant-derived compounds, particularly flavonoids, play a significant role in influencing the cellular and molecular processes and pathways associated with cancer growth and progression. Myricetin, a dietary flavanol, exhibits notable pharmacological activities, especially in the realm of anticancer effects against various human cancers. Numerous *in-vitro*, *in-vivo*, and clinical studies underscore the potential of myricetin in treating cancers affecting various organs such as the breast, lung, prostate, colorectal, skin, bladder, blood, pancreas, thyroid, liver, bone, brain, cervical, and eye.

Despite advancements in cancer treatment, it remains a major global cause of death, often associated with high costs and adverse effects. Medicinal plants and bioactive compounds that are abundant in antioxidants present promising avenues for disease prevention and treatment. In this context, myricetin, a type of flavonoid, has emerged as a significant contributor to the management of health. Its well-documented properties that safeguard the liver, combat inflammation, protect the nervous system, and shield the heart make it a pivotal compound. Furthermore, myricetin has demonstrated remarkable potential in the prevention of cancer through the inhibition of tumor growth, suppression of angiogenesis, regulation of the cell cycle, reduction of inflammation, induction of apoptosis, and modulation of various cellular signaling pathways.

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Conflicts of interest

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