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REVIEW ON ANTIDIABETIC ACTIVITY OF VARIOUS PLANTS

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ABSTRACT

Diabetes mellitus (DM) is a complex and chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The disease encompasses a group of metabolic abnormalities associated with disturbances in carbohydrate, lipid, and protein metabolism, ultimately leading to severe microvascular and macrovascular complications. The global burden of diabetes continues to rise due to increasing prevalence of sedentary lifestyles, obesity, and genetic predisposition, making it a major public health concern.Conventional management of diabetes includes insulin therapy and oral hypoglycemic agents; however, these treatments are often associated with limitations such as adverse effects, high cost, and reduced patient compliance. Consequently, there is growing interest in plant-based therapeutics as safer and cost-effective alternatives. The present review provides a comprehensive overview of the pathophysiology, classification, clinical features, diagnosis, and pharmacological management of diabetes mellitus, along with an in-depth analysis of selected medicinal plants with antidiabetic potential.Plants such as *Azadirachta indica*, *Trigonella foenum-graecum*, *Citrus limon*, *Zingiber officinale*, *Moringa oleifera*, *Punica granatum*, *Syzygium cumini*, *Emblica officinalis*, *Catharanthus roseus*, **and** *Panax quinquefolius* have demonstrated significant antidiabetic activity through diverse mechanisms. These include inhibition of carbohydrate-digesting enzymes, enhancement of insulin secretion and sensitivity, modulation of glucose uptake, antioxidant and anti-inflammatory effects, and protection against diabetic complications.

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent increase in blood glucose levels resulting from defects in insulin secretion, insulin action, or both. Insulin is the key hormone regulating glucose uptake and utilization by body tissues, which is secreted by the β-cells of the pancreatic islets of Langerhans[1]. A deficiency in its secretion from pancreas or an insensitivity of target tissues to its hypoglycemic effects leads to disturbances in metabolism of carbohydrate, lipid, and protein. The common biochemical outcome of all these abnormalities is sustained hyperglycemia [2].

According to both standard pathology texts which highlight that diabetes is not a single disease but a cluster of metabolic disarrangements that share the common feature of hyperglycemia [3]. The consequences of chronic exposure to elevated glucose levelare damage to multiple organs, mainly eyes, kidneys, nerves, and cardiovascular system [4]. The complications likemicrovascular, macrovascular, and neuropathic form are the major causes of morbidity and mortality associated with diabetes. The continuous increase in global diabetes patients is linked to sedentary lifestyle patterns, obesity, and genetic susceptibility, which makes the disease one of the most difficult public health challenges worldwide [5].

TYPES OF DIABETES MELLITUS:

a) Type 1 Diabetes Mellitus (Juvenile Diabetes)

Type 1 DM is primarily an autoimmune disorder leading to the destruction of pancreatic β-cells of the pancreatic islets of Langerhans and consequent insulin deficiency. It often seen in childhood or adolescence and progresses rapidly once the autoimmune destruction crosses a critical threshold. Patients are generally non-obese and prone to episodes of ketoacidosis if untreated. The disease constrains lifelong insulin therapy for survival [6].

b) Type 2 Diabetes Mellitus

Type 2 DM, which represents the vast majority of diabetes cases, arises due to a combination of peripheral insulin resistance and inadequate compensatory insulin secretion. The condition develops insidiously, often in adults, although its incidence is increasing among younger populations due to obesity. Genetic predisposition, physical inactivity, and excess caloric intake play central roles in its onset. While ketosis is rare, chronic hyperglycemia predisposes patients to vascular and neuropathic complications [7]

c) Gestational Diabetes Mellitus

Gestational diabetes occurs when insulin resistance develops during pregnancy due to hormonal influences, leading to hyperglycemia in women without prior diabetes. Although glucose levels usually normalize after delivery, both the mother and offspring remain at increased risk for Type 2 DM later in life.

d) Other Specific Types

Less common variants include diabetes secondary to genetic mutations in β-cell function or insulin receptors, diseases of the exocrine pancreas (such as pancreatitis or neoplasms), endocrinopathies (e.g., Cushing's syndrome, acromegaly), and drug-induced diabetes caused by corticosteroids or thiazide diuretics [8].

