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REVIEW ARTICLE

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Plant-Derived Bioactive Compounds Mitigate Diabetes Globally: An Updated Mini Review

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Abstract

Diabetes mellitus (DM) is one of the most vital health crises across the world in the 21st century, as it affects more than 537 million adults in the world and is projected to increase to 783 million individuals by 2045. The existing pharmacological treatments, such as metformin, sulfonylureas, and insulin therapy, though effective, have significant drawbacks, including gastrointestinal adverse effects, hypoglycemic events, weight gain, and diminishing efficacy over time. Bioactive compounds of plant origin have emerged as new therapeutic options, offering multi-targeted supplementation with a good safety profile. This review provides an in-depth analysis of key groups of antidiabetic phytochemicals, including phenolics (gallic acid, chlorogenic acid), flavonoids (quercetin, kaempferol, anthocyanins), alkaloids (berberine, trigonelline), and terpenoids (ginsenosides, oleanolic acid). Recent discoveries between 2024 and 2026 show significant potential to modulate glucose homeostasis through AMP-activated protein kinase (AMPK), α-glucosidase, β-cell protection, and the anti-inflammatory pathway. Potential drugs such as *Gymnema sylvestre*, *Momordica charantia*, *Azadirachta indica*, and *Celtis tetrandra* have good preclinical and clinical results. Although bioavailability, standardization, and clinical translation are challenging, plant-based bioactive compounds are promising next-generation antidiabetic therapeutic agents, as monotherapy or as a supplement to the current treatment.(**International Journal of Biomedicine. 2026;16(2):151-156.**)

Keywords: diabetes mellitus • bioactive compounds • phenolics • flavonoids • alkaloids • AMP-activated protein kinase • medicinal plants

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Introduction

Diabetes mellitus (DM) is a heterogeneous group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, and both.[1] The prevalence of diabetes nowadays is at epidemic levels, and the International Diabetes Federation (IDF) estimates 537 million cases in 2021, and it will reach 783 million by 2045.[2] This increasing rate of prevalence has high economic costs, as the global healthcare spending reaches USD 966 billion yearly.

There are multiple pathophysiological entities associated with diabetes. Type 1 diabetes mellitus (T1DM) is an autoimmune destruction of β-cells in the pancreas, requiring lifelong insulin replacement. Type 2 diabetes mellitus (T2DM) is a progressive insulin resistance disease associated with defective β-cells and 90-95% of all diseases. Gestational diabetes mellitus (GDM) is an illness that arises during pregnancy, placing both mother and child at risk of developing serious metabolic disorders. Maturity-onset diabetes of the young (MODY) is a monogenic disorder caused by mutations in genes that regulate β-cell function.[3]

Existing treatment approaches are largely limited. The pharmacotherapy of T2DM, which is the first-line option, Metformin, is most commonly associated with gastrointestinal intolerance, manifesting as diarrhea and nausea, and thus discontinuations are as high as 5-10% in patients. Sulfonylureas encourage hypoglycemia and weight gain, and thiazolidinediones heighten cardiovascular risks and fluid retention. T1DM and advanced T2DM insulin therapy are vital and necessitate close observation and carry the threat of hypoglycemia. Moreover, several traditional agents have shown a decreasing efficacy with time owing to progressive β-cell failure.[4]

Such restrictions have prompted the exploration of medicinal plants and their bioactive compounds. The use

of botanical preparations in the management of diabetes has a history of thousands of years in traditional medicine, which includes Ayurveda, Traditional Chinese Medicine, and Arabic medicine. In modern pharmacognosy, some bioactive molecules responsible for the aforementioned antidiabetic effects have been identified, enabling mechanistic evaluation of these effects and the application of therapeutic strategies in a standardized manner. The mentioned effects of plant-derived compounds include the following: multi-targeted activity, reduced toxicity, and potential β-cell protection.[5]

This review aims to present a timely overview of recent developments (2024-2026) in the field of plant-derived bioactive compounds for the management of diabetes, with a focus on mechanistic insights, clinical trials, and potential therapeutic implications.

