Letters in Applied NanoBioScience
Open-Access Journal (ISSN: 2284-6808)

Review
Volume 15, Issue 2, 2026, 72
https://doi.org/10.33263/LIANBS152.072

Unraveling the Pharmacological Potential of Myricetin: From Therapeutic Applications to Recent Trends in Nanoformulations

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Received: 22.04.2025; Accepted: 21.02.2026; Published: 30.06.2026

Abstract: The flavonoids are natural compounds with a polyphenolic structure that are generated from plant metabolites and are frequently present in fruits, vegetables, and some beverages. Myricetin (MYR) has the IUPAC name 3, 5, 7, 3′, 4′, 5′-hexahydroxyflavonol and belongs to the polyhydroxyflavonol. The compound was originally isolated from the bark of the tree *Myrica rubra*. This review elucidates the uses and principal mechanisms of MYR in anticancer, antidiabetic, anti-inflammatory, antioxidant, anti-Alzheimer's, cardioprotective, and antiepileptic actions. MYR has low solubility, permeability, and bioavailability, which can be addressed through the development of nanotechnology-based formulations that increase surface area, enhance dissolution rates, and facilitate transport across biological barriers. This review provides extensive updates on MYR-loaded nanoformulations such as silver nanoparticles, starch nanoparticles, solid lipid nanoparticles, gold nanoparticles, bovine serum albumin nanoparticles, nanomicelles, liposomes, polymeric micelles, and magnetic nanoparticles. The published patents highlighted the therapeutic applications of MYR for a range of conditions, including oesophageal diseases, viral infections, facial vascularity, bacterial infections, anti-senile dementia activity, Covid-19, coronal pneumonia, as well as anti-cholinesterase and anti-enzyme activities.

Keywords: myricetin; flavonoids; solid lipid nanoparticles, liposomes, nanomicelles; nanotechnology.

1. Introduction

Herbal drugs have always aroused the curiosity of scientists, and a wide range of research is underway to identify their therapeutic potential. Alkaloids, tannins, terpenoids, flavonoids, lignans, curcumins, saponins, phenolics, and glucosides are all regarded as secondary metabolites and are the most prevalent naturally occurring ingredients [1,2]. Flavonoids are secondary metabolites present in plants, characterized by two benzene rings connected by an oxygen-containing pyran ring in a C6-C3-C6 structure. According to alterations in their key structure, these can be classified into flavanols, flavanones, flavonols, isoflavones, flavones, and anthocyanins [3]. Flavonoids are found in many natural sources, including fruits, vegetables, flowers, cereals, bark, roots, stems, and wine [4]. Flavonoids exhibit significant pharmacological potential; however, their low bioavailability limits clinical

applicability, which is directly linked to their intestinal absorption and metabolism due to short gastric residence time, as well as their restricted permeability and solubility within the gastrointestinal tract [5].

Myricetin (MYR) is often extracted from the bark of the *Myrica rubra* tree [6,7]. It is frequently found in berries, fruits, vegetables, honey, red wine, tea, and other daily foods. It is also widely distributed in natural plants belonging to various families, including Myricaceae, Vitaceae, Leguminosae, Primulaceae, Rosaceae, Ericaceae, Fagaceae, and Compositae [8]. MYR has a history of more than hundreds of years and is also known as hydroxyquerctein (because of its structural similarities with other phenolic compounds), a compound under flavonoids called flavonols was basically first discovered as a yellowish needle crystal and first identified in the late 18th century from the bark of *Myrica negi* from the family of *Myricaceae*. MYR is often extracted from the bark of the *Myrica rubra* tree and has been prevalent in other plant families such as *Anacardiacae*, *Pinaceae*, *Myricaceae*, *Anacardiaceae*, *Polygonaceae*, *Pinaceae*, and *Primulaceae* [6-8]. The empirical formula of MYR is $C_{15}H_{10}O_8$ with a molecular weight of 318.23 g/mol [7,9]. It exists in the form of a crystalline solid having a melting point of 368°C. Its partition coefficient is 2.76 ± 0.05 and has a λmax at two different wavelengths of 369 nm and 328 nm. MYR has potential anti-oxidant effect, anti-inflammatory [9], anti-diabetic [10], and anti-cancer activity [11-13]. MYR has potent free radical scavenging ability with wide applications in cancer, diabetes, thrombosis, oxidative stress, and hypercholesterolemia [14]. MYR functions to inhibit DNA polymerases, reverse transcriptase, telomerase, kinase, and helicase activities. It also demonstrates an impact on the central nervous system and may be efficacious against Alzheimer's and Parkinson's disorders [15]. However, MYR has an apparent permeability coefficient (Papp) of 1.7 x 10^6 cm/s, indicating moderate membrane permeability [16]. The study revealed that MYR exhibits poor oral bioavailability primarily as a result of limited absorption. MYR showed T_{max} and C_{max} of 6.4 hours and 1488.75±200.78 ng/mL, which might be due to its poor solubility in water [17-19]. However, MYR has limited solubility, permeability, and bioavailability challenges, which can be mitigated through the advancement of nanotechnology-based formulations. These nanoformulations operate by augmenting surface area, improving dissolution rates, and facilitating transport across biological barriers [20-22].

The scope of this review is to highlight applications and key mechanisms of MYR in anticancer, antidiabetic, antioxidant, anti-inflammatory, anti-Alzheimer's, cardioprotective, and antiepileptic activities. This review provides comprehensive updates on MYR-loaded nanoformulations, including silver nanoparticles, starch nanoparticles, solid lipid nanoparticles, gold nanoparticles, bovine serum albumin nanoparticles, nanomicelles, liposomes, polymeric micelles, and magnetic nanoparticles. For these purposes, an inclusive literature search was conducted on PubMed, Google Scholar, and ScienceDirect databases. A literature review was performed utilizing articles published in peer-reviewed journals from 2000 to 2025. The literature was examined utilizing various combinations of multiple keywords, including 'myricetin', 'anticancer', 'antidiabetic', 'anti-inflammatory', 'antioxidant', 'anti-alzheimer', 'anti-epileptic', 'cardioprotective', 'silver nanoparticles', 'starch nanoparticles', 'solid lipid nanoparticles', 'gold nanoparticles', 'bovine serum albumin nanoparticles', 'nanomicelles', 'liposomes', 'polymeric micelles', and 'magnetic nanoparticles', adhering to defined inclusion and exclusion criteria. The scientific papers incorporated were characterized by the following criteria: (i) written in English, (ii) sourced from the previously indicated key search, and (iii) providing adequate information. Papers authored in any language apart from

English, as well as letters to the editor and editorials, were removed from the search strategy. This article also reviews patent-related information from the World Intellectual Property Organization regarding the pharmacological potential of MYR.