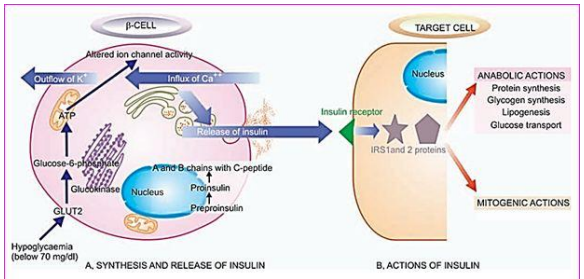

Fig. 1.A. Pathway of normal insulin synthesis and release in ß-cells of pancreatic islet, **B**. Chain of events in action of insulin on target cell

Clinical features of diabetes mellitus:

Type 1 Diabetes Mellitus

The onset of Type 1 DM is typically abrupt. The classic triad of polyuria, polydipsia, and polyphagia is often accompanied by unexplained weight loss despite increased appetite. Fatigue, weakness, and blurred vision

are frequent complaints. In severe insulin deficiency, enhanced lipolysis leads to ketone formation, culminating in diabetic ketoacidosis, a life-threatening condition characterized by acidosis, dehydration, and electrolyte imbalance [9].

i) Patients of type 1 DM usually manifest at early age, generally below the age of 35.
ii) The onset of symptoms is often abrupt.
iii) At presentation, these patients have polyuria, polydipsia and polyphagia.
iv) The patients are not obese but have generally progressive loss of weight.
v) These patients are prone to develop metabolic complications such as ketoacidosis and hypoglycemic episodes.

- **Type 2 Diabetes Mellitus**

Symptoms in Type 2 DM are more subtle and develop gradually. Patients may experience polyuria, thirst, or general tiredness, but many remain asymptomatic and are diagnosed incidentally during routine testing. Obesity and central fat distribution are typical findings. Though ketoacidosis is rare, chronic uncontrolled hyperglycemia may culminate in hyperosmolar non-ketotic coma, particularly in elderly or dehydrated patients. Long-term complications such as retinopathy, nephropathy, and neuropathy commonly dominate the clinical picture.

i) This form of diabetes generally manifests in middle life or beyond, usually above the age of 40.
ii) The onset of symptoms in type 2 DM is slow and insidious.
iii) Generally, the patient is asymptomatic when the diagnosis is made on the basis of glucosuria or hyperglycemia during physical examination, or may present with polyuria and polydipsia.
iv) The patients are frequently obese and have unexplained weakness and loss of weight.
v) Metabolic complications such as ketoacidosis are infrequent [10].

1.2 ETIOPATHOGENESIS OF DIABETESE MELLITUS :

A. Type 1 Diabetes Mellitus

a) Genetic Susceptibility:

Certain genetic factors, particularly within the HLA region on chromosome 6 (HLA-DR3, DR4, and DQ alleles), markedly increase susceptibility to autoimmune β-cell destruction. Familial aggregation and high concordance in identical twins further support a hereditary component.

b) Autoimmune Mechanisms:

The immune system erroneously targets pancreatic β-cell antigens such as insulin, glutamic acid decarboxylase (GAD), and islet cell antigen-2 (IA-2). Activated T-lymphocytes mediate β-cell cytotoxicity, while circulating autoantibodies serve as disease markers. Histologically, pancreatic islets exhibit lymphocytic infiltration termed insulitis followed by progressive β-cell depletion.

c) **Environmental Triggers:**

In genetically predisposed individuals, viral infections such as coxsackievirus B, mumps, or rubella may initiate autoimmune destruction through molecular mimicry. Certain environmental or dietary factors may also influence immune tolerance and disease onset [11].

