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Review Paper

Antidiabetic Potential of *Berberis aristata*, *Glycyrrhiza glabra* and *Terminalia chebula*: Phytochemical and Pharmacological Perspectives

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ABSTRACT

Diabetes mellitus (DM) is a chronic metabolic condition that is becoming more common worldwide and has significant treatment hurdles. Traditional plant-based medicines have gained popularity due to the limitations of contemporary methods. Licorice (*Glycyrrhiza glabra*), Indian barberry (*Berberis aristata*), and haritaki (*Terminalia chebula*), which are utilized in Ayurvedic and Chinese medicine for diabetes, exhibit encouraging antidiabetic qualities. Berberine and similar isoquinoline alkaloids (*B. aristata*), glycyrrhizin saponins and flavonoids (*G. glabra*), and hydrolyzable tannins (*T. chebula*, including chebulagic/chebulinic acids) are among the many antidiabetic phytochemicals found in these herbs. Mechanistically, berberine suppresses hepatic gluconeogenesis and lipogenesis, increases GLUT4 translocation and glycolysis, and activates AMP-activated protein kinase (AMPK) to produce multi-target actions. Glycyrrhizin, glabridin, and other components of licorice increase insulin secretion, encourage GLUT4-mediated uptake and α -glucosidase inhibition, and lessen oxidative stress. *T. chebula* tannins activate PPAR γ /GLUT4 pathways and strongly block intestinal α -amylase/ α -glucosidase (blunting postprandial glucose), while their antioxidant/glycation-inhibitory activities shield β -cells. In type 2 diabetes patients, berberine-containing extracts (e.g., 0.5–1.5 g/day) significantly lower fasting glucose, HbA1c, and lipids; however, there is little human data for licorice and *T. chebula* (frequently in polyherbal formulations). Safety profiles differ: excessive glycyrrhizin can result in pseudoaldosteronism; *T. chebula* extracts seem safe in animal tests; berberine is typically well-tolerated but contraindicated in pregnancy/G6PD deficiency. Therefore, these botanicals have therapeutic potential for managing diabetes; nevertheless, more carefully planned clinical trials and extract standardization are required to verify their safety and effectiveness.

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INTRODUCTION

Diabetes mellitus (DM) is a chronic, diverse metabolic disease characterized by persistent hyperglycemia caused by deficits in insulin secretion, action, or both. The main biochemical abnormality in diabetes mellitus is an elevated blood glucose level caused by disturbances in the metabolism of proteins, lipids, and carbs. Long-term hyperglycemia, which also directly causes tissue damage, dysfunction, and failure in several organ systems, including the heart, kidneys, eyes, nerves, and vasculature, is the source of the disease's progressive nature. Persistent hyperglycemia and the associated metabolic dysfunction are associated with an increased risk of morbidity and early death worldwide, placing a heavy burden on both individuals and society. These consequences have made DM a significant global public-health challenge of the 21st century^[1]. Clinical manifestations of diabetes mellitus range widely, from acute metabolic emergencies such as diabetic ketoacidosis to asymptomatic hyperglycemia found by chance during routine screening. The pathophysiology of diabetes mellitus (DM) involves complex interplay between genetic predisposition, environmental triggers, and lifestyle variables, regardless of how the disease manifests. The balance between β -cell activity, insulin sensitivity, and compensatory metabolic responses to food loads is reflected in the phenotypic presentation of diabetes. Accurately classifying diseases, developing successful treatment plans, and forecasting long-term clinical outcomes all depend on an understanding of this intricate interaction^[2].

Classification of Diabetes Mellitus

Targeted clinical therapy, epidemiologic research, and comprehension of disease mechanisms all depend on the accurate classification of diabetes mellitus. Modern classification schemes prioritize

etiologic groups based on underlying pathophysiology, clinical manifestation, and particular diagnostic standards. In the past, diabetes mellitus (DM) was classified as either "insulin-dependent" or "non-insulin-dependent," but contemporary frameworks differentiate between several categories that represent a range of pathogenic mechanisms^[3].