2. Pharmacological Potential of Myricetin

The studies revealed that MYR have numerous therapeutic applications, including cardioprotective, anti-allergic, anti-inflammatory, anticancer, anti-diabetic, antioxidant, anti-neurodegenerative, anti-Alzheimer, and anti-Parkinson activities [10,23-28]. Figure 1 describes the key aspects of the mechanism of action of various pharmacological activities of MYR. Details of several investigations related to the abovementioned activities conducted over the last few years are described further in this review.

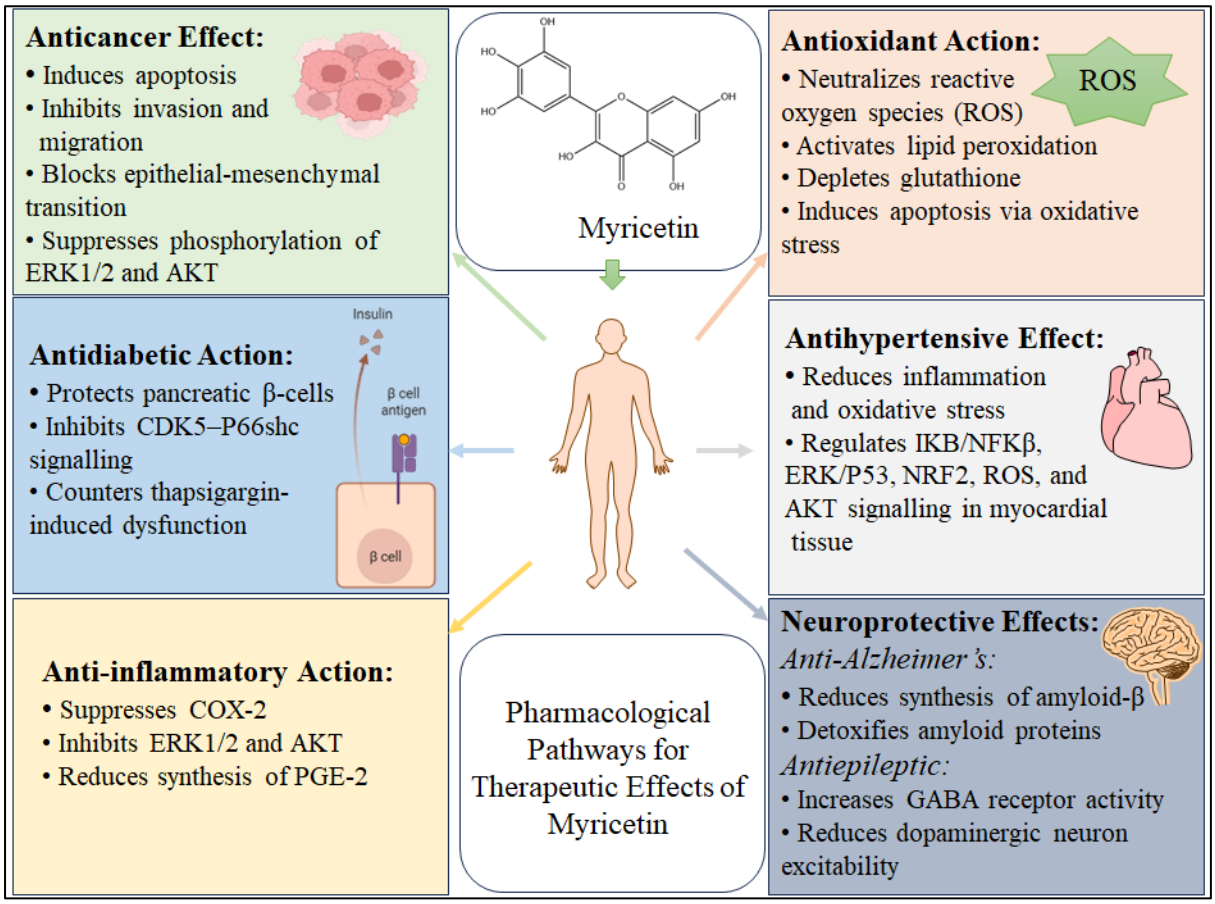

Figure 1. The depiction of pharmacological applications of MYR and their mechanism of action.

2.1. Anti-cancer activity.

An unwanted or uncontrollable effect of a gene, called cancer, is caused by uncontrolled cell growth [29]. MYR imparts chemopreventive effects in various tumors by cell proliferation, apoptosis, angiogenesis, and metastasis. MYR inhibits the growth of cancer cells into neoplastic tissue by interacting with the Akt and JAK1-STAT3 pathways. By competing with adenosine triphosphate and suppressing Akt expression, it primarily targets epidermal growth factor-induced cell transformation, which involves the Akt signaling pathway [30]. Recent studies showed that MYR reduced invasion in A549 cells by inhibiting matrix metalloproteinase 2 (MMP-2) and urokinase-type plasminogen activator (u-PA) activity [31]. The transcription factor nuclear factor kappa B (NF-κB), which binds to activator protein-1,

helps stimulate metastatic growth. It was observed that MYR therapy prevented NF-κB and activator protein-1 from binding to DNA [32]. In a research study, MYR demonstrated anticancer potential, with different cancer cell lines exhibiting different IC_{50} values. For example, HeLa and T47D cells showed more cytotoxicity than normal Vero cells, with IC_{50} values of 22.70 μg/mL and 51.43 μg/mL, respectively [33].