Plant-Derived Bioactive Compounds with Antidiabetic Potential

Phytochemicals are secondary metabolites produced by plants to protect against external stressors. These compounds have developed complex molecular structures that facilitate interaction with mammalian biological targets, including glucose homeostatic biological targets. The significant classes that have antidiabetic effects are:

The phenolic compounds are aromatic secondary metabolites with hydroxylated benzene rings. This heterogeneous group consists of phenolic acids (gallic acid, caffeic acid, chlorogenic acid), stilbenes (resveratrol), and lignans. Their antidiabetic effect is mediated by antioxidant activity, enzyme inhibition, and alterations in glucose transporters.[6]

The largest group of polyphenols is the flavonoids, which share the diphenylpropane (C6-C3-C6) skeleton. They have subclasses of flavonols (quercetin, kaempferol), flavones (apigenin), flavanols (catechins), and anthocyanins (cyanidin-3-glucoside). Such compounds show insulin secretagogue effects, β-cell protection, and anti-inflammatory properties.

Alkaloids are nitrogen-based basic compounds that are based on the amino acid precursors. Some well-known antidiabetic alkaloids include berberine (isoquinoline type), trigonelline (pyridine type), and piperine. These molecules first and foremost activate AMP-activated protein kinase (AMPK), which regulates cellular energy metabolism.[7]

Saponins and terpenoids are derivatives of isoprene, such as triterpenes (oleanolic acid, ursolic acid) and saponins that are steroids (ginsenosides). These chemicals improve insulin sensitivity, maintain lipid metabolism, and prevent diabetic complications.[7]

The general mechanisms underlying the antidiabetic effects of these compounds are: (1) blockage of carbohydrate-digesting enzymes (α-amylase, α-glucosidase); (2) enhancement of insulin secretion and regeneration of β-cells; (3) promotion of peripheral glucose uptake through GLUT4 translocation; (4) inhibition of hepatic gluconeogenesis; (5) inhibition of oxidative stress and inflammation; and (6) alteration of gut microbiota.[8]

Major Classes of Antidiabetic Bioactive Compounds

Phenolic Compounds

Phenolic acids are important antidiabetic agents, and their mechanisms are well documented. Chlorogenic acid, which is present in large quantities in coffee, apples, and blueberries, exhibits strong hypoglycemic and hypolipidemic properties. Recent reports show that chlorogenic acid inhibits α-amylase and enhances insulin action in antioxidant and anti-inflammatory mechanisms. Gallic acid and caffeic acid exhibit similar enzyme-inhibitory profiles; caffeic acid shows stronger α-glucosidase inhibitory activity (IC50 8.00 ± 0.40 mg/mL) than the standard drugs. Rosmarinic acid, a compound extracted from *Rosmarinus officinalis* and other species of the *Lamiaceae* family, increases insulin sensitivity by regulating the expression of GLUT4 and suppressing phosphoenolpyruvate carboxykinase (PEPCK). The molecular docking studies show that rosmarinic acid binds to the α-glucosidase active site, forming stable hydrogen bonds with binding energies similar to those of acarbose. According to recent studies, rosmarinic acid modulates the activity of glycogen phosphorylase and glycogen synthase in diabetic animals and restores hepatic and muscular glycogen levels after 30 days of administration.

Phenolic antioxidant capacity is very instrumental in alleviating diabetic complications. These scavenge reactive oxygen species (ROS), prevent the formation of advanced glycation end-products (AGEs), and chelate transition metals (Fe^{2+}, Cu^{2+}) that catalyze oxidative reactions. Quercetin and resveratrol, in particular, exhibit AGE-inhibitory activity, trapping reactive dicarbonyl compounds and inhibiting protein glycation.[6]

Flavonoids

The variety of antidiabetic mechanisms of flavonoids is impressive. Quercetin, which is rich in onions, apples, and broccoli, exhibits a variety of activities, including inhibition of DPP-4 (IC50 1.150 mg/mL), α-glucosidase, and β-cell apoptosis. According to recent mechanistic research, quercetin alleviates endoplasmic reticulum stress and oxidative harm in pancreatic β-cells, maintaining insulin production and secretion.[9]

