https://doi.org/10.15255/KUI.2025.017
KUI-7/2026
Review
Received March 30, 2025
Accepted June 2, 2025

# Therapeutic Bioactives in Medicinal and Edible Plants for Hyperuricemia Treatment

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**Abstract**

Hyperuricemia, a widespread metabolic disorder, is marked by abnormally high blood uric acid concentrations. This condition arises primarily from either excessive uric acid synthesis, impaired renal excretion, or a combination of both. Beyond its well-established link to gout, hyperuricemia is also a contributing factor to various other health complications. While conventional pharmacological treatments offer therapeutic benefits, their adverse side effects pose potential risks to patients. This review explores common medicinal and edible plants with demonstrated uric acid-lowering properties, emphasising their active constituents. These bioactive agents function through multiple mechanisms, such as suppressing uric acid synthesis, enhancing renal excretion, and reducing inflammatory responses. By evaluating the therapeutic potential of these plant-based compounds, this study aims to provide valuable insights into alternative strategies for hyperuricemia treatment.

**Keywords**

*Anti-gout, antioxidant, anti-inflammatory, uric acid, xanthine oxidase*

## 1 Introduction

Hyperuricemia is a medical condition characterised by elevated uric acid levels (approximately 6.8 mg $dl^{-1}$) in the blood.[1,2] This condition arises through three main mechanisms: increased endogenous production of uric acid, decreased renal excretion, or a combination of these pathologic processes.[3] Hyperuricemia may result from various factors, including a diet high in purines (*e.g.*, red meat and seafood), obesity, excessive alcohol consumption, certain diseases (such as gout, kidney disease, and psoriasis) and some medications.[4,5] Uric acid is the terminal metabolic by-product of purine catabolism —purines being organic compounds found in specific dietary sources and also formed by the body. The kidneys normally filter uric acid and eliminate it through urine. However, when there is an overproduction of uric acid or the kidneys cannot eliminate it efficiently, hyperuricemia may develop.[6–8] Hyperuricemia is often asymptomatic (approximately 75 to 90 %), which means that individuals with elevated uric acid levels may not experience noticeable symptoms.[9] Persistent elevation of uric acid concentrations in biological fluids (serum or urine) may precipitate various pathological manifestations. Most notably, this includes the crystallisation of monosodium urate deposits characteristic of gouty arthritis and the formation of uric acid renal calculi in nephrolithiasis. Furthermore, recent clinical evidence shows that there is a significant association between chronic hyperuricemia and multiple systemic disorders, including components of metabolic syndrome (insulin resistance, central adiposity), cardiovascular pathologies (hypertension, atherosclerotic disease), and progressive renal impairment.[10–12] The therapy for hyperuricemia involves various approaches, including lifestyle modifications, dietary changes, and medications.[13,14]

Urate-lowering therapy (ULT) represents the primary intervention for hyperuricemia[5,15], and it can be classified into three distinct mechanistic classes: xanthine oxidase inhibitors, uricosuric drugs, and recombinant uricase inhibitors. Although these treatments can effectively reduce uric acid levels, they may be associated with various adverse effects.[16–19] As a widely utilised xanthine oxidase inhibitor, allopurinol frequently causes gastrointestinal side effects, such as nausea and diarrheal symptoms, and in some cases, hypersensitivity reactions including skin rashes or severe systemic reactions.[20,21Taipei] In rare cases, allopurinol has been associated with liver and kidney damage.[22,23]

The limitations of current conventional urate-lowering therapies emphasise the urgent need to develop safer and more effective natural alternatives for the treatment of hyperuricemia. Herbal medicine, considered safe and effective in reducing uric acid levels, is commonly used in countries such as China, Indonesia, and Thailand to treat hyperuricemia. This review highlights edible plants and their bioactive components in the prevention and treatment of hyperuricemia.

## 2 Hyperuricemia

Uric acid, the terminal metabolic by-product of purine nucleotide catabolism, is produced in the liver. Purines are also produced within the body as part of normal cellular metabolism. Cells generate purines during processes like DNA and RNA synthesis, as well as the breakdown of ATP (adenosine triphosphate), a compound that helps in energy transfer within cells.[25,26] Purines undergo a metabolic process known as purine catabolism, where they are broken down into simpler compounds.[27]

The enzyme xanthine oxidoreductase mediates the terminal metabolic steps in uric acid biosynthesis, sequentially

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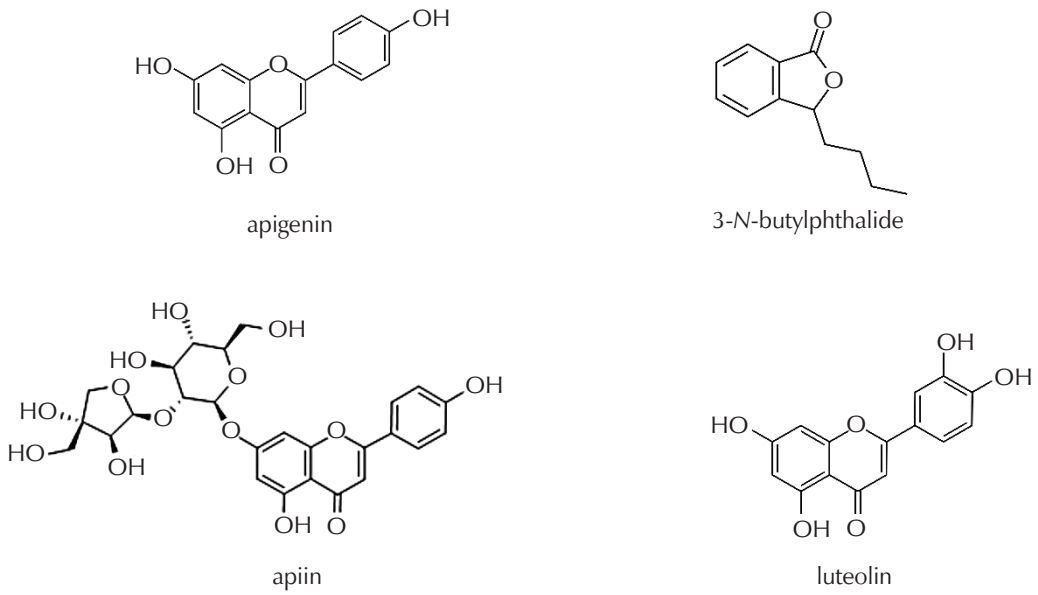



*Fig. 1* – Bioactive compounds of celery have a potential hyperuricemic effect[36]

converting hypoxanthine to xanthine, and subsequently xanthine to uric acid. Hypoxanthine and xanthine are intermediate products of this catabolism.[28,29] In the human body, uric acid circulates in the blood in an unbound form with protein. Uric acid is freely filtered in the glomerulus, but 90 % of it is reabsorbed.[30] An increase in uric acid production, a decrease in renal uric acid excretion, or a combination of both leads to hyperuricemia. Elevated uric acid concentrations in the blood are the main cause of gout, as they facilitate crystal precipitation and disease development.[31]

## 3 Plant-based hypouricemic activity

### 3.1 Celery (*Apium graveolens* L.)

*A. graveolens* L. is a biennial plant belonging to the *Apiaceae* family. This plant is commonly cultivated for its crisp leaf stalks and leaves, which are used in various culinary applications. Celery has demonstrated promising bioactive properties, including antioxidant, anticancer, antimicrobial, antidiabetic, analgesic activity, and anti-inflammatory properties.[32,33] *Dolati et al*. reported that celery extract can reduce serum uric acid levels *via* hepatic xanthine dehydrogenase/xanthine oxidase (XDH/XO) inhibition, highlighting its potential as an effective hypouricemic bioactive agent or functional food.[34] In one report, the ethanolic extract of celery seeds, which contains flavone glycosides (apigenin and apiin), can reduce cytochrome C production and enhance uric acid elimination through improved kidney filtration function. This was reflected in lowered serum urea nitrogen (SUN) and serum creatinine (SCr) levels.[35]

*A. graveolens* synthesises a diverse spectrum of bioactive constituents that underlie its organoleptic characteristics and pharmacological properties. The principal phytochemicals identified in this species include: (i) flavonoid derivatives, notably apigenin, apiin, kaempferol, and luteolin; (ii) furocoumarin-class compounds such as apigravin, celerin, and umbelliferone; (iii) phenolic acids including caffeic acid, *p*-coumaric acid, and ferulic acid; (iv) hydrolysable tannins; and (v) phthalide-rich essential oil fractions.[36] The chemical structure of compounds found in celery is shown in Fig. 1.