2.2. Anti-diabetic activity.

Hyperglycemia, a key characteristic of diabetes mellitus (DM), is a condition marked by elevated blood glucose levels resulting from insulin resistance, insufficient insulin production, or excessive glucagon secretion [10]. MYR increased Nrf2 expression and its downstream genes, and reduced DM-induced kidney impairment, fibrosis, and oxidative damage. MYR decreased DM-induced kidney impairment and fibrosis when Nrf2 was knocked down; this effect may be related to suppression of I-κB/NFκ-B and p65 signaling pathway [34,35]. MYR inhibits the mitochondrial-dependent apoptotic pathway, thereby partially limiting the apoptosis induced by high glucose in β-cells by reducing endoplasmic reticulum stress. MYR helps prevent β-cell apoptosis by subsequent overexpression of pancreatic duodenal homeobox-1 and sarcoendoplasmic reticulum calcium ATPase 2b, driven by cyclin-dependent kinase 5 inactivation [36]. In a study, treatment with MYR at doses between 100 and 200 mg/kg significantly improved HDL-cholesterol levels, whereas doses below 100 mg/kg markedly reduced them [37].

2.3. Anti-oxidant activity.

Antioxidants have a protective role against reactive oxygen species (ROS), which protect the body from damage caused by oxidants [38]. MYR has the ability to neutralize ROS and bind to the intracellular transition metal ions that cause ROS. *In vitro* studies revealed that MYR dramatically enhanced glutathione S-transferase activity. It was discovered that MYR can neutralize free radicals produced by enzymatic or non-enzymatic processes [39]. It has been shown that MYR inhibited ascorbate-stimulated malondialdehyde production in rat liver [34]. It significantly prevented the lipid peroxidation in mitochondria caused by ascorbate and ferrous sulfate [40].

2.4. Anti-inflammatory activity.

The inflammatory process involves multiple steps, including the activation of enzymes, the release of mediators, fluid extravasation, cell movement, tissue disintegration, and repair. MYR has a tendency to reduce edema induced by xylene, a property called antiphlogistic [24]. This has been found effective against periodontitis, an infectious inflammatory disease that affects the supporting bone and connective tissue around the teeth. MYR has the potential to suppress lipoteichoic acid-induced cyclooxygenase-2 expression and activation of extracellular signal-related kinase-1/2 (ERK-1/2), Akt, and p38 in human gingival fibroblasts. It also prevented the breakdown of IκB and the production and synthesis of Prostaglandin E2 [41,42]. MYR may successfully inhibit the exudative phase of acute inflammation and has a dual effect on the inflammatory response of host cells and the periodontal pathogenic bacteria, namely Porphyromonas gingivalis [43].

2.5. Anti-Alzheimer activity.

Alzheimer's disease is a neurodegenerative condition that impairs cognition and behavior, and symptoms worsen over time, with a clinical course of 8–10 years, in which age and genes are the main genetic contributors. The most typical symptom to appear is memory loss [44]. The effects of MYR on the neurotoxicity caused by the amyloid β peptide, a substance found in senile plaques in the brains of people with Alzheimer's disease. MYR has the potential to reduce amyloid β peptide synthesis and its detoxification, thereby slowing the progression of disease [45]. A research study using the intracerebroventricular streptozotocin-induced rat model of Alzheimer's disease showed that MYR at a dose of 10 mg/kg administered intraperitoneally significantly increased the number of hippocampal CA3 pyramidal neurons, thereby improving learning and memory deficits [46].

2.6. Anti-epileptic activity.

Epilepsy is a prevalent and serious neurological condition that encompasses various subtypes, each defined by unique electroclinical patterns, imaging results, and genetic characteristics [47]. MYR therapy can reduce pentylenetetrazole-induced increase in the number of apoptotic cells and apoptotic protein expression. According to the current study's findings, MYR may have protective benefits through regulating the molecular pathways involved in epileptogenesis. MYR therapy resulted in the expression of glutamic acid decarboxylase and the gamma-aminobutyric acid receptor, as well as the restoration of their equilibrium ratio and reduced dopaminergic neurons [48].

2.7. Cardioprotective activity.

Cardiovascular diseases encompass a wide range of conditions that affect the heart muscle and the blood vessels that deliver oxygen and nutrients to the heart, brain, and other essential organs [49]. MYR showed cardioprotective properties against isoproterenol-induced myocardial infarction, hypertension, ischemia/reperfusion injury, and lipopolysaccharide [23]. In both serum and cardiac tissue, MYR dramatically decreased the generation of inflammatory cytokines. MYR lowers oxidative stress by controlling the IB/NF-κB, ERK/P53, Nrf2/HemeOxygenase-1, ROS, and Akt signaling pathway in myocardial damage [50]. Through its capacity to reduce infarct size and cardiomyocyte apoptosis, MYR therapy has been shown to confer benefits. It possesses cardiovascular protective properties against ischemia/reperfusion-induced myocardial damage [51]. Table 1 recapitulates the dosage regimen, including dose and duration, route of administration, and research inference from various investigations of MYR conducted in previous years using different animal models.

Table 1. An outline of the pharmacological relevance of MYR has been previously investigated in various animal models.

Dosage regimen [Route of administration]	Research outcomes	Animal model	Ref
Anticancer			
5 mg/kg per day 5 times [i.p.]	The study revealed that MYR can induce apoptosis in T24 cells in the G2/M phase by altering the expression of G2/M regulatory proteins. After 12 hours, MYR treatment reduced cell viability at concentrations of 20–100 μM in a range of 2.6%–61%, whereas after 24 and 48 hours, the reductions were 2.9%–70% and 3%–80%, respectively.	Mice	[52]