B. Type 2 Diabetes Mellitus

a) Increased Hepatic Glucose Production:

The liver fails to suppress glucose output despite high circulating insulin, resulting in fasting hyperglycemia. This overproduction of glucose contributes significantly to early metabolic abnormalities.

b) β-cell Dysfunction:

Progressive decline in β-cell performance underlies the disease's chronic nature. Initially, β-cells compensate for insulin resistance by hypersecretion, but over time they exhibit functional exhaustion. Histologically, amyloid deposition and mild atrophy of islets are characteristic features.

c) Insulin Resistance and Obesity:

Central (visceral) obesity plays a pivotal role in insulin resistance. Free fatty acids and adipokines released by adipose tissue disrupt insulin signaling pathways in muscle and liver, reducing glucose uptake and increasing hepatic glucose synthesis [12].

d) Inflammation:

Chronic low-grade inflammation associated with obesity releases pro-inflammatory cytokines—such as TNF-α, IL-6, and C-reactive protein—which interfere with insulin receptor function, perpetuating resistance.

e) Genetic Factors:

Type 2 DM demonstrates strong familial clustering, and multiple susceptibility genes influence both insulin action and secretion. However, lifestyle and environmental factors largely determine disease manifestation.

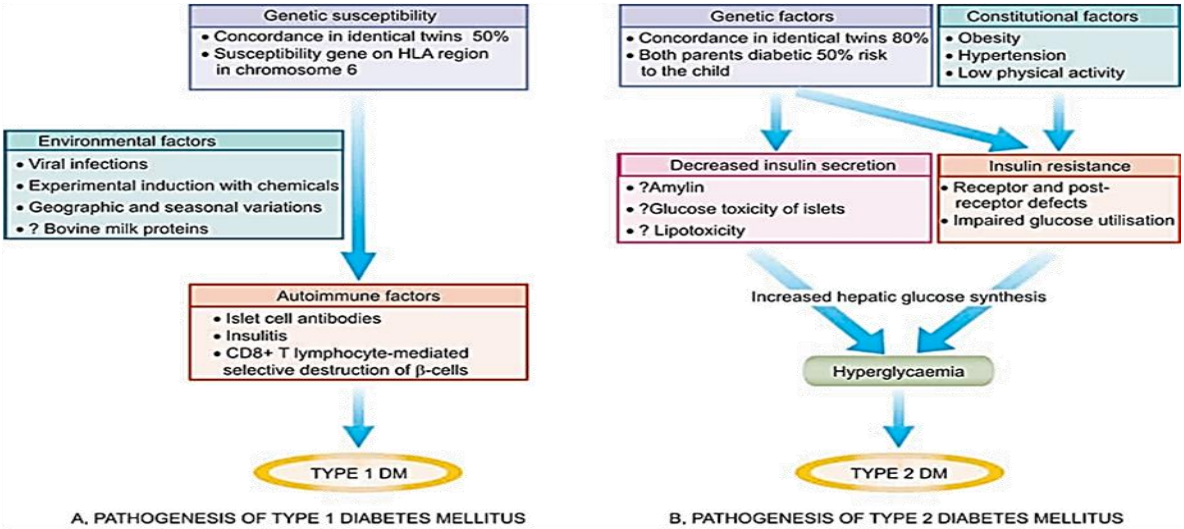

Fig. 2. Schematic mechanisms involved in pathogenesis of two main types of diabetes mellitus.

DIAGNOSIS OF DIABETESE MELLITUS

a) Urine Examination: Detection of glucose or ketone bodies in urine can suggest diabetes but is not diagnostic on its own, as glycosuria may occur in other conditions or physiological states.

b) Random Blood Glucose:A random plasma glucose concentration of ≥200 mg/dL, in the presence of classical symptoms, is highly suggestive of diabetes.

c) Fasting Plasma Glucose:A fasting glucose value of ≥126 mg/dL after an overnight fast confirms the diagnosis.

d) Two-Hour Plasma Glucose (Postprandial):A 2-hour post-meal glucose level of ≥200 mg/dL following a standardized meal or oral glucose load is diagnostic.

e) Oral Glucose Tolerance Test (OGTT):OGTT evaluates the body's response to a fixed glucose challenge and is particularly useful for identifying impaired glucose tolerance and prediabetes states.

f) HbA1c Estimation: Glycosylated hemoglobin (HbA1c) reflects average plasma glucose over the preceding two to three months. A value ≥6.5% is diagnostic of diabetes and is also used to monitor glycemic control.

g) **Other Investigations:**C-peptide levels help distinguish between insulin deficiency and insulin resistance. Autoantibody testing supports diagnosis of autoimmune Type 1 DM. Additional metabolic assessments, including lipid profile and renal function tests, assist in evaluating systemic involvement and disease management [13].