*Soliman et al*.[37] reported that administration of celery water extract significantly decreased blood urea nitrogen (BUN) and SUA levels in hyperuricemic rats. These findings suggest that celery extract has the potential to reduce the adverse effects of hyperuricemia.

Many researchers report that apigenin, apiin, and luteolin are responsible for anti-gout activity. Apigenin has been shown to reduce serum uric acid levels, blood urea nitrogen (BUN), serum creatinine (CRE), and renal inflammatory factors in rats. Furthermore, apigenin improved renal fibrosis by suppressing the Wnt/β-catenin pathway.[38] The mechanism by which apigenin ameliorates hyperuricemia involves the inhibition of serum uric acid (SUA) overproduction and the promotion of SUA excretion, as well as regulation of the JAK2/STAT3 signalling pathway.[39] Contrary to these findings, *Zhang et al.*, through *in vivo* studies, demonstrated that apiin significantly reduced serum urate levels in hyperuricemic rodent models, with a clear dose-dependent response. In comparison, apigenin showed only moderate hypouricemic activity. Mechanistic investigations suggested that both flavonoids promote urate excretion by augmenting glomerular filtration rates.[35] Recent studies by *Xiaona et al*. have demonstrated, through enzymatic inhibition assays, a dose-dependent XOD suppression by multiple celery-derived compounds, with the following potency profile (expressed as $IC_{50}$ values): crude celery seed extract (1.98 mg $ml^{-1}$), luteolin (69.23 μM), apigenin (92.56 μM), and chrysoeriol (40.52 μM) demonstrated particularly strong inhibitory effects. Among the glycosylated derivatives, luteolin-7-O-glucoside (975.83 μM) showed moderate activity, while luteolin-7-O-apinosyl glucoside (3140.51 μM) and luteolin-7-O-6›-malonyl glu-

coside (2018.37 μM) exhibited comparatively weaker inhibition.[40] Complementing these findings, *James et al*.[41] identified luteolin as a potent xanthine oxidase (XOD) inhibitor *in vitro*, demonstrating superior efficacy compared to allopurinol. Their quantitative analysis revealed an exceptional $IC_{50}$ of 4.79 μM, indicating significantly stronger enzyme inhibition than allopurinol.[42]

### 3.2 Coffee (*Coffea*)

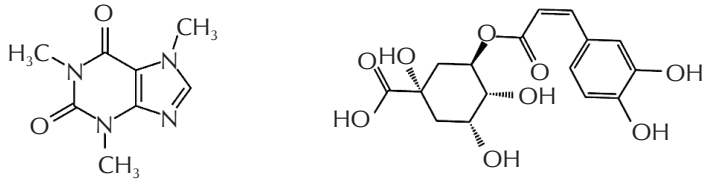
Coffee is derived from the roasted seeds, or beans, of the *Coffea* plant, which is native to tropical regions of Africa and has since been cultivated and consumed worldwide.[43] The global coffee industry predominantly cultivates two commercially significant *Coffea* species: *C. arabica* and *C. canephora* (commonly termed *arabica* and *robusta*, respectively). These varieties exhibit distinct organoleptic and biochemical profiles, with *arabica* beans characterised by their delicate aromatic qualities, while *robusta* beans demonstrate markedly higher caffeine concentrations and more pronounced bitter notes.[44] A meta-analysis carried out by *Park et al*. revealed that coffee consumption was associated with a decrease in serum uric acid levels and the risk of gout in both genders. It is important to note that women require a higher intake of coffee to lower serum uric acid levels compared to men.[45,46] Moreover, habitual coffee consumption was found to be significantly and inversely correlated with gout. In studies comparing coffee-drinking subjects to non-coffee-drinking subjects, the former were found to have lower levels of uric acid, making coffee a potentially viable non-pharmacological alternative for the treatment and prevention of gout.[47]

Coffee beans contain various carbohydrates (sucrose, galactose, glucose, and fructose), proteins, fats, volatile components, melanoidins, alkaloids, phenolic acids, terpenoids, and other ingredients.[48,49] Phenolic acids are the primary compounds that contribute to the pigment, taste, and flavour formation when coffee beans are roasted.[50] The polyphenol from coffee that has been identified as an antioxidant is chlorogenic acid. This compound is formed during roasting.[51] The chemical structure of chlorogenic acid is shown in Fig. 2. Chlorogenic acid has been found to inhibit xanthine oxidase activity.[52] Chlorogenic acid prevents hyperuricemia and nephropathy by regulating gut microbes associated with trimethylamine *N*-oxide (TMAO) and inhibiting the PI3K/AKT/mTOR pathway.[53] Chlorogenic acid administration significantly reduced circulating LPS concentrations and downregulated mRNA expression of key inflammatory mediators, including pro-inflammatory cytokines (IL-1β, TNF-α) and NLRP3 inflammasome components (NLRP3, caspase-1).[54] Furthermore, treatment with mei extract or allopurinol successfully lowered the elevated serum uric acid levels caused by potassium oxonate. The high dosage of mei extract showed a superior anti-hyperuricemia effect compared to the low dosage, and the reduction of serum uric acid levels was found to be statistically significant when compared with the PO group. Statistical analysis revealed no dose-dependent effect of mei extract on lowering serum urate with comparable antihypertensive effect observed between the high and low dose treatment groups ($p > 0.05$ for between-group comparison).[55]

Caffeine (1, 3, 7-trimethylxanthine) is one of the alkaloids found in coffee has and is responsible for its bitter taste.[56] The chemical structure of caffeine is shown in Fig. 2. The effect of caffeine on SUA levels remains controversial. Most studies suggest that coffee (caffeine) intake has an effect on reducing serum uric acid levels. *Liu et al*. investigated the relationship between caffeinated beverages and the risk of gout using Mendelian random analysis. Their findings revealed a significant association between tea or coffee consumption and the risk of gout, suggesting a potential link between caffeine and hyperuricemia.[57] In contrast, several studies have indicated that caffeine is not associated with increased serum uric acid levels. While coffee consumption is associated with lower serum uric acid levels and an increased frequency of hyperuricemia, tea consumption does not exhibit a similar association. These findings suggest that caffeine may not play a significant role in lowering serum uric acid levels.[58]

### 3.3 Ginger (*Zingiber officinale*)

Ginger (*Z. officinale*), a member of the *Zingiberaceae* family, is widely valued both as a culinary spice and as a raw material in traditional medicine, particularly for its rhizome. This part of the plant can be consumed fresh, dried, or in powdered form.[59] The distinctive finger-shaped, swollen middle segments of the ginger rhizome are responsible for its unique spicy flavour.[60] The bioactive compounds in ginger are responsible for its numerous health benefits. Studies have demonstrated that ginger possesses numerous biological activities, including antioxidant, anticancer, anti-inflammatory, antimicrobial, neuroprotective, cardio-




caffeine (1,3,7-trimethylxanthine) chlorogenic acid (3-O-caffeoylquinic acid)

*Fig. 2* – Structures of bioactive compounds found in coffee with potential hyperuricemic effect[51,56]

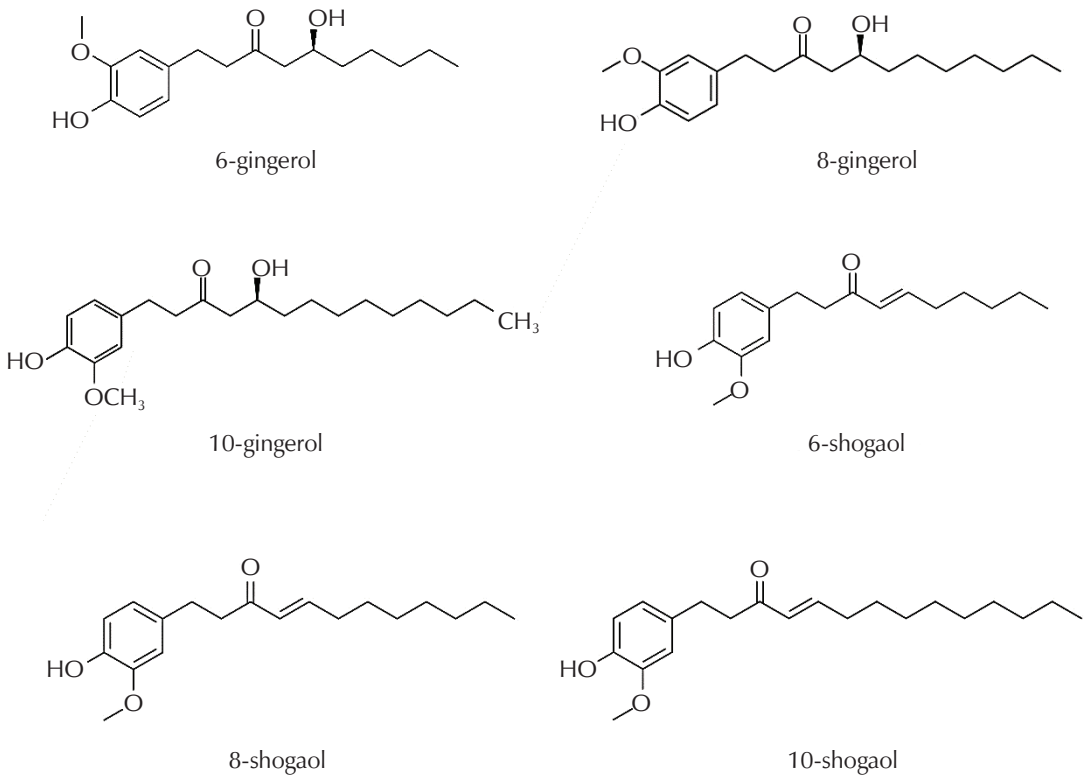



*Fig. 3* – Bioactive compounds of ginger with potential hyperuricemic effect[62]

vascular protective, anti-gout, anti-obesity, antitumor, antidiabetic, antinausea, and antiemetic effects.[61,62]