Dosage regimen [Route of administration]	Research outcomes	Animal model	Ref
25 and 50 mg/kg once a day for 2 weeks [i.p.]	The study demonstrated decreased expression of vascular endothelial growth factor and reduced p38 MAPK phosphorylation, along with reduced angiogenesis.	Mice	[53]
30 mg/kg in the MIA PaCa-2 model and 50 mg/kg in the S2-013 model [i.p.]	MYR induced apoptosis in pancreatic cancer cells and inhibited the PI-3 kinase pathway, leading to cell death. In the MIA PaCa-2 model, 57% of the mice in the MYR group experienced local tumor spread, compared with 80% in the control group. The S2-013 model with similar outcomes was 54.5% of mice that received MYR treatment, 100% of animals in the control group had local tumor spread.	Mice	[54]
Cardioprotective			
100 mg/kg for 7 days [p.o.]	The overexpression of inducible nitric oxide synthase was decreased, and reduced oxidoreductase (superoxide dismutase and glutathione peroxidase) and attenuated lipopolysaccharide-induced cardiac injury. Around 67% of the nuclei were p-P65 positive after 12 hours of LPS stimulation, and post-MYR administration, the ratio declined to about 17%.	Mice	[50]
100 and 300 mg/kg once a day for 21 days [p.o.]	MYR demonstrated the potential to mitigate the cardiotoxic effects caused by isoproterenol (ISO) and exhibited considerable therapeutic value in the prophylactic treatment of myocardial infarction in male Wistar rats subjected to ISO-induced myocardial infarction. Pre-administration of MYR at 100 mg/kg and 300 mg/kg (p.o.) over 21 days resulted in a significant reduction in ISO-induced changes in heart rate.	Rats	[55]
200 mg/kg/day for 6 weeks [Gastric needle]	MYR decreased the neonatal rat cardiomyocyte hypertrophy induced by phenylephrine and pressure overload-induced pathological hypertrophy by increasing Nrf2 activity and preventing the TAK1/P38/JNK1/2 phosphorylation through controlling Traf6 ubiquitination.	Mice	[56]
Neuroprotective			
50 mg/kg MYR 60 minutes for 21 consecutive days for 4 hours/day [i.p.] (before repeated restraint stress)	MYR reduced immobility duration in mice, increased glutathione peroxidase activity in the hippocampus of stressed mice, decreased plasma corticosterone levels, and restored diminished BDNF levels.	Mice	[57]
Anti-diabetic			
3 mg/12 hours [i.p.]	In diabetic rats, hyperglycemia decreased by 50%, and the hypertriglyceridemia commonly associated with diabetes was normalized. It elevated the hepatic glycogen synthase I activity, but total glycogen synthase and phosphorylase-alpha activity remained unaffected.	Rats	[58]
6 mg/day every 12 hours for 10 days [i.p.]	MYR administration improved altered renal functions and restored renal activities of glutathione peroxidase and xanthine oxidase, suggesting that MYR may have therapeutic potential in diabetic nephropathy. It also significantly reduced glomerulosclerosis and lowered blood urea nitrogen levels.	Rats	[59]
MYR (100 mg/ kg) [p.o]	MYR had the potential to manage hyperglycemia in people with diabetes mellitus, in part by inhibition of β-glucosidase activity. The inhibitory effect of MYR was observed after 7 weeks of consumption at 101.5 mg/kg on fasting hyperglycemia and maltase activity, comparable to the results with acarbose at 39.0 mg/kg.	Rats	[60]
Antiepileptic			
100 mg/kg body weight for 26 days, once a day. [Oral]	MYR demonstrated preventive properties by decreasing the intensity of seizures and controlling glutamic acid decarboxylase 65-kilodalton/γ-Aminobutyric acid type A and BDNF-TrkB pathways	Mice	[48]
Anti-inflammatory			
5, 50, 150 mg/kg, twice a day for 14 days [p.o]	MYR-3-O-b-D-glucuronide inhibited the COX and 5-LOX, with an IC50 value of 0.5 µM for COX-1, 10 µM for COX-2, and 0.1 µM for 5-LOX in intact cells.	Rats	[61]
5 mg/kg [intracerebroventricular]	MYR inhibited the COX-1 by 79%, COX-2 by 88%, and TNF-α by 70%.	Rats	[62]
[(57)	MYR protective properties were linked to its ability to suppress NF-κB/A and p38/MAPK signalling pathways.	Mice	[63]

Dosage regimen [Route of administration]	Research outcomes	Animal model	Ref
10, 20, and 40 mg/kg [i.p.]	After MYR treatment, IL-6 and TNF-α levels were increased by 27.5 and 29.6 times ($p < 0.01$).	Rats	[64]

3. Nanotechnology: A Boon for Drug Delivery of Myricetin.

MYR has inadequate water solubility and limited absolute bioavailability, which complicate its development as a medicinal formulation. Therefore, to fully exploit the therapeutic advantages of MYR in a clinical context, an alternative oral formulation with enhanced solubility and bioavailability is necessary [65]. Nanoformulations such as solid lipid nanoparticles, starch nanoparticles, silver nanoparticles, gold nanoparticles, bovine serum albumin nanoparticles, liposomes, nanomicelles, polymeric micelles, and magnetic nanoparticles have been well documented for their potential to overcome the low oral bioavailability of insoluble medicines [66]. To achieve the drug's site-specific therapeutic action at an appropriate rate and dose regimen, the key aim of creating nanoparticles as a delivery vehicle is to control particle size, surface composition, and release profile [67-70]. Figure 2 depicts the structures of the various types of nanoformulations explored for MYR drug delivery. Table 2 summarizes the comparative features of nanoparticles, specifically with respect to their composition, size range, biocompatibility, stability, and applications [71-77].

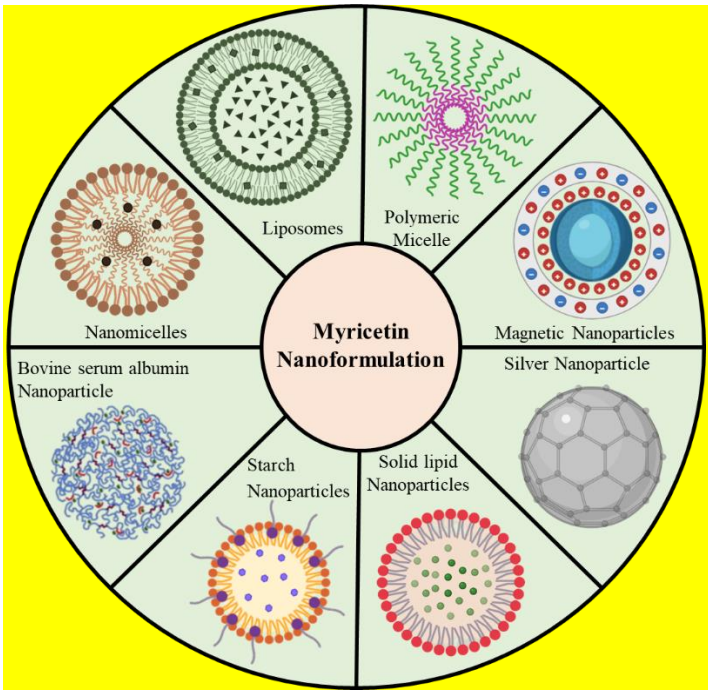

Figure 2. Various types of nanoformulations have been explored for the drug delivery of MYR.

Table 2. Comparison of various types of nanoparticles with reference to composition, size range, biocompatibility, stability, and common applications.