Classification of antidiabetic drugs:

The management of diabetes mellitus involves the use of various pharmacological agents that either enhance insulin action, increase its secretion, or reduce glucose absorption and production. These drugs are broadly classified based on their mechanism of action and therapeutic role in controlling blood glucose levels. Understanding this classification helps in selecting appropriate treatment strategies and exploring plant-derived compounds with similar antidiabetic potential.

Drugs for type 1 diabetes mellitus

Insulin is a key anabolic hormone that promotes the storage and utilization of nutrients like glucose, amino acids, and fatty acids. It enhances glucose uptake in muscle and fat via GLUT4 transporters, while tissues like the brain and liver absorb glucose independently. Insulin stimulates glycogen synthesis by activating glucokinase and glycogen synthase, and suppresses glycogen breakdown. It inhibits gluconeogenesis by downregulating key enzymes, reducing glucose overproduction. In adipose tissue, insulin curbs lipolysis and supports triglyceride formation; its deficiency leads to increased ketone body production. It also boosts lipoprotein lipase activity, aiding lipid clearance. Additionally, insulin facilitates amino acid uptake and protein synthesis while preventing proteolysis. Deficiency results in muscle wasting and negative nitrogen balance. Insulin's effects range from rapid metabolic shifts to longer term gene expression changes and cellular growth regulation [14].

Table 1. Types of insulin preparations and insulin analogues

Type	Onset (Hr)	Peak (Hr)	Duration (Hr)
Rapid acting			
Insulin lispro	0.2-0.3	1-1.5	3-5
Insulin aspart	0.2-0.3	1-1.5	3-5
Insulin glulisine	0.2-0.4	1-2	3-5
Short acting			
Regular (soluble) insulin	0.5-1	2-3	6-8
Intermediate acting			
Insulin zinc suspension or Lente	1-2	3-10	20-24
Neutral protamine Hagedorn (NPH)	1-2	8-10	20-24
Long acting			
Insulin glargine	2-4	-	24
Insulin detemir	1-4	-	20-24

Drugs for type 2 Diabetes mellitus (oral hypoglycaemic)

These drugs lower blood glucose levels and are effective orally. The early sulfonamides tested in 1940s produced hypo glycaemia as side effect. Taking this lead, the first clinically acceptable sulfonylurea tolbutamide was introduced in 1957. In the 1970s many so called 'second generation' sulfonylureas were developed which are 20–100 times more potent. Clinically useful biguanide phenformin was produced parallel to sulfonylureas in 1957. Newer approaches have constantly been explored and have lately yielded thiazolidinediones, meglitinide analogues, α-glucosidase inhibitors and the latest are dipeptidyl peptidase-4 (DPP-4) inhibitors [15].

Table 2. Important features of oral hypoglycaemic

Drug	Plasma $t_{1/2}$ (hr)	Duration of action (hr)	Clearance route	Daily dose	No. of doses per day
SULFONYLUREAS					
Tolbutamide	6	6-8	L	0.5-3g	2-3
Glibenclamide	2-4	24	L	2.5-15mg	1-2
Glipizide	3-5	12	L	5-20mg	1-2
Gliclazide	8-20	12-24	L	40-240mg	1-2
Glimepiride	5-7	24	L	1-6mg	1-2
MEGLITINIDE / PHENYLALANINE ANALOGUES					
Repaglinide	<1	3-5	L	1-8mg	3-4
Nateglinide	1.5	2-4	L	180-480mg	3-4
DPP-4 INHIBITORS					
Sitagliptin	~12	24	K	100mg	1
Vildagliptin	2-4	12-24	L,K	50-100mg	1-2
BIGUANIDE					
Metformin	1.5-3	6-8	K	0.5-2.5g	1-2
THIAZOLIDINEDIONE					
Pioglitazone	3-5	24	L	15-45 mg	1