1. Type 1 Diabetes Mellitus (T1DM)

An utter lack of insulin results from the autoimmune-mediated death of pancreatic β -cells in type 1 diabetes mellitus. HLA class II genotypes that confer vulnerability and circulating islet autoantibodies against β -cell antigens are commonly linked to the autoimmune process. Despite having a peak onset in childhood and adolescence, type 1 diabetes can manifest at any age and occasionally be mistaken for type 2 diabetes in adults. To maintain glycemic control and avoid ketoacidosis, people with type 1 diabetes need exogenous insulin for the rest of their lives^[4].

2. Type 2 Diabetes Mellitus (T2DM)

About 85–95% of adult diagnoses of diabetes worldwide are type 2 diabetes mellitus. Peripheral insulin resistance and relative reduction in insulin secretion combine to cause type 2 diabetes. Pancreatic β -cells produce more insulin in the early stages to make up for decreased tissue sensitivity; as β -cell dysfunction progresses, insufficient insulin is produced in comparison to metabolic demand. Major risk factors for the development of type 2 diabetes include obesity, physical inactivity, aging, and genetic predispositions^[5].

3. Gestational Diabetes Mellitus (GDM)

When glucose intolerance is initially identified during pregnancy, usually in the second or third



trimester, it is referred to as gestational diabetes mellitus. Increased insulin resistance brought on by placental hormones and metabolic changes during pregnancy is the cause of GDM. GDM can result in problems like preeclampsia, macrosomia, and neonatal hypoglycemia if it is not well treated. Additionally, women with GDM are more likely to develop type 2 diabetes in the future^[6].

Pathophysiology of Diabetes Mellitus

Chronic hyperglycemia and metabolic dysregulation are the results of a complex interaction between genetic, molecular, metabolic, and environmental variables in the pathogenesis of diabetes mellitus (DM). While high plasma glucose is a common clinical symptom of diabetes, different kinds of diabetes have different underlying causes. Insulin shortage is absolute in type 1 diabetes (T1DM) due to autoimmune death of pancreatic β -cells; relative insulin insufficiency is caused by a combination of peripheral insulin resistance and increasing β -cell malfunction in type 2 diabetes (T2DM). Additional mechanistic factors, such as placental hormones, genetic polymorphisms, medications, or pancreatic pathology, are involved in gestational diabetes and other particular types of diabetes. Through processes including glucotoxicity, lipotoxicity, inflammation, and oxidative stress, chronic glucose and lipid disruption exacerbate metabolic abnormalities in all forms, resulting in a vicious cycle that quickens the course of the disease and its repercussions^[7]. Diabetes mellitus (DM) affects hundreds of millions of people worldwide and can have major consequences if left untreated. Research on medicinal plants with glucose-lowering properties has increased due to the shortcomings of current treatments and interest in natural medicines. *Berberis aristata* (Indian barberry), *Glycyrrhiza glabra* (licorice), and *Terminalia chebula* (haritaki) have long been used to treat metabolic diseases in Ayurvedic and

traditional Chinese medicine^[8]. This review compares the bioactive components, antidiabetic processes, and supporting data of these three species. Improved glucose regulation has been associated with glycyrrhizin saponins/flavonoids (in *G. glabra*), hydrolyzable tannins (in *T. chebula*), and berberine alkaloids (in *B. aristata*)^{[8][9]}.

Botanical and Phytochemical Profiles

Berberis aristata (Indian Barberry)

Native to the Himalayas, *B. aristata* (family Berberidaceae) is a spiky shrub that has long been utilized in Ayurveda to treat "pittaja" conditions like diabetes. Isoquinoline alkaloids, primarily berberine, palmatine, and berbamine, are produced by its roots and stem bark and are thought to be the main bioactive substances. Berberine is plentiful and responsible for the majority of pharmacological actions, according to contemporary research. Its historical use is supported by clinical research that show benefits in glycemic and lipid profiles, frequently using standardized berberine formulations^[8].