Nanoparticle type	Composition (size range)	Biocompatibility	Stability	Common applications
Silver nanoparticles	Metallic silver (10-100 nm)	Moderate	High	Antibacterial, anticancer, and wound healing
Gold nanoparticles	Metallic gold (1-100 nm)	High	Very High	Cancer therapy, diagnostics, and imaging
Starch nanoparticles	Natural polysaccharide (starch) (50-200 nm)	Excellent	Moderate	Controlled drug delivery, oral delivery
Solid lipid nanoparticles	Lipid matrix (solid at room temp) (50-1000 nm)	High	Moderate to High	Topical, oral, anticancer, anti-inflammatory
Bovine serum albumin NPs	Protein (BSA) (100-300 nm)	Excellent	Moderate	Drug delivery, gene delivery, diagnostics

Nanoparticle type	Composition (size range)	Biocompatibility	Stability	Common applications
Nanomicelles	Amphiphilic surfactants/polymers (10-100 nm)	High	Good in aqueous solution	Poorly soluble drug delivery, cancer therapy
Liposomes	Phospholipid bilayers (50-1000 nm)	Excellent	Moderate	Cancer, antifungal, vaccine delivery
Polymeric Micelles	Amphiphilic block copolymers (10-100 nm)	High	High	Cancer, ocular, oral, poorly soluble drugs
Magnetic Nanoparticles	Iron oxide (10-100 nm)	Good (with coating)	High	Magnetic resonance imaging contrast, hyperthermia, and targeted drug delivery

3.1. Silver nanoparticles (AgNPs).

AgNPs range in size from 1 to 100 nm and can be synthesized using methods such as physical vapor condensation, pyrolysis, non-enzymatic reduction, green synthesis, chemical reduction, microemulsion, and microwave-assisted synthesis. Biologically produced AgNPs exhibit high yields, high solubility, and stability [78-81]. According to a study, AgNPs exhibit a biocidal effect by slowly releasing Ag+. Furthermore, AgNPs are a prime candidate for antibacterial application due to their huge surface area, which promotes pathogen reactivity and sorption [82]. AgNPs have a significant impact on medical research and have long been recognized for their strong bactericidal and inhibitory effects, with a wide range of antibacterial activity [83].

3.2. Starch nanoparticles.

Starch is one of the most prevalent forms of biomass in nature and a common energy source, as many plants retain it because it is a natural, biocompatible, renewable, and biodegradable polymer [84]. Starch appears to be an excellent substrate for the creation of nanoparticles. The linear amylose and branching amylopectin are the two molecules that make up the glucose-based carbohydrate polymer. In the granules, they are arranged in alternating crystalline and amorphous lamellae [85-87]. Microfluidization, a technology that combines complex formation and enzymatic hydrolysis procedures, is used to produce starch nanoparticles, which can be used as carriers for drugs and biofunctional materials [88]. Starch nanocrystals and nanoparticle derivatives are increasingly being adopted for drug delivery platforms due to their improved absorption and biocompatibility [89].

3.3. Solid lipid nanoparticles (SLNs).

SLNs having size ranges between 10 and 1000 nm have proved to be a successful drug delivery strategy for hydrophobic and hydrophilic medicines [90]. SLNs are colloidal carrier systems having a solid lipid core encased in aqueous surfactant. Lipid pellets for the administration of oral medications are a well-known example of the use of solid lipids as matrix materials for drug delivery [91]. With their excellent physical stability for targeted drug delivery and environment-sensitive drug protection, SLNs have been proven successful in the nutraceutical, cosmetic, pharmaceutical, and biomedical industries [92]. SLNs are prepared by methods such as homogenization, microemulsion, solvent evaporation, and spray drying techniques [93-95]. SLNs can be administered via topical, pulmonary, oral, parenteral, and ocular routes [96,97].

3.4. Gold nanoparticles.

Gold nanoparticles use their unique chemical and physical properties to load and unload drugs [98]. Recently, gold nanoparticles have emerged as a promising alternative for delivering drugs to their target sites [99-101]. The approaches, including physical, chemical, biological, γ-irradiation, seeding growth, and green synthesis, are used to create gold nanoparticles [102-104]. Gold nanoparticles have applications in DNA labeling, cancer therapy, drug delivery, and photothermal therapy [105]. To create functional nanoparticles that can cross biological membranes and target the nucleus, 20 nm gold nanoparticles have been coupled to several cellular targeting peptides [106].

3.5. Bovine serum albumin nanoparticles.

These nanoparticles possess non-immunogenic, biodegradable, and biocompatible properties and have emerged as promising nanocarriers [107,108]. A globular, water-soluble protein called bovine serum albumin tends to aggregate to form larger macromolecular complexes. The three-dimensional structure consists of three domains, each containing a secondary structure that is essentially a helix formed by six helices. Bovine serum albumin nanoparticles have recently attracted interest as carriers for hydrophobic medications and have been intensively studied as nanoscale drug-delivery systems. The nanoparticles prepared from bovine serum albumin are water-soluble, biodegradable, non-toxic, non-immunogenic, and metabolized *in vivo* to produce safe breakdown products [109,110]. Mainly, these are prepared by the desolvation crosslinking method and are effective for anticancer activity, targeted drug delivery, and stability enhancement [111,112].

3.6. Nanomicelles.

Amphiphilic molecules are generally used as constituents of supramolecular structures, including micelles and nanomicelles, due to their dual composition of hydrophilic and hydrophobic moieties. The various applications of nanomedicines include their use in cancer treatment, ocular drug delivery, and skin treatment [113-116]. By limiting direct interactions between medications and the in vivo environment, nanomicelles act as a protective shell, improving drug bioavailability and minimizing undesirable side effects [117].

3.7. Liposomes

A portion of the surrounding solvent can be trapped inside the spherical, closed structures known as liposomes, which are composed of lipid bilayers. The medical applications of liposomes include vaccination, gene therapy, antimicrobial therapy, cancer therapy, and brain-targeted drug delivery [118-123]. Pro-liposome is the most popular and economical technique because they are easily distributed, transferred, and measured in dry powder form [124]. When in contact with water, proliposomes can instantly transform into a liposomal suspension. Since proliposomes differ from conventional liposomes in that they may deliver therapeutic medications to tissues other than reticuloendothelial tissues, it is speculated that they serve as a controlled-release drug delivery system inside the vasculature [125-127]. Proliposomes are prepared using the film-deposition-on-carrier technique [128], spray drying [129], and the fluidized-bed method [130], and are used in parenteral, pulmonary, ocular, and oral drug delivery systems. The stability problems relating to liposomes have been greatly improved by pro-liposomes [131,132].