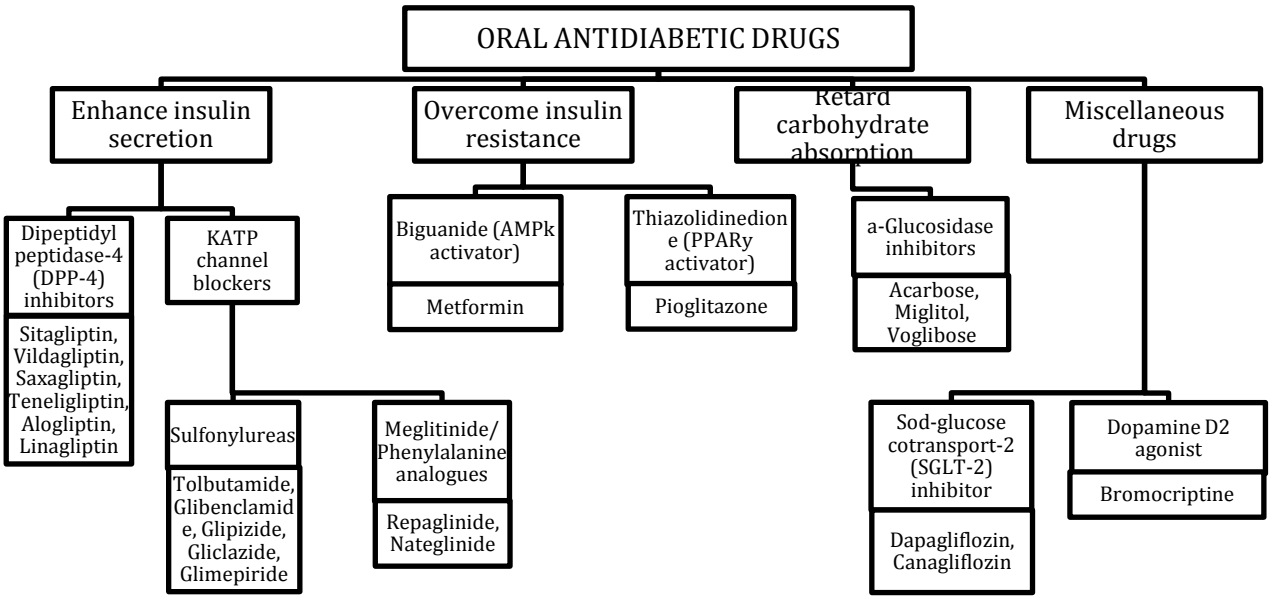

Fig. 3. Classification of Oral Antidiabetic Drugs

Antidiabetic Role and Mechanistic Insights of Selected Medicinal Plants

1. *Azadirachta indica*

Azadirachta indica demonstrates significant antidiabetic activity through multifactorial mechanisms targeting glucose homeostasis and pancreatic protection. Preclinical studies reveal its ability to reduce fasting blood glucose levels and improve glucose tolerance, particularly in streptozotocin (STZ)-induced diabetic models. Mechanistically, neem-derived limonoids such as nimbolide and azadirachtin, along with

flavonoids like quercetin, contribute to inhibition of carbohydrate-digesting enzymes (α-glucosidase and α-amylase), thereby attenuating postprandial hyperglycemia.

Additionally, neem exhibits pancreatic β-cell protective effects, attributed to its potent antioxidant activity that reduces ROS-mediated cellular damage. Studies also suggest enhanced insulin secretion and improved peripheral glucose utilization, possibly through modulation of insulin signaling pathways. Furthermore, its anti-inflammatory properties may help in mitigating chronic inflammation associated with insulin resistance [16].

2. *Trigonella foenum-graecum*

Trigonella foenum-graecum is one of the most extensively validated antidiabetic plants, supported by robust preclinical and clinical evidence. The amino acid derivative 4-hydroxyisoleucine plays a crucial role in stimulating glucose-dependent insulin secretion, thereby improving glycemic control. Additionally, the high content of soluble fiber (galactomannan) significantly delays gastric emptying and glucose absorption, leading to reduced postprandial glucose spikes.