Among the flavonols with a promising future is kaempferol, according to research in 2024-2025. In vitro experiments indicate that kaempferol inhibits α-glucosidase and α-amylase with IC 50 values of 2.33 mg/mL and 52.95mg/mL, respectively. Kaempferol enhances hexokinase activity in both skeletal muscle and liver in streptozotocin-induced diabetic mice but inhibits hepatic gluconeogenesis through the inhibition of pyruvate carboxylase. It is important to note that kaempferol upregulates autophagy in β-cells, which helps defend against lipotoxic injury by maintaining lipid homeostasis.[10]

The pigmented flavonoids, anthocyanins, that color berries and grape plants blue, purple, and red have shown specific protective effects on pancreatic β-cell function. A

systematic review of in vitro studies of cyanidin-3-glucoside (C3G) found that the compound can enhance β-cell function under conditions of glucotoxicity and oxidative stress, alleviating endoplasmic reticulum (ER) stress, decreasing apoptosis, and promoting insulin production. Cyanidin-3-rutinoside (C3R) stimulates insulin release by elevating intracellular Ca^{2+} and raising ATP synthesis by glucokinase up-regulation.

Anthocyanins at the transcriptional level activate genes essential to β-cells (Ins1/Ins2, Slc2a2 (GLUT2), Gck (glucokinase), and the transcription factor PDX-1) and regulate ion channel genes (Cav1.2, Kir6.2), thereby affecting membrane excitability and insulin exocytosis. These data are consistent with larger studies on dietary polyphenols that protect β-cells against metabolic stress, but not by acting on individual molecular targets.[11]

Alkaloids

One of the most studied plant-derived antidiabetic agents is berberine, an isoquinoline alkaloid found in *Coptis chinensis*, *Berberis* species, and the bark of *Phellodendron (Phellodendron amurense tree*). New 2024-2025 studies have clarified the advanced mechanisms of action underlying berberine's therapeutic effects. The compound triggers the AMP-activated protein kinase (AMPK) via LKB1-mediated phosphorylation, thereby regulating several metabolic processes simultaneously.

Berberine-induced increases in AMPK activity in skeletal muscle and adipose tissue stimulate GLUT4 translocation to the plasma membrane to enable glucose uptake, irrespective of insulin receptor signaling, and are significantly beneficial in insulin-resistant conditions. Berberine inhibits gluconeogenesis hepatically through the AMPK-TORC2 pathway by reducing PEPCK and glucose-6-phosphatase (G6Pase). Recent randomized clinical trials with the gut-targeted berberine ursodeoxycholate co-crystal (HTD1801) showed AMPK activation and inhibition of the gluconeogenic gene signature, with 0.5 percentage point reductions in HbA1c compared to placebo.[12]

In addition to glucose homeostasis, berberine has lipid-lowering effects distinct from those of statins, enhancing LDR expression and cholesterol clearance. Berberine affects adipokine profiles as well, lowering pro-inflammatory leptin and MCP-1 levels and increasing adiponectin levels, thereby improving insulin sensitivity.

The main alkaloid of fenugreek (*Trigonella foenum-graecum)*, trigonelline, is the antidiabetic material that activates AMPK and regulates carbohydrate metabolism. Recent results suggest that trigonelline improves glucose tolerance and insulin sensitivity in experimental animals, although clinical evidence is less robust than that for berberine.[13]

Terpenoids and Saponins

Panax ginseng contains triterpene saponins (ginsenosides), which have a complex metabolism. These substances enhance insulin sensitivity by stimulating the AMPK system and PI3K/Akt pathway. Recent network pharmacology research indicates that ginsenosides Rb1 and Rg1 are among the most important bioactive compounds that interact with various proteins, such as PPAR-g, IRS-1, and GLUT4, in diabetes.

Oleanolic acid is a pentacyclic triterpene that is abundant in olive leaves, garlic, and other medicinal plants, which has been shown to have an antidiabetic effect by both increasing insulin secretion by pancreatic β-cells and reducing peripheral insulin resistance. The compound binds to the TGR5 receptor, activating GLP-1 release and energy consumption while preventing β-cell oxidative stress-related apoptosis.