Glycyrrhiza glabra (Licorice)

Ayurveda and Traditional Chinese medicine both respect the climbing herb *G. glabra* (Leguminosae/Fabaceae). Triterpenoid saponins (glycyrrhizin, glycyrrhetic acid) and flavonoids (liquiritin, isoliquiritigenin, glabridin) are found in the sweet root^[9]. Preclinical research has demonstrated the anti-inflammatory and antioxidant properties of glycyrrhizinic acid and flavonoids, as well as their ability to enhance aspects of glucose metabolism. Although licorice extracts have been utilized as metabolic tonics and demulcents, there is little clinical data supporting their efficacy in diabetes^[9].



Terminalia chebula (Haritaki)

One significant Ayurvedic plant that is frequently utilized in the triphala formulation is *T. chebula* (Combretaceae). Chebulinic acid, chebulagic acid, gallic acid, corilagin, and ellagic acid are among the hydrolyzable tannins found in its ripe fruit. These polyphenols provide potent anti-glycation and antioxidant qualities. According to experimental research, *T. chebula* extracts (or isolated chebulagic acid) significantly reduce postprandial glucose spikes by inhibiting intestinal α -glucosidase/ α -amylase enzymes^[10]. Additionally, in vivo research on diabetes mice shows improved insulin signaling and decreased oxidative stress (via SIRT1 overexpression)^[11].

(GLP-1)^{[8][12]}.

Mechanisms of Antidiabetic Action

Multi-target impacts on glucose homeostasis are seen in all three plants (Figure 1). Numerous investigations on *B. aristata* show that berberine activates AMP-activated protein kinase (AMPK), which suppresses hepatic gluconeogenesis and lipogenesis while increasing glucose absorption and glycolysis. Additionally, berberine increases GLUT4 translocation and insulin signaling (IRS/PI3K/Akt pathway), which improves peripheral insulin sensitivity. It suppresses intestinal enzymes that break down carbohydrates, lowers inflammation (by inhibiting NF- κ B), and may increase β -cell survival by raising incretin

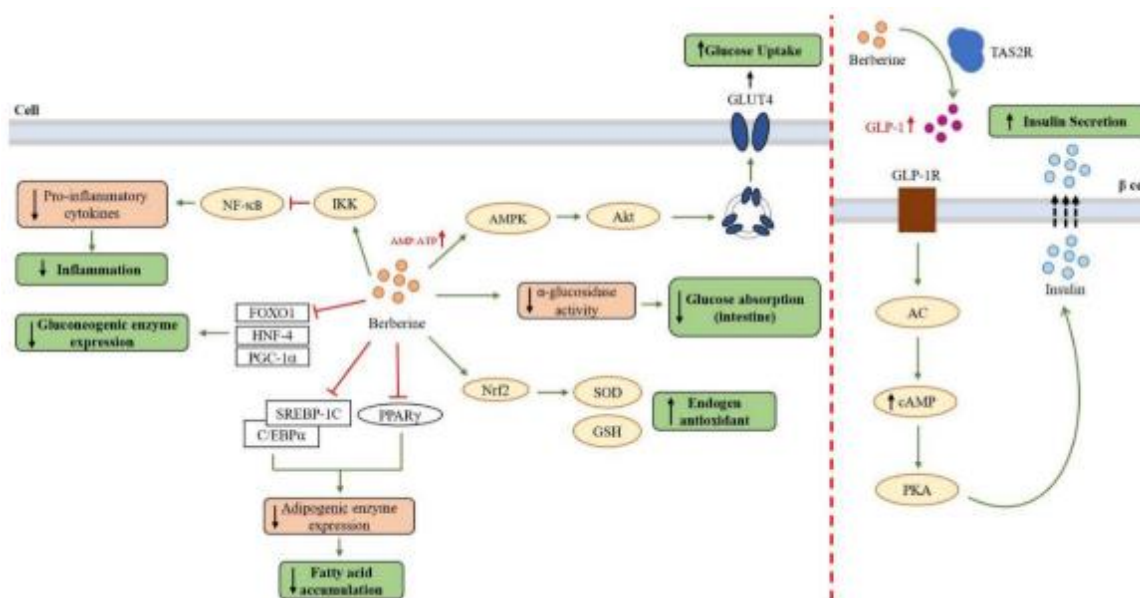


Figure 1: Multitarget antidiabetic mechanisms of berberine derived from *Berberis aristata*.