Fenugreek also enhances insulin sensitivity in peripheral tissues, possibly through modulation of insulin receptor signaling and increased glucose uptake. Clinical trials have reported significant reductions in fasting blood glucose, postprandial glucose, and HbA1c levels, supporting its role as an effective adjunct therapy. Moreover, bioactive compounds such as trigonelline and diosgenin contribute to lipid-lowering and antioxidant effects, addressing metabolic syndrome components associated with diabetes [17].

3. *Citrus limon*

The antidiabetic effects of *Citrus limon* are primarily attributed to its rich flavonoid content, including hesperidin, eriocitrin, and naringin. These compounds exert glucose-modulatory effects by enhancing insulin signaling pathways, particularly through activation of key metabolic regulators involved in glucose uptake and utilization.

In vitro and in vivo studies demonstrate inhibition of α-amylase and α-glucosidase enzymes, reducing carbohydrate digestion and glucose absorption. Additionally, citrus flavonoids promote GLUT-mediated glucose uptake in skeletal muscle and adipose tissues, contributing to improved glycemic control. The strong antioxidant and anti-inflammatory properties further help in reducing oxidative stress and preventing diabetic complications, including endothelial dysfunction and insulin resistance [18].

4. *Zingiber officinale*

Zingiber officinale exerts its antidiabetic effects through multiple complementary mechanisms involving glucose metabolism and insulin sensitivity. Active constituents such as gingerols and shogaols enhance GLUT4 translocation in muscle and adipose tissues, thereby facilitating increased glucose uptake.

Ginger also inhibits key carbohydrate-metabolizing enzymes (α-glucosidase and β-glucosidase), reducing intestinal glucose absorption. Its potent antioxidant activity mitigates oxidative stress, a critical factor in β-cell dysfunction and insulin resistance. Additionally, anti-inflammatory effects mediated through downregulation of pro-inflammatory cytokines contribute to improved metabolic outcomes. Experimental and clinical studies indicate significant reductions in fasting blood glucose and improvement in lipid profiles, highlighting its potential in managing diabetes and associated dyslipidemia [19].

5. *Moringa oleifera*

Moringa oleifera has emerged as a promising antidiabetic agent due to its diverse bioactive compounds, including isothiocyanates, flavonoids, and phenolic acids. These constituents contribute to enhanced insulin sensitivity and improved glucose utilization.

Mechanistically, moringa inhibits intestinal glucose absorption through suppression of carbohydrate-digesting enzymes, while also modulating hepatic gluconeogenesis. Its strong antioxidant activity protects pancreatic β-cells from oxidative damage and improves insulin secretion.

Animal studies and preliminary human trials have demonstrated significant reductions in fasting blood glucose levels and improved glucose tolerance. Additionally, its anti-inflammatory effects help in alleviating chronic low-grade inflammation associated with insulin resistance and type 2 diabetes [20].

6. *Punica granatum*

Punica granatum exhibits antidiabetic activity primarily through its rich content of ellagitannins (punicalagin, punicalin), ellagic acid, and anthocyanins. These compounds exert glucose-lowering effects by improving insulin sensitivity and reducing oxidative stress.

Experimental studies demonstrate that pomegranate extracts can modulate carbohydrate metabolism enzymes and enhance glucose uptake, leading to improved glycemic control. Furthermore, its antioxidant properties reduce lipid peroxidation and inflammatory responses, which are critical in the progression of diabetes and its complications.

Although clinical evidence remains limited and somewhat variable, available studies suggest potential benefits in reducing fasting glucose levels and improving cardiovascular risk markers, including lipid profiles and endothelial function [21].

7. *Syzygium cumini*

Syzygium cumini seeds are widely recognized for their antidiabetic efficacy, supported by both traditional use and modern scientific studies. The plant exhibits hypoglycemic activity through inhibition of α-amylase and β-glucosidase enzymes, thereby reducing carbohydrate digestion and glucose absorption. Additionally, its polyphenolic compounds provide antioxidant and antiglycation effects, which help prevent the formation of advanced glycation end-products (AGEs), a key factor in diabetic complications. Studies also suggest improvement in insulin sensitivity and lipid metabolism, contributing to overall metabolic regulation.Clinical and preclinical studies report reductions in fasting and postprandial glucose levels, supporting its therapeutic relevance in diabetes management [22].