Momordica charantia (bitter melon) saponins, such as charantin and momordicosides, have insulin-like properties and inhibit intestinal glucose absorption. Recent molecular docking studies show that these compounds also bind several targets in diabetes, such as β-glucosidase and protein tyrosine phosphatase 1B (PTP1B).

Antidiabetic Medicinal Plants

Gymnema sylvestre

Gymnemic acids and gurmarin peptides are present in *Gymnema sylvestre* (an herb used in Ayurvedic medicine), which has potent antidiabetic effects, as the plant is known as gurmar (sugar destroyer). Recent network pharmacology and molecular docking studies in 2024 have identified bioactive substances and their molecular targets. Gymnemic acids exhibit α-glucosidase-inhibitory action and restore the pancreatic β-cells in rodent diabetic models. The intestinal glucose absorption is decreased, and the endogenous insulin secretion is increased by the plant extracts, which act by modulating ATP-sensitive potassium channels.[14]

Momordica charantia

Momordica charantia is among the most widely researched antidiabetic plants worldwid. It has charantin (steroidal saponin), vicine, and polypeptide-p (insulin of the plants) in its fruit.[15] Recent combined computational analysis based on network pharmacology and molecular docking has helped elucidate the molecular pathophysiology underlying its antidiabetic action by identifying key targets, including PI3K, Akt, and MAPK signaling pathways. Some trials show substantial decreases in fasting blood glucose and HbA1c values comparable to those with metformin.

Azadirachta indica

Neem (*Azadirachta indica*) is a plant that incorporates azadirachtin, nimbin, and nimbidin with proven hypoglycemic properties. The extracts of leaves and bark enhance insulin sensitivity and are powerful antioxidants and anti-inflammatory agents. Recent studies have focused on neem's capacity to regulate the Nrf2/ARE antioxidant pathway and to prevent NF-kB-mediated inflammation in diabetic tissues.[5]

Celtis tetrandra

Celtis tetrandra Roxb. is a member of the Cannabaceae family and a promising antidiabetic plant according to recent 2024-2025 studies. Profiling of gas chromatography-mass spectrometry has revealed the presence of various

bioactive compounds, including flavonoids, phenolic acids, and terpenoids, with antimicrobial, anti-inflammatory, and cytotoxic properties.[16] Leaf extracts showed strong antidiabetic and antihyperlipidemic effects in diabetic rats, improving lipid profiles and characterized by protective histopathological changes in pancreatic tissue. The results contribute to the established use of *C. tetrandra* in the management of diabetes and warrant further clinical study.

Other medicinal plants, their bioactive compounds, and reported antidiabetic activity are shown in Table 1.

Mechanisms of Antidiabetic Action

Plant-derived bioactive compounds exert antidiabetic effects through multiple organ-specific mechanisms, as illustrated in Figure 1, and the details are described next.

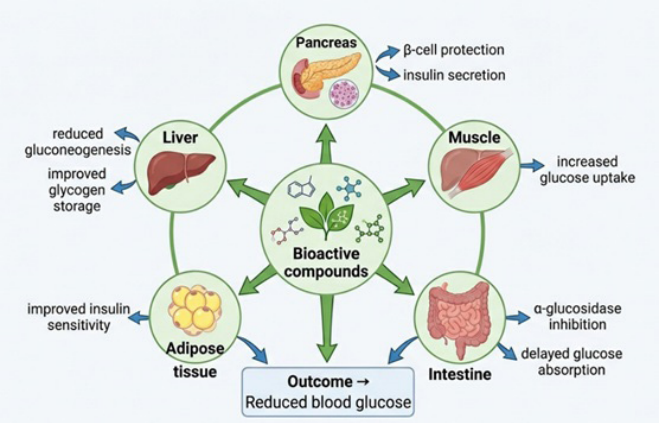

Fig. 1. *Organ-specific mechanisms of antidiabetic action of plant-derived bioactive compounds. Bioactive phytochemicals induce antihyperglycemic effects in the body by acting on several mechanisms, such as stimulation of insulin secretion and β-cells in the pancreas, and enhancing glucose uptake in skeletal muscle, α -glucosidase inhibition and slowed glucose absorption in the intestine, decreased hepatic gluconeogenesis and glycogen storage in the liver, and enhanced insulin sensitivity of adipose tissue, which results in low levels of blood glucose.*