Ginger rhizomes contain a diverse range of components, including fatty oils, protein, carbohydrates, crude fibre, organic acid, volatile oils, and non-volatile compounds.[63] The essential oils and oleoresins are responsible for the strong, sour, and pungent flavour associated with ginger. The volatile compounds in ginger essential oil consist mainly of sesquiterpenoids, such as α-zingiberene, β-sesquiphellandrene, β-bisabolene, α-farnesene, α-curcumene, and α-zingiberol, among others. The major oleoresin compounds obtained through phenolic extraction include gingerols, shogaols, paradols, quercetin, zingerone, gingerenone-A, and 6-dehydrogingerdione.[62]

Several bioactive compounds in ginger, including 6-gingerol, 8-gingerol, 10-gingerol, and 6-shogaol, demonstrate antioxidant activity. The chemical structures of gingerol and shogaol are shown in Fig. 3. *In vitro*, 6-gingerol exhibited the highest antioxidant activity, followed by 6-shogaol. *In vitro* and *in vivo* studies have shown that 6-shogaol can effectively reduce inflammatory mediator systems, such as COX-2 and iNOS, and affect NFκB and MAPK signalling pathways. Additionally, this compound has been shown to increase ocytoprotective HO-1 levels.[64] Several *in vitro* studies have offered deeper mechanistic insights into the anti-inflammatory effects of 6-shogaol, highlighting its involvement in pathways such as PPAR-γ, JNK/Nrf2, p38/HO-1, and NFκB. Furthermore, the oral administration of microemulsified 6-shogaol in hyperuricemic rats was found to significantly decrease uric acid levels and xanthine oxidase activity.[65] Histological studies also confirmed that the formulation groups offered better protection for the kidneys than the free drug groups. The reduced lysosomal enzyme activities observed in monosodium urate (MSU) crystal-induced mice following 6-shogaol treatment suggest that this compound may inhibit the release of lysosomal enzymes through its stabilising effects. Additionally, the decrease in MSU crystal-induced paw oedema after 6-shogaol administration indicates a significant reduction in total leukocyte migration, as well as in the migration of lymphocytes and monocytes/macrophages from the blood into the synovial cavity.[66] Application of 6-shogaol as a nanoparticle formula reduces the uric acid level by inhibiting xanthine oxidase (XO) activity, and reducing interleukin-1β (IL-1β) and tumor necrosis factor (TNF-α) production.[67] *Marahatha et al.* reported that 6-gingerol can inhibit xanthine oxidase, an enzyme that catalyses the conversion of uric acid from xanthine and hypoxanthine to xanthine during the final step of purine metabolic breakdown by producing reactive oxygen species.[68]

### 3.4 Cherry (*Prunus* sp.)

Cherries belong to the genus *Prunus* within the *Rosaceae* family. There are two primary types of cherries: sweet cherries (*P. avium*), which are typically consumed fresh, and sour cherries (*P. cerasus*), which are often frozen, canned, and utilised in sauces and pastries.[69] Cherries provide a variety of health benefits due to their excellent nutritional content. They support the immune system and help reduce the risk of diseases such as cardiovascular disease,

diabetes, inflammatory diseases, cancer, and gout.[70] Cherries are rich in vitamins A, C, and E, sugar, carotenoids, quercetin, essential minerals, organic acids, flavonols, and phenolics like anthocyanins. The primary phenolic acids present in sour cherries include 3-caffeoylquinic acid, 5-caffeoylquinic acid, and p-coumaric acid. Flavanols are primarily composed of catechin and epicatechin derivatives, whereas flavonols are predominantly represented by quercetin and kaempferol glycosides.[71–73] The chemical structure in bioactive compounds of cherry is presented in Fig. 4.

Polyphenols and vitamin C may be responsible for antioxidant and anti-inflammatory activity.[74] Research shows that cherries, their extract, and their products have antioxidant properties, inhibit inflammation against urate crystals, and reduce SUA.[75,76] In one research report, phenolics and anthocyanins found abundantly in cherries have been associated with the inhibition of interleukin-6 (IL-6), tumour necrosis factor alpha (TNF-α), IL-1β, IL-8, COX-I, and COX-II. This suggests that sweet cherries may have the ability to reduce both acute and chronic inflammation, which may play a role in the recurrence of gout and in chronic destructive arthropathy.[77] Sweet cherry extract showed significant inhibitory effects on the XO system.

Major compounds of the methanolic extracts are hydroxycinnamic acids (3-O-caffeoylquinic acid, 4-O-caffeoylquinic acid, 3-*p*-coumarylquinic acid), anthocyanins (cyanidin-3,5-O-dihexoside, cyanidin-3-O-glucoside, cyanidin-3-O-rutinoside, and peonidin-3-O-rutinoside), and flavonols (isorhamnetin-O-hexoside, quercetin-3-O-rutinoside, kaempferol-3-O-rutinoside, kaempferol-3-O-glucoside).[78] Methanolic extracts of sour cherry inhibited xanthine oxidase with an $IC_{50}$ of 2.619 mg $ml^{-1}$. Polyphenols and flavonoids are important compounds in the inhibition of XO activity in sour cherry extract, not anthocyanins.[79] Cherry contains quercetin, which has been established as an effective inhibitor of xanthine oxidase (XO) for several years. Numerous studies have demonstrated that quercetin