3.8. Polymeric micelles.

Solvophilic and solvophobic components make up polymeric micelles that are able to form above the critical micellar concentration. In aquatic environments, the solvophilic section provides the core, while the solvophobic section forms the shell, also known as the corona. These two constituents are covalently bonded to each other as either grafts or blocks [133]. Polymeric micelles are categorized into three primary groups, i.e., generated by hydrophobic interactions, electrostatic interactions, and metal complexation [134]. For weakly water-soluble anticancer medications, polymeric micelles constitute an efficient delivery mechanism with a small size (10-100 nm) and a hydrophilic PEG shell [135,136]. Polymeric micelles are excellent for a nanodrug delivery system because of several essential properties, including a hydrophilic coating around a drug-loading core, an appropriate size, and a biocompatible polymer [137-139].

3.9. Magnetic nanoparticles.

Magnetic nanoparticles are more biocompatible than hematite, cobalt ferrite, magnetite, and nickel ferrite. They are a great option for use in magnetic resonance imaging and biomedical applications [140-142]. These nanoparticles are synthesized by solvothermal, hydrothermal, microemulsion, sol-gel, co-precipitation, thermal decomposition, solution combustion, and microemulsion methods [143-145]. The current status of various nanoformulations synthesized by researchers to enhance the therapeutic efficacy of MYR in several disease conditions has been summarized in Table 3.

Table 3. An insight into various nanotechnology-based strategies explored for the drug delivery of MYR has been investigated in the last few decades.

Technique	Excipients	Research outcome	Diseases condition	Ref.
Silver nanoparticles				
Green Synthesis Method	Silver nitrate	Particles exhibited a size range from 20 to 50 nm. The results showed that the percentage of free radical scavenging ranged from 60 to 87% while MYR-AgNPs (MYR-loaded silver nanoparticles) demonstrated significant antibacterial activity against *Salmonella* and *Escherichia coli* with minimal inhibitory concentrations of 10^{-4} and 10^{-5} g/L.	Oxidative stress	[146]
Green Synthesis method	Silver nitrate	MYR effectively suppressed the growth of HCT116 cells in a dose-dependent manner. Showed that MYR effectively kills human colorectal cancer cell lines and becomes a suitable candidate for colorectal cancer by demonstrated 50% cell apoptosis below 200 µg/ml concentrations. The size range of NP's were 12–20 nm.	Colorectal cancer	[147]
Starch nanoparticles				
High speed-jet	Amylose starch, tapioca starch, lipid protein	A sustained release of loaded MY was seen in both simulated intestinal (pH 7.0) and gastric (pH 2.0) fluids. Starch nanoparticles enhanced the rate of clearance of DPPH free radicals and lowered the IC_{50} value. For pure MYR, the concentration was 18.27 µg/mL, but while using MYR starch nanoparticles, it was reduced to 14.42 µg/mL.	-	[148]
Solid Lipid nanoparticles				
Hot homogenization method	Polyxamer 407, Tween 80, sodium sulfite	Exhibited a particle size of less than 200 nm. Microencapsulation significantly decreased the degradation rate constant by 300-fold, sustained drug release (less than 50% released over 8 hours), and increased the half-life by 2.6 to 5.2 times that of free MYR.	-	[149]

Technique	Excipients	Research outcome	Diseases condition	Ref.
Ultrasonication/ homogenization method	Compritol 888 ATO, Stearoyl polyoxyl-32 glycerides, Tween80	Particle size and entrapment efficiency were 89.23±6.9 nm and 69.2%, respectively. They also reported a low IC50 (50% inhibitory concentration) value for inhibiting A549 cell growth in cytotoxicity assays. MYC-SLNs might cause A549 cells to upregulate necroptosis-associated genes, such as MLKL and RIPK3.	Lung adenocarcinoma	[150]
High shear homogenization and ultrasonication	Stearoyl polyoxyl-32 glycerides, PEG-8 hydrogenated coco-glycerides, glyceryl monostearate	These findings suggested that SLNs produced protective antioxidant activity against t-BHP-induced oxidative stress. The most effective defense against oxidative stress caused by t-BHP was provided by the EGCG: resveratrol combination.	Skin disorder	[151]
High-pressure homogenization	Lecithin, Chitosan, Tween 80	MYR-SLN-CS-FA suppressed angiogenesis and downregulated the production of embryonic growth factors. It decreased the expression of angiogenesis-related genes, such as VEGF and VEGF-R. Additionally, it decreased the levels of TNF-alpha and HER2 in tumor tissues, thereby reducing the inflammatory response. *In vitro* drug release of MYR from SLN was found 50% within 4 hours.	Anti-angiogenic activity	[152]
Gold nanoparticles				
Ultra-sonication-assisted	Hydrogen tetrachloroaurate	In breast cancer, MYR-AuNPs caused programmed cell death, Oxidative stress, and a reduction in mitochondrial membrane potential, resulting in a cellular viability of 69±1.57% at a concentration of 6.25 g mL-1, but 100 g mL^{-1} of pure MYR showed 64.24±2.58% of cellular viability.	Breast cancer and oxidative stress	[153]
Bovine serum albumin nanoparticles				
Desolvation cross-linking method	Glutaraldehyde and bovine serum albumin	Study revealed that MCF-7 cells' viability was significantly reduced after 24 hours, with an IC_{50} value of 72.45 g/mL at pH 5.4, and became a suitable approach for the treatment of breast cancer. FA-Myr-BSA nanoparticles can induce rapid release of Myr in an acidic environment (pH 5.4) and demonstrate significant biocompatibility in a physiological medium.	Cancer	[154]
Lipid nanoparticles				
O/w Emulsification technique	Tristearin and tricaprylin	MYR lipid nanoparticles demonstrated a CC50 value that was 1.5–2.5 times greater than MYR alone, which proved that MYR is more effective in encapsulated form than its free form. M-LNP (0.8 mg kg^{-1}) applied to the mouse footpad reduced sweating by 55%.	Focal sweating	[155]
Homogenization method	Miglyol, compritol 888, poloxamer 407	MYR-NLC lowered cell viability from 50 ± 2.3% to 40%±1.3%. The combination therapy of MYR and docetaxel in a compaction-based SLN was disclosed as a novel approach in chemotherapy for the treatment of different types of malignancies, including breast cancer. MYR-Compritol treatment of cancer cells for 24 hours resulted in 12.1±3.45% apoptosis and 12.04±0.7% G2/M phase arrest.	Breast cancer	[156]
Mesoporous silica nanoparticles				
Emulsification Method	Cetyltrimethylammonium bromide, tetraethylorthosilicate	The viability of lung cancer cell lines, namely A549 and NCI-H1299, was markedly reduced. These nanoparticles loaded with MYR and MRP-1 significantly enhanced the cleavage of Caspase-3 and PARP, thereby inducing apoptosis in lung cancer cells. Myr was released from Myr-MRP-1/MSN-FA nanoparticles in a more controlled manner compared to free Myr and Myr-MRP-1/MSN.	Lung cancer	[157]
Stöber method		UV-spectroscopy showed loading efficiencies of 43.24% and 22.23% for MYR and doxorubicin, respectively, with an entrapment efficiency of 88.97% and sustained drug release rates of 82% and 76%. MYR helps lower the cytotoxic effects of nanoparticles in healthy cells by reducing oxidative stress, thereby improving overall therapeutic efficacy. It works synergistically with doxorubicin, enabling a reduction	Oxidative stress	[158]