Insulin Secretion Stimulation

Plant bioactive compounds stimulate insulin secretion through multiple pathways. Sulfonylurea-like mechanisms involve closure of ATP-sensitive potassium (KATP) channels on β-cell membranes, causing depolarization and calcium influx. However, unlike synthetic sulfonylureas, phytochemicals such as gymnemic acids and naringenin exhibit glucose-dependent insulin secretion, thereby reducing the risk of hypoglycemia. Recent studies reveal that anthocyanins upregulate glucokinase (GCK) expression, enhancing glucose sensing and ATP production in β-cells.[17]

β-Cell Regeneration and Protection

Chronic hyperglycemia induces β-cell apoptosis through oxidative stress, ER stress, and inflammation. Flavonoids, including quercetin and kaempferol, protect β-cells by upregulating antioxidant enzymes (SOD, CAT, GPx), suppressing caspase-3 activation, and promoting autophagy. Berberine stimulates mitochondrial biogenesis through the AMPK-SIRT1-PGC-1α axis, restoring β-cell energy metabolism.

Enzyme Inhibition

Inhibition of α-glucosidase and α-amylase enzymes slows carbohydrate digestion, thereby lowering blood glucose levels. The phenolic compounds exhibit non-competitive inhibition, unlike acarbose, and are not affected by substrate concentration. The structure-activity relationship shows that hydroxylation of the flavonoids is responsible for the increased inhibitory potential.[7]

Glucose Uptake Enhancement

Activation of the AMPK signaling pathway is a core mechanism for improving glucose uptake. Berberine, resveratrol, and galegine activate the AMPK signaling pathway by inhibiting mitochondrial Complex I and increasing the AMP/ATP ratio. Activated AMPK increases GLUT4 translocation to the plasma membrane in skeletal muscle and adipose tissue, thereby increasing glucose uptake independently of insulin action. Moreover, rosmarinic acid activates the AMPK signaling pathway in L6 muscle cells and increases glucose uptake.[18]

Antioxidant Activity

Hyperglycemia-induced oxidative stress drives diabetic complications through ROS generation and AGE formation. Phenolic compounds scavenge free radicals, chelate metal ions, and inhibit lipid peroxidation. Quercetin and resveratrol specifically inhibit the Maillard reaction, preventing protein glycation and AGE accumulation.

Anti-Inflammatory Pathways

Chronic low-grade inflammation characterizes T2DM, with elevated TNF-α, IL-6, and IL-1β contributing to insulin resistance. Flavonoids suppress NF-κB activation and MAPK signaling, reducing proinflammatory cytokine production. Berberine activates the AMPK-SIRT1 axis, suppressing NF-κB and limiting MCP-1 production in adipose tissue.[17]

Many medicinal plants and their bioactive compounds have demonstrated significant antidiabetic activity and exhibit diverse mechanisms of action, as shown in Table 1.

Challenges and Future Perspectives

Although preclinical evidence of the ability of plant-derived antidiabetic compounds to be translated into clinical settings appears promising, a number of obstacles hinder such translation:

Revealing Clinical Trials: Although berberine has been extensively studied in clinical trials, most plant compounds have not undergone rigorous randomized controlled trials across various population groups. Most of the evidence has been based on in vitro research and small-animal models, which require large-scale human studies to determine efficacy and safety profiles.

Standardization Problems: Botanical extracts exhibit high batch-to-batch variation in bioactive compound content

due to geographical location, harvest timing, and processing procedures. Phytochemical profile standardization and the implementation of quality control markers remain the keys to consistent therapeutic responses.