The components of licorice (*G. glabra*) behave similarly. In vitro, glycyrrhizin and licorice flavonoids have been demonstrated to inhibit α -glucosidase and promote insulin production and GLUT4 translocation. Additionally, glabridin and isoliquiritigenin prevent β -cells from dying and lessen oxidative stress. For instance, glycyrrhizin derivatives stimulate

PI3K/Akt signaling and glucose absorption, whereas glabridin enhances insulin sensitivity in animal models. The flavonoid-rich composition of licorice indicates broad insulintropic and antioxidant effects consistent with antidiabetic action, despite the paucity of direct citations on GLUT4 overexpression in open source^[9].



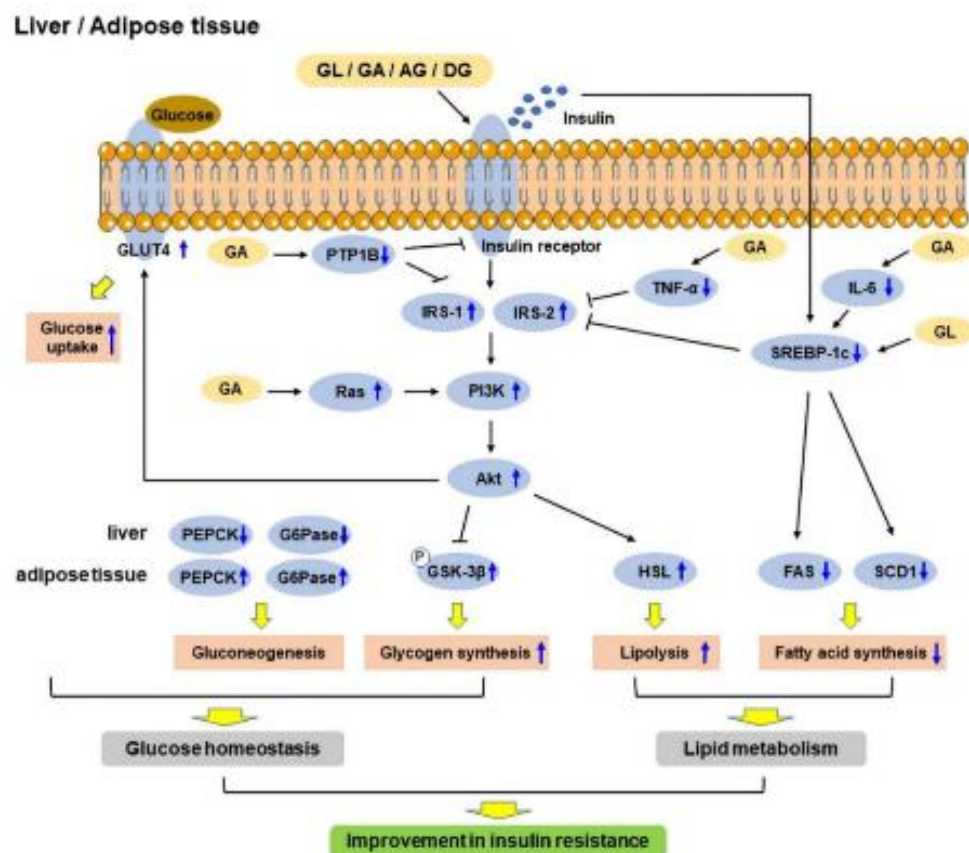
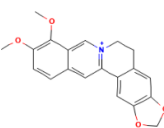
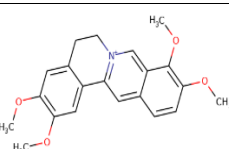
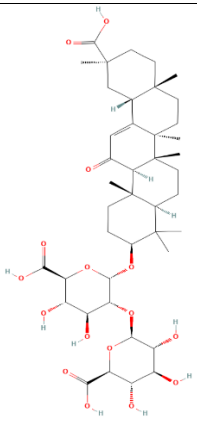
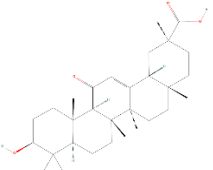
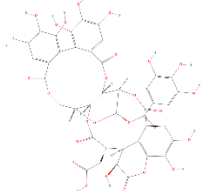
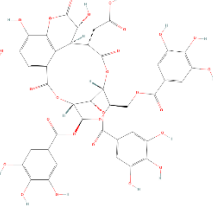