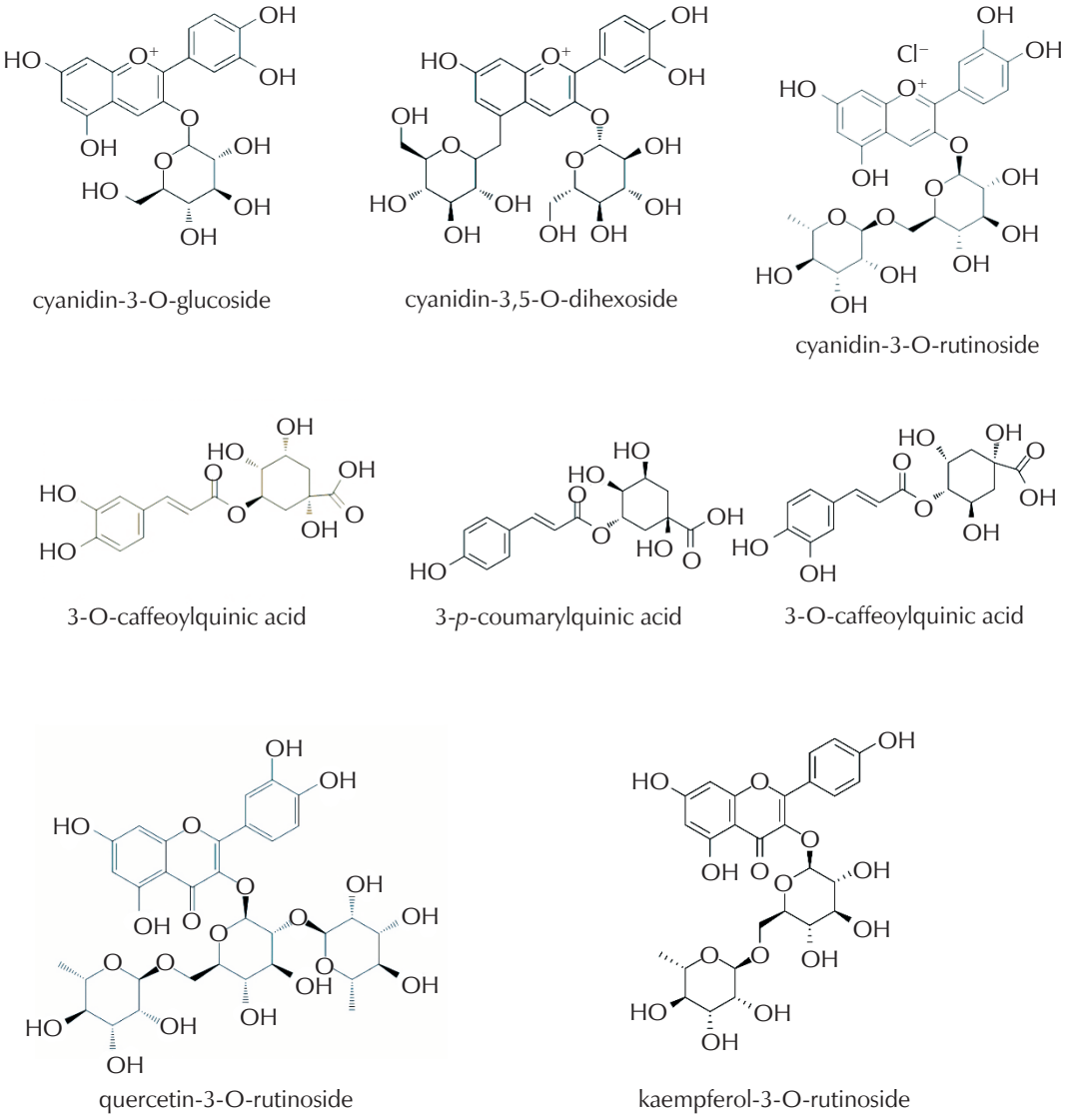



*Fig. 4* – Bioactive compounds of cherry with potential hyperuricemic effect[71–73]

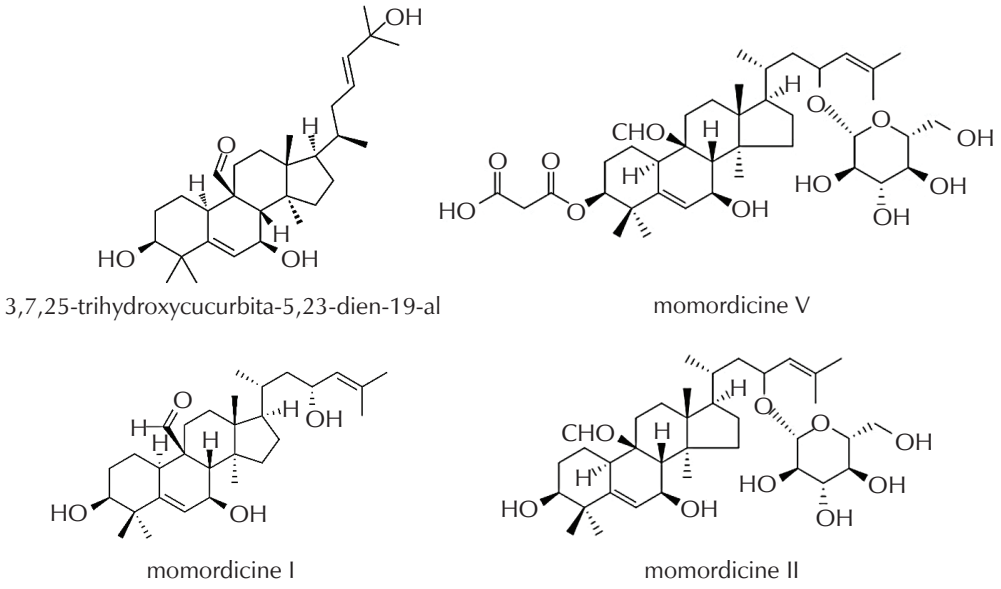



*Fig. 5* – Structure of five cucurbitane-type of *M. charantia*[86]

exerts a potent inhibitory effect on xanthine oxidase (XO), with an $IC_{50}$ value of 0.44 μM, which is stronger than the standard allopurinol, which has an $IC_{50}$ value of 0.77 μM. In another study, quercetin isolated from *Filipendula ulmaria*, a plant traditionally used for gout, inhibited XO with an $IC_{50}$ value of 1.07 ± 0.06 μg ml$^{-1}$, also surpassing allopurinol, which exhibited an $IC_{50}$ value of 2.0 ± 0.1 μg ml$^{-1}$. These findings offer valuable insights into the potential efficacy of Montmorency tart cherry juice as a treatment for gout.[80]

### 3.5 Bitter Melon (*Momordica charantia*)

*M. charantia*, commonly known as bitter melon or pare in Indonesia, is a tropical and subtropical vine belonging to the gourd family (*Cucurbitaceae*). It is cultivated for its fruit, which is used both as a culinary ingredient and in traditional medicine in various cultures.[81] The use of bitter melon in traditional medicine dates back centuries, and it is believed to possess a range of potential health benefits. These include antidiabetic, hypoglycemic, antibacterial, anti-gout, anti-inflammatory, antiviral, antioxidant, anti-helminthic, hepatoprotective, antileukemic, antitumor, and immunomodulatory properties.[82,83] *M. charantia* contains a variety of biologically active compounds, including glycosides, lipids, proteins, alkaloids, triterpenes, flavonoids, phenolics, glycosides, steroids, chalcones, carotenoids, tannins, saponins, iridoids, ursolic acid, and imidazolines.[84,85]

The potential of *M. charantia* extracts as an antioxidant agent has been demonstrated by *in vitro* and *in vivo* studies. An *in vivo* biological investigation was conducted on the ethanolic extract of *Momordica charantia*, a plant from the Cucurbitaceae family, to evaluate its antihyperlipidemic activity and serum uric acid-reducing potential. The results demonstrated that the ethanolic extract of *M. charantia* effectively reduced serum uric acid levels in both experimental groups.[87] Cucurbitane-type triterpene glycosides (derivated compounds are shown in Fig. 5) isolated from *M. charantia* part significantly inhibit XO activity.[88] Triterpenoids isolated from the stems of *M. charantia* displayed ABTS radical cation scavenging activity with $IC_{50}$ values of 268.5 and an inhibitory effect on xanthine oxidase (XO) activity with $IC_{50}$ values of 36.8 μM.[89] Based on the database, more than 90 triterpenes have been obtained from different parts of *M. charantia*.[90]

### 3.6 Other plants

In addition to the primary species discussed in earlier sections, a variety of other plant species exhibit notable bioactivity through their rich content of phenolic compounds or other bioactive molecules. These species may contribute to the modulation of uric acid levels and the inhibition of xanthine oxidase (XO), which plays a central role in the pathogenesis of hyperuricemia and gout.

1. Apigenin, apart from being found in celery, apigenin is also found in several plants such as parsley, basil, chamomile tea, and kumquat, as well as fruits and vegetables such as guava and bilimbi fruit.[91]
2. Quercetin and its derivatives have exhibited good potential in exerting xanthine oxidase (XO) inhibitory activity. Many plant species contain quercetin, *e.g., Quercus robur* (oak), garlic, apple, moringa.[92]
3. Reservatol can act as a xanthine oxidase inhibitor, meaning it can slow down or stop the enzyme xanthine oxidase from converting xanthine into uric acid, contained in grapes, raspberries, mulberries, pistachios, and peanuts.[93,94]

## 4 Conclusion

Hyperuricemia results from increased uric acid production, decreased renal excretion of uric acid, or a combination of both. It is a major risk factor for gout and a prerequisite for crystal precipitation and disease development. Herbal medicine has been explored for the prevention and treatment of hyperuricemia, with several plants commonly available in daily life. Extracts or bioactive compounds from the reviewed plants demonstrate potential hypouricemic activity both *in vivo* and *in vitro*. Herbal medicines contain a variety of active constituents that act as anti-inflammatory, antioxidant, and uric acid transport modulators. Many of these herbs exert their effects through multiple mechanisms, such as improving hyperuricemia and lowering serum uric acid levels. A phytochemical matrix often comprises several components, rather than relying on a single active compound.

### ACKNOWLEDGEMENTS

Not applicable.

#### Author contributions

All authors had equal contributions as the main contributors to this manuscript paper.

#### Funding

No funding resource could be reported for this publication.

#### Availability of data and materials

Not applicable.

### DECLARATIONS

#### Ethics approval and consent to participate

Not applicable.

#### Consent for publication

The authors have given approval for the publication of this manuscript.

#### Competing interests

The authors declare no competing interests.