Bioavailability Limits: Oral bioavailability of many polyphenols and alkaloids is also low due to low aqueous solubility, high first-pass metabolism, and P-glycoprotein-mediated efflux. This is one such instance of berberine in which oral bioavailability is less than 1%. High-tech drug delivery systems—such as nanoparticles, liposomes, and phospholipid complexes—can enhance absorption and ensure targeted delivery to tissues.

Toxicity and Dosage Concerns: High doses can cause hepatotoxicity, nephrotoxicity, or drug interactions, though these are considered safe. It is necessary to obtain systematic toxicological profiling and calculate therapeutic indices.

Regulatory Frameworks: The regulatory pathways for Herbal medicines have become complicated, with specifications for consistency, purity, and stability similar to those for synthetic pharmaceuticals.

The future directions include (1) applying network pharmacology and systems biology to identify synergistic combinations of compounds; (2) developing semi-synthetic analogs with improved pharmacokinetics; (3) combining metabolomics analysis with gut microbiome analysis to personalize phytotherapy; (4) adopting the precision medicine-based approaches that are based on genetic variants of drug metabolism.

Conclusion

Plant-derived bioactive compounds offer a useful therapeutic pool for the management of diabetes, providing multi-target effects that address the complex pathophysiology of metabolic disease. Recent developments between 2024 and 2026 have greatly enhanced our understanding of the mechanisms by which phenolics, flavonoids, alkaloids, and terpenoids regulate glucose homeostasis by activating AMPK, inhibiting enzymes, protecting β cells, and mediating anti-inflammatory responses. Medicinal plants, such as *Gymnema sylvestre*, *Momordica charantia*, and newer ones which show clinical promise through mechanistic research, include *Celtis tetrandra*. Although bioavailability, standardization, and clinical validation remain challenging, these natural compounds offer promising opportunities for developing safer, more effective treatments for diabetes. To maximize the therapeutic potential of plant-derived bioactive compounds for managing diabetes worldwide, future studies should focus on rigorous clinical trials, sophisticated formulations, and their combination with precision medicine to achieve the desired outcome.

Table 1.

An overview of plant-based bioactive compounds with documented antidiabetic properties, their sources, key phytochemical compounds, experimental models, and mechanism of action, and significant results of major studies.

Plant source	Bioactive compound(s)	Experimental model	Mechanism of action	Key findings	Reference
Momordica charantia	Charantin, polypeptide-p	Diabetic animal models	Insulin secretion, glucose uptake	Reduced blood glucose	15
Gymnema sylvestre	Gymnemic acids	Animal models	β-cell regeneration	Improved insulin secretion	14
Azadirachta indica	Nimbin, flavonoids	In vitro / in vivo	α-glucosidase inhibition	Reduced hyperglycemia	5
Celtis tetrandra	Phenolics, flavonoids	Alloxan-induced rats	Antioxidant, β-cell protection	Improved pancreatic histology	16
Berberis vulgaris	Berberine	Clinical & animal	AMPK activation	Reduced HbA1c	12
Camellia sinensis	Catechins (EGCG)	Animal studies	Antioxidant, insulin signaling	Improved glucose metabolism	19
Curcuma longa	Curcumin	Animal models	Anti-inflammatory	Reduced insulin resistance	5
Allium sativum	Allicin	Diabetic rats	Insulin secretion	Lower blood glucose	20
Trigonella foenum-graecum	Trigonelline	Human & animal	Insulin sensitivity	Reduced fasting glucose	5
Panax ginseng	Ginsenosides	Animal model	β-cell protection	Improved glycemic control	19
Aloe vera	Aloin	Animal model	Glucose uptake	Reduced blood glucose	20
Ocimum sanctum	Eugenol	Animal model	Antioxidant	Improved glucose tolerance	20
Zingiber officinale	Gingerols	Animal model	Insulin sensitivity	Lower glucose levels	19

Author Contributions

Faiza Siddique: Data curation and analysis, Writing – review and editing.
Duaa Qaiser: Data curation and analysis, Writing – review and editing.
Tahir Mehmood: Supervision, Writing – review and editing.
Faiza Siddique and **Duaa Qaiser** contributed equally and share first authorship.
All authors have approved the final article.

Conflict of Interest

The authors have declared no conflict of interest.

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