Figure 2. Mechanisms of insulin-sensitizing and glucose-lowering actions of *Glycyrrhiza glabra* (glycyrrhizin and glycyrrhetic acid) in liver and adipose tissue.

Tannins' inhibition of α -glucosidase and α -amylase is the primary mechanism for *T. chebula*. In Caco-2 cell tests, chebulagic acid showed >70% inhibition of maltose digestion, delaying the absorption of carbohydrates. This has the potential to considerably reduce postprandial hyperglycemia. Furthermore, gallic/ellagic acids and tannins have strong antioxidant properties that lessen oxidative stress in the pancreas. According to one study, *T. chebula* extract protected β -cells and

enhanced insulin sensitivity in diabetic rats by upregulating SIRT1 and antioxidant enzymes (SOD, GSH). The bioactive tannins of *Terminalia chebula* function through complimentary methods. It has been demonstrated that chebulagic acid (and similar polyphenols) activates PPAR γ in muscle and adipocytes, increasing GLUT4 expression and enhancing insulin sensitivity [10][11].

Table 1: Major Phytochemicals Identified in *Berberis aristata*, *Glycyrrhiza glabra* and *Terminalia chebula*

Plant	Phytochemical	Structural Representation (SMILES)	Identification Method	Citation
Berberis aristata	Berberine		TLC, HPLC, NMR	[13], [14], [15]
	Palmatine		HPLC, UV, NMR	[14], [16]
Glycyrrhiza glabra	Glycyrrhizin		HPLC, TLC	[17]
	Glycyrrhetinic acid		Acid hydrolysis, HPLC, NMR	[18], [19]
Terminalia chebula	Chebulagic acid		HPLC, NMR	[20], [21]
	Chebulinic acid		Column chromatography, NMR	[20][21]

Comparative Profile

Key characteristics of the three plants, such as botanical family, primary phytochemicals, mechanisms, clinical evidence, dose, and safety, are compiled in Table 2.

Table 2: Comparative botanical, phytochemical, pharmacological, and clinical profile of *Berberis aristata*, *Glycyrrhiza glabra*, and *Terminalia chebula* in diabetes management.