## References
## Literatura

1. *M. Skoczyńska, M. Chowaniec, A. Szymczak, A. Langner-Hetmańczuk, B. Maciążek-Chyra, P. Wiland*, Pathophysiology of hyperuricemia and its clinical significance – a narrative review, Reumatologia **58** (2020) 312–323, doi: https://doi.org/10.5114/reum.2020.100140.
2. *T. Petreski, R. Ekart, R. Hojs, S. Bevc*, Hyperuricemia, the heart, and the kidneys – to treat or not to treat?, Ren. Fail. **42** (2020) 978–986, doi: https://doi.org/10.1080/0886022X.2020.1822185.
3. *A. F. G. Cicero, F. Fogacci, M. Kuwabara, C. Borghi*, Physiology of hyperuricemia and urate-lowering treatments, Med. **57** (2021) 58, doi: https://doi.org/10.3390/medicina57010058.
4. *L. Qiu, X. Q. Cheng, J. Wu, J. T. Liu, T. Xu, H. T. Ding, Y. H. Liu, Z. M. Ge, Y. J. Wang, H. J. Han, J. Liu, G. J. Zhu*, Prevalence of hyperuricemia and its related risk factors in healthy adults from Northern and Northeastern Chinese provinces, BMC Public Health **13** (2013) 664, doi: https://doi.org/10.1186/1471-2458-13-664.
5. *C. L. Benn, P. Dua, R. Gurrell, P. Loudon, A. Pike, R. Ian Storer, C. Vangjeli*, Physiology of hyperuricemia and urate-lowering treatments, Front. Med. **5** (2018) 160, doi: https://doi.org/10.3389/fmed.2018.00160.
6. *A. Kushiyama, Y. Nakatsu, Y. Matsunaga, T. Yamamotoya, K. Mori, K. Ueda, Y. Inoue, H. Sakoda, M. Fujishiro, H. Ono, T. Asano*, Role of uric acid metabolism-related inflammation in the pathogenesis of metabolic syndrome components such as atherosclerosis and nonalcoholic steatohepatitis, Mediators Inflamm. **2016** (2016) 603164, doi: https://doi.org/10.1155/2016/8603164.
7. *M. E. Gherghina, I. Peride, M. Tiglis, T. P. Neagu, A. Niculae, I. A. Checherita*, Uric Acid and Oxidative Stress—Relationship with Cardiovascular, Metabolic, and Renal Impairment, Int. J. Mol. Sci. **23** (2022) 3188, doi: https://doi.org/10.3390/ijms23063188.
8. *H. Yin, N. Liu, J. Chen*, The Role of the Intestine in the Development of Hyperuricemia, Front. Immunol. **13** (2022) 845684, doi: https://doi.org/10.3389/fimmu.2022.845684.
9. *T. Sapankaew, K. Thadanipon, N. Ruenroengbun, K. Chaiyakittisopon, A. Ingsathit, P. Numthavaj, N. Chaiyakunapruk, G. McKay, J. Attia, A. Thakkinstian*, Efficacy and safety of urate-lowering agents in asymptomatic hyperuricemia: systematic review and network meta-analysis of randomized controlled trials, BMC Nephrol. **23** (2022) 223, doi: https://doi.org/10.1186/s12882-022-02850-3.
10. *L. A. B. Joosten, T. O. Crişan, P. Bjornstad, R. J. Johnson*, Asymptomatic hyperuricaemia: a silent activator of the innate immune system, Nat. Rev. Rheumatol. **16** (2020) 75–86, doi: https://doi.org/10.1038/s41584-019-0334-3.
11. *D. J. Stewart, V. Langlois, D. Noone*, Hyperuricemia and hypertension: Links and risks, Integr. Blood Press. Control **12** (2019) 43–62, doi: https://doi.org/10.2147/IBPC.S184685.
12. *W. Yu, J. D. Cheng*, Uric Acid and Cardiovascular Disease: An Update From Molecular Mechanism to Clinical Perspective, Front. Pharmacol. **11** (2020) 582680, doi: https://doi.org/10.3389/fphar.2020.582680.
13. *M. Kakutani-Hatayama, M. Kadoya, H. Okazaki, M. Kurajoh, T. Shoji, H. Koyama, Z. Tsutsumi, Y. Moriwaki, M. Namba, T. Yamamoto*, Nonpharmacological Management of Gout and Hyperuricemia: Hints for Better Lifestyle, Am. J. Lifestyle Med. **11** (2017) 321–329, doi: https://doi.org/10.1177/1559827615601973.
14. *Q. Li, X. Li, J. Wang, H. Liu, J. S. W. Kwong, H. Chen, L. Li, S. C. Chung, A. Shah, Y. Chen, Z. An, X. Sun, H. Hemingway, H. Tian, S. Li*, Diagnosis and treatment for hyperuricemia and gout: A systematic review of clinical practice guidelines and consensus statements, BMJ Open **9** (2019) e026677, doi: https://doi.org/10.1136/bmjopen-2018-026677.
15. *T. Aung, G. Myung, J. D. FitzGerald*, Treatment approaches and adherence to urate-lowering therapy for patients with gout, Patient Prefer. Adherence **11** (2017) 795–800, doi: https://doi.org/10.2147/PPA.S97927.
16. *S. Li, H. Yang, Y. Guo, F. Wei, X. Yang, D. Li, M. Li, W. Xu, W. Li, L. Sun, Y. Gao, Y. Wang*, Comparative efficacy and safety of urate-lowering therapy for the treatment of hyperuricemia: A systematic review and network meta-analysis, Sci. Rep. **6** (2016) 33082, doi: https://doi.org/10.1038/srep33082.
17. *M. Ortiz-Uriarte, J. Betancourt-Gaztambide, A. Perez, Y. M. Roman*, Urate-Lowering Therapy Use among US Adults with

Gout and the Relationship between Patients' Gout Treatment Status and Associated Comorbidities, Rheumato **3** (2023) 74–85, doi: https://doi.org/10.3390/rheumato3010006.