Technique	Excipients	Research outcome	Diseases condition	Ref.
		in the drug's dose by about 3.7 times due to a strong interaction.		
Nanomicelles				
Self-assembly method	Sodium hydroxide, chloral hydrate	Zeta potential of casein-MYR nanomicelles was [–(6.65±0.11) mV]. Small intestine absorption of casein-MYR nanomicelles was superior to MYR alone, with high solubility in water. When compared to 0 hours, the concentration of MYR increased by 0.36±0.27% after 1 hour and by 0.45±0.33% after 2 hours.	-	[159]
Liposomes				
Thin film technique	Chloroform, cholesterol	MYR has been demonstrated to be the strongest antioxidant at pH 5.4 at a concentration of 102 mol/mol phospholipid than quercetin and α-tocopherol	Oxidative stress	[160]
Emulsification-evaporation	Poloxamer 188, cholesterol, soya lecithin	The MYR-NLs exhibit stability, with a zeta potential of 15.49 ± 1.28% and 89.35 ± 3.18% for DLC and DLE, respectively. The downregulation of SIRT3 and phosphorylated p53 by MYR-NLs indicated that MYR-NLs prevented the development of glioblastoma multiforme cells *via* the mitochondrial, PI3K/Akt-ERK, and SIRT3/p53 pathways.	Glioblastoma	[161]
Thin film hydration method	Soybean lecithin, Sodium cholate, cholesterol	High encapsulation efficiency (>94%), drug loading capacity (>6.5%), and small particle size (<50 nm) revealed a considerable affinity between MRC and the nano pro-liposome core. The study showed that MYR loaded into TPGS-modified pro-liposomes enhanced oral bioavailability by 7.2-fold, resulting in improved therapeutic effectiveness.	Liver diseases	[162]
Micelles				
Thin film hydration method	Polyvinyl caprolactam-Polyvinyl acetate-Polyethylene glycol	At 30-, 60-, and 120-minute time intervals, Cou6-labeled MYR micelle showed 4.36, 2.54, and 2.54-fold greater ocular drug concentration in comparison to free Cou6 solution.	Ocular diseases and inflammatory	[163]
Thin film hydration method	Fetal bovine serum	The particle diameter and zeta potential were 51.5±13.2nm and 22.38±4.15mV, respectively, which results in better cellular absorption and anti-carcinogenic efficacy. At 24 hours, MYR-micelles at a dose of 4 mM reduced relative cell viability by 13%. In the treatment group, tumor volume was decreased by 33.5% compared to the control group after 14 days of 25 mg/kg therapy. Over 90% of MYR was released within 12 hours, with MYR-micelles showing a 3.27 times higher cellular uptake rate than free MYR at varying concentrations.	Glioblastoma cancer	[164]
Solvent evaporation method	Soyalecithin, polysorbate 80, Labrasol	The average diameter of micelle was 96.3 ± 42.7 nm, which showed that MYR-micelles penetrated the brain more than free MYR alone. MYR-loaded micelle showed bioavailability enhancement. MYR-micelles showed 1.5 times larger cytotoxicity than MYR at concentrations of 50-200 mM, with a Cmax of 28.84 pg/L after oral delivery, with AUC0-∞ values of 10.67 for MYR and 14.91 for micelles.	Glioblastoma cells	[165]
Magnetic nanoparticles				
Hydrothermal method	Sodium dodecyl iron chloride and manganese chloride	The study demonstrated that $MnFe_2O_4$NPs exhibited size stability of 4.53 times greater on the incorporation of MYR in magnetic nanoparticles. The observable size variations in formulations over the course of 112 days were absent.	-	[166]
Nanosuspension				
High-pressure homogenization	Soya lecithin and poloxamer 188	In an animal study using a rat model, the relative bioavailability of MYR-nanosuspensions stabilized with TPGS, soya lecithin, a combination of soya lecithin/TPGS, and a blend of HP-β-CD/TPG in rats were 2.44, 3.57, 1.61, and 2.96, respectively, and the solubility was 399.63, 367.15, 466.28, and 267.90 in mg/mL, respectively. In contrast, the solubility of plain MYR was 6.23 0.12 mg/ mL.	-	[65]