Feature	<i>Berberis aristata</i>	<i>Glycyrrhiza glabra</i>	<i>Terminalia chebula</i>
Family	Berberidaceae	Leguminosae (Fabaceae)	Combretaceae
Plant Part (used)	Root and stem bark (rich in berberine)	Root (licorice extract)	Fruit (haritaki; dried pericarp)
Key Phytochemicals	Berberine, palmatine, berbamine (isoquinoline alkaloids)	Glycyrrhizin (triterpenoid saponin); flavonoids (liquiritin, glabridin, etc.)	Hydrolyzable tannins: chebulinic/chebulagic acid, gallic/ellagic acid
Antidiabetic Mechanisms	↑AMPK activation, ↑GLUT4 translocation, ↑insulin secretion, ↓hepatic gluconeogenesis and lipogenesis; α-glucosidase inhibition, anti-inflammatory effects	↑GLUT4 translocation and insulin secretion; α-glucosidase inhibition; antioxidant/anti-inflammatory actions	Inhibition of α-amylase/α-glucosidase (chebulagic acid); antioxidant/glycation inhibition; β-cell protection (↑SIRT1)
Clinical Evidence	RCTs: Berberine (0.5–1.5 g/day) for 8–12 weeks lowers FBG, HbA1c, triglycerides, LDL	Limited: few trials of licorice extract in T2DM; some benefit on dysglycemia and NAFLD parameters (via anti-inflammatory effects)	Few clinical data on <i>T. chebula</i> alone; triphala formulations (1–3 g/day) modestly reduce fasting glucose and HbA1c in diabetics; animal studies show ↓glucose, ↑insulin
Typical Dose (studied)	500–1500 mg/day berberine-containing extract or tablets	100–500 mg glycyrrhizin per day (in whole extracts); deglycyrrhized licorice to avoid side effects	1–3 g/day <i>T. chebula</i> powder or extract (often in combination products)
Safety	Generally well tolerated; minor GI upset common; avoid in pregnancy/G6PD deficiency	High glycyrrhizin intake causes pseudohyperaldosteronism (hypertension, hypokalemia, edema); use DGL (deglycyrrhized licorice) if long-term	Well tolerated in studies (no acute toxicity at high doses); may potentiate hypoglycemia if combined with other antidiabetics
References in Table	[22],[23]	[24],[25]	[26],[27]

Limitations of Current Evidence

The majority of information regarding the antidiabetic properties of these herbs is preclinical. There are comparatively few rigorous human trials, whereas in vitro and animal studies predominate^[8]. Comparisons are challenging because many research employ crude extracts with varying compositions. Although berberine's limited oral bioavailability is a known problem, its

therapeutic benefits (such as in T2DM) have been shown^[28]. There aren't many studies on licorice specifically for diabetes; those that do exist typically combine licorice with other herbs or are observational. Despite encouraging in vivo findings, *T. chebula* is not well investigated in people. Sample sizes are generally limited, and there is a dearth of long-term safety data. Notably, highlights the need for "further studies, mainly clinical trials involving mono-preparations and



extended pharmacokinetic and toxicological studies." Marchelak et al. (2025)^[8].

FUTURE DIRECTIONS

The goal of future research should be to close existing gaps. To confirm the effectiveness and ideal dosage of each herb (or their essential components) in diabetes patients, carefully planned randomized controlled trials are required. Novel formulations (such as phospholipid complexes and nanoparticles) have been suggested due to berberine's limited bioavailability and should be investigated. It would be beneficial to look at synergy in multi-herb formulations (like triphala, which contains T. chebula). Specific targets, such as PPAR γ activation by chebulic drugs, should be clarified by molecular studies. For long-term use, safety monitoring procedures must be set up. Lastly, results will be more consistent and repeatable if extracts are standardized (berberine, glycyrrhizin, and chebulagic/ellagic acids are quantified).

CONCLUSION

Bioactive substances found in *Terminalia chebula*, *Glycyrrhiza glabra*, and *Berberis aristata* affect several facets of glucose metabolism. Together, they provide antioxidant support, increase glucose uptake (via AMPK/GLUT4), boost insulin secretion/sensitivity, and block enzymes that break down carbohydrates. Comparative investigation reveals complimentary and overlapping pathways (Table 1). While licorice and T. chebula require more research, berberine from B. aristata has the best clinical data to date (with glucose-lowering effects comparable to metformin. Safety profiles vary (T. chebula is generally benign; berberine is contraindicated in pregnancy; licorice requires vigilance about blood pressure). All things considered; these herbs show promise as ethnopharmacological treatments for diabetes. To

convert old knowledge into contemporary therapy, however, superior clinical research and product standardization are crucial next steps.

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