18. *F. Perez-Ruiz, N. Dalbeth*, Combination urate-lowering therapy in the treatment of gout: What is the evidence?, Semin. Arthritis Rheum. **48** (2019) 658–668, doi: https://doi.org/10.1016/j.semarthrit.2018.06.004.
19. *C.-Y. Wang, J.-G. Dai*, Research progress on the prevention and treatment of hyperuricemia by medicinal and edible plants and its bioactive components, Front. Nutr. **10** (2023) 1186161, doi: https://doi.org/10.3389/fnut.2023.1186161.
20. *M. D. Do, T. P. Mai, A. D. Do, Q. D. Nguyen, N. H. Le, L. G. H. Le, V. A. Hoang, A. N. Le, H. Q. Le, P. Richette, M. Resche-Rigon, T. Bardin*, Risk factors for cutaneous reactions to allopurinol in Kinh Vietnamese: Results from a case-control study, Arthritis Res. Ther. **22** (2020) 182, doi: https://doi.org/10.1186/s13075-020-02273-1.
21. *A. Qurie, C. V. Preuss, R. Musa*, Allopurinol. in StatPearls [Internet], StatPearls Publishing, 2023, doi: https://doi.org/10.1016/j.ijbiomac.2012.08.003.
22. *C. A. Roncal-Jimenez, Y. Sato, T. Milagres, A. A. Hernando, G. García, P. Bjornstad, J. B. Dawson, C. Sorensen, L. Newman, L. Krisher, M. Madero, J. Glaser, R. Gárcía-Trabanino, E. J. Romero, Z. Song, T. Jensen, M. Kuwabara, B. Rodriguez-Iturbe, L. G. Sanchez-Lozada, M. A. Lanaspa, R. J. Johnson*, Experimental heat stress nephropathy and liver injury are improved by allopurinol, Am. J. Physiol. Ren. Physiol. **315** (2018) F726–F733, doi: https://doi.org/10.1152/ajprenal.00543.2017.
23. *A. Rey, B. Batteux, S. M. Laville, J. Marienne, K. Masmoudi, V. Gras-Champel, S. Liabeuf*, Acute kidney injury associated with febuxostat and allopurinol: a post-marketing study, Arthritis Res. Ther. **21** (2019) 229, doi: https://doi.org/10.1186/s13075-019-2011-y.
24. *L. Xu, L. L. Lu, J. D. Gao*, Traditional Chinese Herbal Medicine Plays a Role in the Liver, Kidney, and Intestine to Ameliorate Hyperuricemia according to Experimental Studies, Evidence-based Complement. Altern. Med. **2021** (2021) 618352, doi: https://doi.org/10.1155/2021/4618352.
25. *M. G. Battelli, M. Bortolotti, L. Polito, A. Bolognesi*, The role of xanthine oxidoreductase and uric acid in metabolic syndrome, Biochim. Biophys. Acta - Mol. Basis Dis. **1864** (2018) 2557–2565, doi: https://doi.org/10.1016/j.bbadis.2018.05.003.
26. *J. Maiuolo, F. Oppedisano, S. Gratteri, C. Muscoli, V. Mollace*, Regulation of uric acid metabolism and excretion, Int. J. Cardiol. **213** (2016) 8–14, doi: https://doi.org/10.1016/j.ijcard.2015.08.109.
27. *C. Papandreou, J. Li, L. Liang, M. Bulló, Y. Zheng, M. Ruiz-Canela, E. Yu, M. Guasch-Ferré, C. Razquin, C. Clish, D. Corella, R. Estruch, E. Ros, M. Fitó, F. Arós, L. Serra-Majem, N. Rosique, M. A. Martínez-González, F. B. Hu, J. Salas-Salvadó*, Metabolites related to purine catabolism and risk of type 2 diabetes incidence; modifying effects of the TCF7L2-rs7903146 polymorphism, Sci. Rep. **9** (2019) 2892, doi: https://doi.org/10.1038/s41598-019-39441-6.
28. *C. J. Chen, J. M. Lü, Q. Yao*, Hyperuricemia-related diseases and xanthine oxidoreductase (XOR) inhibitors: An overview, Med. Sci. Monit. **22** (2016) 2501–2512, doi: https://doi.org/10.12659/MSM.899852.
29. *M. Sekine, K. Okamoto, K. Ichida*, Association of mutations identified in xanthinuria with the function and inhibition mechanism of xanthine oxidoreductase, Biomedicines **9** (2021) 1723, doi: https://doi.org/10.3390/biomedicines9111723.
30. *M. Gliozzi, N. Malara, S. Muscoli, V. Mollace*, The treatment of hyperuricemia, Int. J. Cardiol. **213** (2016) 23–27, doi: https://doi.org/10.1016/j.ijcard.2015.08.087.
31. *W.-Z. Zhang*, Why does hyperuricemia not necessarily induce gout?, Biomolecules **11** (2021) 280, doi: https://doi.org/10.3390/biom11020280.
32. *A. K. Al-Asmari, M. T. Athar, S. G. Kadasah*, An Updated Phytopharmacological Review on Medicinal Plant of Arab Region: *Apium graveolens* Linn., Pharmacogn. Rev. **11** (2017) 8–13, doi: https://doi.org/10.4103/phrev.phrev_35_16.
33. *T. Yew, C. Tan, X. Y. Lim, N. A. Norahmad, H. C. Kumar, B. P. Teh, N. M. Lai, A. Fazlin, S. Mohamed*, Neurological Applications of Celery (*Apium graveolens*): A Scoping Review, Molecules **28** (2023) 5824, doi: https://doi.org/10.3390/molecules28155824.
34. *K. Dolati, H. Rakhshandeh, M. Golestani, F. Forouzanfar, R. Sadeghnia, H. R. Sadeghnia*, Inhibitory effects of apium graveolens on xanthine oxidase activity and serum Uric acid levels in hyperuricemic mice, Prev. Nutr. Food Sci. **23** (2018) 127–133, doi: https://doi.org/10.3746/pnf.2018.23.2.127.
35. *C. Zhang, M. Zhao, B. Jiang, J. Yu, Q. Hao, W. Liu, Z. Hu, Y. Zhang, C. Song*, Extraction optimization, structural characterization and potential alleviation of hyperuricemia by flavone glycosides from celery seeds, Food Funct. **13** (2022) 9832–9846, doi: https://doi.org/10.1039/d2fo01715f.
36. *N. T. Hang, T. Thi Tu Uyen, N. Van Phuong*, Green extraction of apigenin and luteolin from celery seed using deep eutectic solvent, J. Pharm. Biomed. Anal. **207** (2022) 114406, doi: https://doi.org/10.1016/j.jpba.2021.114406.
37. *M. M. Soliman, M. A. Nassan, A. Aldhahrani, F. Althobaiti, W. A. Mohamed*, Molecular and Histopathological Study on the Ameliorative Impacts of *Petroselinum Crispum* and *Apium Graveolens* against Experimental Hyperuricemia, Sci. Rep. **10** (2020) 9512, doi: https://doi.org/10.1038/s41598-020-66205-4.
38. *Y. Li, Z. Zhao, J. Luo, Y. Jiang, L. Li, Y. Chen, L. Zhang, Q. Huang, Y. Cao, P. Zhou, T. Wu, J. Pang*, Apigenin ameliorates hyperuricemic nephropathy by inhibiting URAT1 and GLUT9 and relieving renal fibrosis via the Wnt/β-catenin pathway, Phytomedicine **87** (2021) 153585, doi: https://doi.org/10.1016/j.phymed.2021.153585.
39. *T. Liu, H. Gao, Y. Zhang, S. Wang, M. Lu, X. Dai, Y. Liu, H. Shi, T. Xu, J. Yin, S. Gao, L. Wang, D. Zhang*, Pharmaceuticals **16** (2022) 819, doi: https://doi.org/10.3390/ph15111442.
40. *X. Gan, B. Peng, L. Chen, Y. Jiang, T. Li, B. Li, X. Liu*, Identification of Xanthine Oxidase Inhibitors from Celery Seeds Using Affinity Ultrafiltration–Liquid Chromatography–Mass Spectrometry, Molecules **28** (2023) 6048, doi: https://doi.org/10.3390/molecules28166048.
41. *J. M. Pauff, R. Hille*, Inhibition studies of bovine xanthine oxidase by luteolin, silibinin, quercetin, and curcumin, J. Nat. Prod. **72** (2009) 725–731, doi: https://doi.org/10.1021/np8007123.
42. *J. Yan, G. Zhang, Y. Hu, Y. Ma*, Effect of luteolin on xanthine oxidase: Inhibition kinetics and interaction mechanism merging with docking simulation, Food Chem. **141** (2013) 2766–2773, doi: https://doi.org/10.1016/j.foodchem.2013.06.092.
43. *P. Hamon, J. J. Rakotomalala, S. Akaffou, N. J. Razafinarivo, E. Couturon, R. Guyot, D. Crouzillat, S. Hamon, A. De Kochko*, Caffeine-free Species in the Genus Coffea, Elsevier Inc., 2015, doi: https://doi.org/10.1016/B978-0-12-409517-5.00005-X.
44. *A. Simon-Gruita, M. D. Pojoga, N. Constantin, G. Duta-Cornescu*, Genetic engineering in coffee, Elsevier Inc., 2019, doi: https://doi.org/10.1016/B978-0-12-815864-7.00014-3.