Technique	Excipients	Research outcome	Diseases condition	Ref.
Niosomes				
Pegylation	Non-ionic surfactant, PEG	MYR-pegylated niosomes were around 241 nm in size with a zeta potential of −32.7 ± 6.6 mV. These niosomes exhibited greater cytotoxicity against cancer cells than against normal cells, confirming their selective action and minimal harm to healthy cells.	Anticancer	[167]
Nanoemulsion				
Emulsification method	Capryol 90, Tween 20, Transcutol HP	Optimized MYR-nanoemulsion FB-2 demonstrated higher drug loading and sustained release compared to the MYR-DMSO suspension, with a CDR profile of 95.49±2.84 after 24 hours. *In vitro* drug release and cellular uptake of MYR were significantly enhanced in the MYR-NE formulation compared with MYR alone. This formulation showed more effectiveness in promoting oxidative stress and elevating tBax/Bcl-2 ratio, which led to marked reduction in cell viability and clonogenic potential, thereby accelerating cell death in TNBC cells.	Anticancer	[168]
Nanofibres				
-	2-Hydroxypropyl-β-cyclodextrin, PVP K120	The MYR:HPBCD:PVP formulations at different concentrations significantly improved the water solubility of MYR by 2665, 2787, and 2858 times, respectively. gMyNF exhibits lower cytotoxic effects on HaCaT keratinocytes compared to MYR. MyNF demonstrated photoprotective effects by minimizing cell death and suppressing excessive ROS generation. Due to its nanoscale particle size, MYR-nanofibres enhance skin absorption, which contributes to its improved antioxidant activity.	Antioxidant activity	[169]

4. Insight into Patent Status of Myricetin

A published patent literature search was conducted on the official website of the World Intellectual Property Organization to gain insight into novel therapeutic applications and compositions of MYR in recent years (Table 4). The published patents elucidate the therapeutic applications of MYR across a variety of conditions, including oesophageal diseases, viral infections, facial vascularity, bacterial infections, anti-senile dementia activity, Covid-19, coronal pneumonia, anti-cholinesterase activity, and anti-enzyme activity.

Table 4. Outline of recently published patents related to MYR-rich plant extracts or isolated MYR, highlighting their published title, patent number, applicants, and publication date.

Patent title	Patent number	Applicant	Publication date	Ref.
Composition for use in the prevention or treatment of oesophageal diseases linked to epithelial barrier defects	EP4110324	Thelial B V	04.01.2023	[170]
Method and composition for preventing and treating viral infections	US20220401403	Global BioLife Inc.	22.12.2022	[171]
AU, AG and rich phytochemical payload nanomaterials, antiviral/antibacterial products and synthesis methods	WO2022245578	The Curators of the University of Missouri	24.11.2022	[172]
Plant extracts for the treatment of increased facial vascularity	EP4081181	Oriflame Cosmetics AG	02.11.2022	[173]
Formulation, composition or foodstuff additives for the modification of glycemic response methods of manufacturing and using the same	EP4076011	Hoow Foods PTE LTD	26.10.2022	[174]
Anti-senile dementia activity and application of *Xanthoceras Sorbifolia* extract	CN115040583	Inner Mongolia University	13.09.2022	[175]

Patent title	Patent number	Applicant	Publication date	Ref.
Use of myricetin and dihydromyricetin phosphate compound in drug for preventing and treating covid-19	WO2022184102	Shanghai Institute of Materia Medica, Chinese Academy of Sciences, Wuhan Institute of Virology, Chinese Academy of Sciences	09.09.2022	[176]
Application of myricetin and dihydromyricetin phosphate compound in medicine for preventing and treating new coronavirus pneumonia.	CN114983993	Shanghai Institute of Materia Medica, Chinese Academy of Sciences. Wuhan Institute of Virology, Chinese Academy of Sciences	02.09.2022	[177]
Flavonoid compound with anti-cholinesterase and anti-enzyme dual activity and application thereof	CN114796192	North University of China, Shanxi Jingxi Biological Technology CO., LTD.	29.07.2022	[178]

5. Conclusion and Future Perspectives

MYR possesses the capacity to exhibit a wide array of therapeutic effects, including anticancer, antidiabetic, anti-inflammatory, antioxidant, anti-Alzheimer's, anti-epileptic, and cardioprotective activities, interacting with numerous potential targets associated with diverse illnesses. The primary limitations of MYR are its low water solubility, diminished gastrointestinal absorption, and limited bioavailability. Nanotechnology-based formulation techniques have been advantageous for MYR delivery and have demonstrated efficacy in mitigating these limitations. In recent decades, various nanotechnology-based systems, such as solid lipid nanoparticles, starch nanoparticles, silver nanoparticles, gold nanoparticles, bovine serum albumin nanoparticles, liposomes, nanomicelles, polymeric micelles, and magnetic nanoparticles, have shown significant potential to enhance the pharmacological activities of MYR. A significant number of investigations are currently underway in the preclinical stage, focusing on the formulation of nanoencapsulated MYR to improve its absorption and bioavailability. Nevertheless, considerable effort will be required to advance these formulations into clinical trials and ultimately toward human application. To overcome the constraints associated with MYR, including its inadequate bioavailability and loading capacity, it is imperative to advance various nanoformulations of this compound to enable targeted drug release. This review indicates significant opportunities for researchers to investigate the medicinal potential and nanotechnology-based formulations of MYR. The search strategy on the official clinical trials website revealed limited clinical trial data pertaining to MYR. Consequently, future studies on MYR should undergo additional clinical trials to validate its medicinal potential for human health.

Author Contributions

Conceptualization, N.S. and G.M.; investigation, S.S., P.K. and S.V.; writing—original draft preparation, N.S. and G.M.; writing—review and editing, S.S., P.K. and S.V.; visualization, S.S., P.K. and S.V. All authors have read and agreed to the published version of the manuscript.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

No new data were created or analyzed in this study. Data sharing is not applicable.

Funding

The current article did not receive any funding.

Acknowledgments

The authors would like to thank the Department of Pharmaceutics, MM College of Pharmacy, Maharishi Markandeshwar (Deemed to be University), Mullana-Ambala, Haryana, India 133207, for the completion of this review

Conflict of Interest

The authors declare no conflict of interest.

Abbreviations

The following abbreviations are used in this manuscript:

Abbreviation	Definition
MYR	Myricetin
NF-κb	Nuclear Factor Kappa B
SLNs	Solid Lipid Nanoparticles
ROS	Reactive Oxygen Species
TPGS	D-A-Tocopherol Polyethylene Glycol 1000 Succinate
DPPH	2,2-Diphenyl-1-Picrylhydrazyl: Extracellular
PI3	Phosphoinositide 3-Kinases /Phosphoinositide
ERK	Extracellular Signal-Regulated Kinase
Akt	Phosphoinositide 3-Kinases
SIRT1	Sirtuin 1
TNF-α	Tumor Necrosis Factor-Alpha
MAPK	Mitogen-activated protein kinase
COX	Cyclooxygenase
TAK1	Transforming Growth Factor-B-Activated Kinase 1
JNK1/2	Jun N-Terminal Protein Kinase

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