45. *K. Y. Park, H. J. Kim, H. S. Ahn, S. H. Kim, E. J. Park, S. Y. Yim, J. B. Jun*, Effects of coffee consumption on serum uric acid: Systematic review and meta-analysis, Semin. Arthritis Rheum. **45** (2016) 580–586, doi: https://doi.org/10.1016/j.semarthrit.2016.01.003.
46. *Y. Shirai, A. Nakayama, Y. Kawamura, Y. Toyoda, M. Nakatochi, S. Shimizu, N. Shinomiya, Y. Okada, H. Matsuo*, Coffee Consumption Reduces Gout Risk Independently of Serum Uric Acid Levels: Mendelian Randomization Analyses Across Ancestry Populations, ACR Open Rheumatol. **4** (2022) 534–539, doi: https://doi.org/10.1002/acr2.11425.
47. *R. Sunita, Y. Anggraini, Krisyanella*, Lower Uric Acid Levels in Subjects Consuming Coffee compared to Not Consuming Coffee, AHSR **14** (2019) 103–106, doi: https://doi.org/10.2991/icihc-18.2019.26.
48. *S. Saud, A. M. Salamatullah*, Relationship between the chemical composition and the biological functions of coffee, Molecules **26** (2021) 7634, doi: https://doi.org/10.3390/molecules26247634.
49. *G. V. de Melo Pereira, D. P. de Carvalho Neto, A. I. Magalhães Júnior, F. G. do Prado, M. G. B. Pagnoncelli, S. G. Karp, C. R. Soccol*, Chemical composition and health properties of coffee and coffee by-products, 1st ed Elsevier Inc., Vol. **9**, 2020, doi: https://doi.org/10.1016/bs.afnr.2019.10.002.
50. *K. Król, M. Gantner, A. Tatarak, E. Hallmann*, The content of polyphenols in coffee beans as roasting, origin and storage effect, Eur. Food Res. Technol. **246** (2020) 33–39, doi: https://doi.org/10.1007/s00217-019-03388-9.
51. *H. Wu, P. Lu, Z. Liu, J. Sharifi-Rad, H. A. R. Suleria*, Impact of roasting on the phenolic and volatile compounds in coffee beans, Food Sci. Nutr. **10** (2022) 2408–2425, doi: https://doi.org/10.1002/fsn3.2849.
52. *A. Mehmood, J. Li, A. U. Rehman, R. Kobun, I. U. Llah, I. Khan, F. Althobaiti, S. Albogami, M. Usman, F. Alharthi, M. M. Soliman, S. Yaqoob, K. A. Awan, L. Zhao, L. Zhao*, Xanthine oxidase inhibitory study of eight structurally diverse phenolic compounds, Front. Nutr. **9** (2022) 966557, doi: https://doi.org/10.3389/fnut.2022.966557.
53. *X. Zhou, B. Zhang, X. Zhao, Y. Lin, Y. Zhuang, J. Guo, S. Wang*, Chlorogenic Acid Prevents Hyperuricemia Nephropathy via Regulating TMAO-Related Gut Microbes and Inhibiting the PI3K/AKT/mTOR Pathway, J. Agric. Food Chem. **70** (2022) 10182, doi: https://doi.org/https://doi.org/10.1021/acs.jafc.2c03099.
54. *X. Zhou, B. Zhang, X. Zhao, Y. Lin, J. Wang, X. Wang, N. Hua, S. Wang*, Chlorogenic acid supplementation ameliorates hyperuricemia, relieves renal inflammation, and modulates intestinal homeostasis, Food Funct. **12** (2021) 5637–5649, doi: https://doi.org/https://doi.org/10.1039/D0FO03199B.
55. *Y.-C. Wu, P.-C. Kuo, W.-Y. Chen, J. T. C. Tzen*, Reduction of the Plasma Uric Acid Level in Potassium Oxoate-Induced Hyperuricemic Rats by Heat-Concentrated Prunus mume Fruit Extract Containing Three Chlorogenic Acid Isomers, Compounds **3** (2023) 169–179, doi: https://doi.org/10.3390/compounds3010014.
56. *R. Urrialde*, Caffeine. In: Encyclopedia of Human Nutrition, 4th Ed., 2023, pp. 96–104, doi: https://doi.org/10.1016/B978-0-12-821848-8.00062-7.
57. *H. Liu, R. Xie, Q. Dai, J. Fang, Y. Xu, B. Li*, Exploring the mechanism underlying hyperuricemia using comprehensive research on multi-omics, Sci. Rep. **13** (2023) 7161, doi: https://doi.org/10.1038/s41598-023-34426-y.
58. *H. K. Choi, G. Curhan*, Coffee, tea, and caffeine consumption and serum uric acid level: The Third National Health and Nutrition Examination Survey, Arthritis Care Res. **57** (2007) 816–821, doi: https://doi.org/10.1002/art.22762.
59. *N. H. Anh, S. jo Kim, N. P. Long, J. E. Min, Y. C. Yoon, E. G. Lee, M. Kim, T. J. Kim, Y. Y. Yang, E. Y. Son, S. J. Yoon, N. C. Diem, H. M. Kim, S. W. Kwon*, Ginger on Human Health: A Comprehensive Systematic Review of 109 Randomized Controlled Trials, Nutrients **12** (2020) 157, doi: https://doi.org/doi:10.3390/nu12010157.
60. *J. O. Unuofin, N. P. Masuku, O. K. Paimo, S. L. Lebelo*, Ginger from Farmyard to Town: Nutritional and Pharmacological Applications, Front. Pharmacol. **12** (2021), doi: https://doi.org/10.3389/fphar.2021.779352.
61. *S. Zhang, X. Kou, H. Zhao, K. K. Mak, M. K. Balijepalli, M. R. Pichika*, *Zingiber officinale* var. *Rubrum*: Red Ginger's Medicinal Uses, Molecules **27** (2022) 775, doi: https://doi.org/10.3390/molecules27030775.
62. *Q. Q. Mao, X. Y. Xu, S. Y. Cao, R. Y. Gan, H. Corke, T. Beta, H. Bin Li*, Bioactive compounds and bioactivities of ginger (*Zingiber officinale Roscoe*), Foods **8** (2019) 185, doi: https://doi.org/10.3390/foods8060185.
63. *R. Kiyama*, Nutritional implications of ginger: chemistry, biological activities and signaling pathways, J. Nutr. Biochem. **86** (2020) 108486, doi: https://doi.org/10.1016/j.jnutbio.2020.108486.
64. *I. Bischoff-Kent, R. Furst*, Benefits of ginger and Its Constituent 6-shogaol in inhibiting inflammatory processes, Pharmaceuticals **14** (2021) 571, doi: https://doi.org/https://doi.org/10.3390/ph14060571.
65. *Q. Yang, Q. Wang, Y. Feng, Q. Wei, C. Sun, C. K. Firempong, M. Adu-Frimpong, R. Li, R. Bao, E. Toreniyazov, H. Ji, J. Yu, X. Xu*, Anti-hyperuricemic property of 6-shogaol via self-micro emulsifying drug delivery system in model rats: formulation design, in vitro and in vivo evaluation, Drug Dev. Ind. Pharm. **45** (2019) 1265–1276, doi: https://doi.org/10.1080/03639045.2019.1594885.
66. *S. Evan prince, M. Rasool, L. Mathew, P. Ezilrani, H. Indu*, 6-Shogaol inhibits monosodium urate crystal-induced inflammation-An *in vivo* an *in vitro* study, Food Chem. Toxicol. **48** (2010) 229–235, doi: https://doi.org/10.1016/j.fct.2009.10.005.
67. *Q. Wang, Q. Yang, X. Cao, Q. Wei, C. K. Firempong, M. Guo, F. Shi, X. Xu, W. Deng, J. Yu*, Enhanced oral bioavailability and anti-gout activity of [6]-shogaol-loaded solid lipid nanoparticles, Int. J. Pharm. **550** (2018) 24–34, doi: https://doi.org/10.1016/j.ijpharm.2018.08.028.
68. *R. Marahatha, S. Basnet, B. R. Bhattarai, P. Budhathoki, B. Aryal, B. Adhikari, G. Lamichhane, D. K. Poudel, N. Parajuli*, Potential natural inhibitors of xanthine oxidase and HMG-CoA reductase in cholesterol regulation: in silico analysis, BMC Complement. Med. Ther. **21** (2021) 1, doi: https://doi.org/10.1186/s12906-020-03162-5.
69. *V. Joshi, P. Panesar, V. Rana, S. Kaur*, Science and Technology of Fruit Wines : An Overview, in: Science and Technology of Fruit Wine Production, Academic Press, 2017, pp. 1–72, doi: https://doi.org/https://doi.org/10/1016/B978-0-12-800850-8.00001-6.
70. *P. E. Chen, C. Y. Liu, W. H. Chien, C. W. Chien, T. H. Tung*, Effectiveness of Cherries in Reducing Uric Acid and Gout: A Systematic Review, Evidence-based Complement. Altern. Med. **2019** (2019) 896757, doi: https://doi.org/10.1155/2019/9896757.
71. *A. Sokół-Łętowska, A. Z. Kucharska, G. Hodun, M. Gołba*, Chemical composition of 21 cultivars of sour cherry (*Prunus cerasus*) fruit cultivated in Poland, Molecules **25** (2020) 4587, doi: https://doi.org/10.3390/molecules25194587.
72. *L. Cruz-lopes, Y. Dulyanska, I. Domingos, J. Ferreira, A. Fragata, R. Guine, B. Esteves*, Influence of Pre-Hydrolysis on the Chemical Composition of *Prunus avium* Cherry Seeds,

Agronomy **12** (2022) 280, doi: https://doi.org/https://doi.org/10.3390/agronomy12020280.

73. *A. Wojdylo, P. Nowicka, P. Laskowski, J. Oszmianski*, Evaluation of Sour Cherry (Prunus cerasus L.) Fruits for Their Polyphenol Content, Antioxidant Properties, and Nutritional Components, J. Agric. Food Chem. **62** (2014) 12332–12345, doi: https://doi.org/https://doi.org/10.1021/jf504023.
74. *D. S. Kelley, Y. Adkins, K. D. Laugero*, A review of the health benefits of cherries, Nutrients **10** (2018) 368, doi: https://doi.org/10.3390/nu10030368.
75. *Y. Aqil, S. El Hajjaji, W. Belmaghraoui, Y. Mourabit, D. Taha, M. M. Alshahrani, A. A. Al Awadh, A. Bouyahya, I. Gaamoussi, I. Bourais*, Phenolic Profile, Antioxidant, Antidiabetic, and Antigout Potential of Stem Extracts of Four Sweet Cherry Cultivars, Evidence-based Complement. Altern. Med. **2023** (2023) 535139, doi: https://doi.org/10.1155/2023/8535139.
76. *C. Wang, W. Sun, N. Dalbeth, Z. Wang, X. Wang, X. Ji, X. Xue, L. Han, L. Cui, X. Li, Z. Liu, A. Ji, Y. He, M. Sun, C. Li*, Efficacy and safety of tart cherry supplementary citrate mixture on gout patients: a prospective, randomized, controlled study, Arthritis Res. Ther. **25** (2023) 164, doi: https://doi.org/10.1186/s13075-023-03152-1.
77. *M. W. Collins, K. G. Saag, J. A. Singh*, Is there a role for cherries in the management of gout, Ther. Adv. Musculoskelet. Dis. **11** (2019), doi: https://doi.org/10.1177/1759720X19847018.
78. *N. Acero, A. Gradillas, M. Beltran, A. García, D. Muñoz Mingarro*, Comparison of phenolic compounds profile and antioxidant properties of different sweet cherry (*Prunus avium* L.) varieties, Food Chem. **279** (2019) 260–271, doi: https://doi.org/10.1016/j.foodchem.2018.12.008.
79. *R. Wang, F. Zhang, S. Zan, C. Gao, C. Tian, X. Meng*, Quality Characteristics and Inhibitory Xanthine Oxidase Potential of 21 Sour Cherry (*Prunus cerasus* L.) Varieties Cultivated in China, Front. Nutr. **8** (2021) 796294, doi: https://doi.org/10.3389/fnut.2021.796294.
80. *K. L. Lamb, A. Lynn, J. Russell, M. E. Barker*, Effect of tart cherry juice on risk of gout attacks: Protocol for a randomised controlled trial, BMJ Open **10** (2020) e035108, doi: https://doi.org/10.1136/bmjopen-2019-035108.
81. *A. A. Farooqi, S. Khalid, F. Tahir, U. Y. Sabitaliyevich, I. Yaylim, R. Attar, B. Xu*, Bitter gourd (*Momordica charantia*) as a rich source of bioactive components to combat cancer naturally: Are we on the right track to fully unlock its potential as inhibitor of deregulated signaling pathway, Food Chem. Toxicol. **119** (2018) 98–105, doi: https://doi.org/10.1016/j.fct.2018.05.024.
82. *J. K. Grover, S. P. Yadav*, Pharmacological actions and potential uses of *Momordica charantia*: a review, J. Ethno. **93** (2004) 123–132, doi: https://doi.org/10.1016/j.jep.2004.03.035.
83. *M. M. Ali, I. H. Borai, H. M. Ghanem, A. H. Abdel-halim, F. M. Mousa*, Biomedicine and Pharmacotherapy The prophylactic and therapeutic effects of Momordica charantia methanol extract through controlling different hallmarks of the hepatocarcinogenesis, Biomed. Pharmacother. **98** (2018) 491–498, doi: https://doi.org/10.1016/j.biopha.2017.12.096.
84. *B. Svobodova, L. Barros, R. C. Calhelha, S. Heleno, M. Jose, S. Walcott, M. Bittova, V. Kuban, I. C. F. R. Ferreira*, Bioactive properties and phenolic profile of *Momordica charantia* L. medicinal plant growing wild in Trinidad and Tobago, Ind. Crop. Prod. **95** (2017) 365–373, doi: https://doi.org/10.1016/j.indcrop.2016.10.046.
85. *B. Xu, Z. Li, T. Zeng, J. Zhan, S. Wang, C.-T. Ho, S. Li*, Bioactives of Momordica charantia as Potential Anti-Diabetic/Hypoglycemic Agents, Molecules **27** (2022) 2175, doi: https://doi.org/https:// doi.org/10.3390/molecules27072175.
86. *Y. Jiang, X. R. Peng, M. Y. Yu, L. S. Wan, G. L. Zhu, G. T. Zhao, L. Zhou, M. H. Qiu, J. Liu*, Cucurbitane-type triterpenoids from the aerial parts of *Momordica charantia* L, Phytochem. Lett. **16** (2016) 164–168, doi: https://doi.org/10.1016/j.phytol.2016.04.007.
87. *S. Hussain, N. Jahan, M. Or, M. Salim, U. Chen, N. Rahman*, Antihyperlipidemic screening and plasma uric acid reducing potential of Momordica charantia seeds on Swiss albino mice model, Heliyon **5** (2019) e01739, doi: https://doi.org/10.1016/j.heliyon.2019.e01739.
88. *Z. Y. Lin, X. Liu, F. Yang, Y. Yu*, Structural characterization and identification of five triterpenoid saponins isolated from Momordica cochinchinensis extracts by liquid chromatography/tandem mass spectrometry, Int. J. Mass Spectrom. **32** (2012) 43–66, doi: https://doi.org/10.1016/j.ijms.2012.07.022.
89. *C. H. Liu, M. H. Yen, S. F. Tsang, K. H. Gan, H. Hsu, Y., C. N. Lin*, Antioxidant triterpenoids from the stems of Momordica charantia, Food Chem. **118** (2010) 751–756, doi: https://doi.org/10.1016/j.foodchem.2009.05.058.
90. *L. Sun, X. Zhang, L. Dong, C. Zhang, P. Guo, C. Wu*, The triterpenoids of the bitter gourd (*Momordica charantia*) and their pharmacological activities: A review, J. Food Compos. Anal. **96** (2021) 103726, doi: https://doi.org/10.1016/j.jfca.2020.103726.
91. *D. Singh, K. Kumari, S. Ahmed,* Chapter 17 - Natural herbal products for cancertherapy, In Understanding Cancer, 2022, pp. 257–268, doi: https://doi.org/10.1016/B978-0-323-99883-3.00010-X.
92. *I. Shabir, V. K. Pandey, R. Shams, A. H. Dar, K. K. Dash, S. A. Khan, I. Bashir, G. Jeevarathinam, A. V. Rusu, T. Esatbeyoglu, R. Pandiselvam*, Promising bioactive properties of quercetin for potential food applications and health benefits: A review, Front. Nutr. **30** (2022) 999752, doi: https://doi.org/10.3389/fnut.2022.999752.
93. *J. Chen, J. Chen, B. Feng, M. Ning, W. Wu, S. Zou*, Investigation of resveratrol as a xanthine oxidase inhibitor: Mechanistic insights and therapeutic implications for gout and hyperuricemia, Biotechnol. Appl. Biochem. **3** (2025) 695–708, doi: https://doi.org/10.1002/bab.2690.
94. *H. Rocha-Gonzalez, M. Ambriz-Tututi, V. Granados-Soto*, Resveratrol: A Natural Compound with Pharmacological Potential in Neurodegenerative Diseases. CNS Neurosci. Ther. **14** (2008) 234–247, doi: https://doi.org/10.1111/j.1755-5949.2008.00045.x.

## SAŽETAK

### Terapeutski bioaktivni spojevi ljekovitih i jestivih biljaka u liječenju hiperurikemije

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Hiperurikemija, čest metabolički poremećaj, obilježena je neuobičajeno visokim koncentracijama mokraćne kiseline u krvi. To stanje nastaje ponajprije zbog pretjeranog stvaranja mokraćne kiseline, smanjene bubrežne eliminacije ili kombinacije oboje. Osim dobro poznate povezanosti s giht-om, hiperurikemija doprinosi i razvoju brojnih drugih zdravstvenih komplikacija. Iako standardni farmakološki tretmani pružaju terapijsku korist, njihovi nuspojavni profili mogu predstavljati rizik za pacijente.

Ovaj pregledni rad istražuje uobičajene ljekovite i jestive biljke s dokazanim učinkom snižavanja mokraćne kiseline s naglaskom na njihove aktivne sastojke. Ti bioaktivni spojevi djeluju kroz više mehanizama, uključujući inhibiciju sinteze mokraćne kiseline, poticanje bubrežnog izlučivanja te smanjenje upalnih reakcija. Procjenom terapijskog potencijala biljnih bioaktivnih spojeva, rad pruža vrijedan uvid u alternativne strategije liječenja hiperurikemije.

**Ključne riječi**
*Anti-giht, antioksidans, protuupalno djelovanje, mokraćna kiselina, ksantin-oksidaza*

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Pregled
*Prispjelo 30. ožujka 2025.*
*Prihvaćeno 2. lipnja 2025.*