8. *Emblica officinalis*

Emblica officinalis exerts potent antidiabetic effects due to its high content of hydrolyzable tannins (emblicanin A and B), gallic acid, and vitamin C. These compounds provide strong antioxidant activity, which plays a crucial role in protecting pancreatic β-cells and improving insulin secretion.

Amla also demonstrates inhibition of carbohydrate-digesting enzymes and reduction in oxidative stress-induced damage, leading to improved glycemic control. Additionally, it reduces the formation of AGEs and enhances lipid metabolism, thereby preventing diabetic complications such as neuropathy and cardiovascular disorders.

Both animal studies and clinical data indicate significant reductions in blood glucose levels andimprovement in lipid profiles, highlighting its role as a multifunctional antidiabetic agent [23].

9. *Catharanthus roseus*

Catharanthus roseus has shown promising antidiabetic activity in preclinical studies, primarily through enhancement of insulin secretion and increased peripheral glucose uptake. Its bioactive alkaloids and phenolic compounds contribute to improved glucose metabolism and insulin signaling.However, the presence of potent cytotoxic alkaloids such as vincristine and vinblastine limits its therapeutic application in diabetes due to potential toxicity. While experimental studies demonstrate

significant hypoglycemic effects in diabetic models, its clinical use as an antidiabetic agent remains limited and requires further safety evaluation [24].

10. *Panax quinquefolius*

Panax quinquefolius (American ginseng) has been extensively studied for its role in postprandial glucose regulation. Ginsenosides, the primary active constituents, enhance glucose uptake in peripheral tissues and improve insulin sensitivity.Clinical studies indicate that administration of American ginseng prior to carbohydrate intake results in significant reductions in postprandial blood glucose levels. The mechanisms involve modulation of glucose transporters, improvement in insulin signaling, and possible effects on gastric emptying [25].

Fig.4.Medicinal plants used in DM

Although evidence supports its efficacy in controlling postprandial hyperglycemia, its impact on long-term glycemic markers such as HbA1c remains variable and dependent on dosage, formulation, and duration of treatment [26].

CONCLUSION:

Diabetes mellitus is a multifactorial metabolic disorder with complex pathophysiology involving impaired insulin secretion, insulin resistance, and dysregulated glucose metabolism. Despite significant advances in pharmacotherapy, the management of diabetes remains challenging due to associated side effects, economic burden, and the progressive nature of the disease.Medicinal plants have gained considerable attention as potential therapeutic agents owing to their multitargeted mechanisms of action. The plants reviewed in this study exhibit significant antidiabetic effects

through diverse pathways, including inhibition of carbohydrate-digesting enzymes, enhancement of insulin secretion, improvement in insulin sensitivity, modulation of glucose uptake, and reduction of oxidative stress and inflammation. Additionally, several plants demonstrate protective effects against diabetic complications such as neuropathy, nephropathy, and cardiovascular disorders.Among the reviewed plants, *Trigonella foenum-graecum, Syzygium cumini,* and *Panax quinquefolius* show relatively stronger clinical evidence, while others such as *Azadirachta indica, Moringa oleifera,* and *Emblica officinalis* exhibit promising preclinical and emerging clinical data. However, variability in phytochemical composition, dosage, and study design limits direct clinical translation.Therefore, future research should focus on standardization of plant extracts, identification of active constituents, elucidation of molecular mechanisms, and large-scale clinical trials to validate their efficacy and safety. Integration of plant-based therapies with conventional treatment may provide a more comprehensive and effective approach for diabetes management.

REFERENCES

1. Kumar V, Abbas AK, Aster JC. Robbins and Cotran Pathologic Basis of Disease. 10th ed. Philadelphia: Elsevier; 2021.
2. Hall JE. Guyton and Hall Textbook of Medical Physiology. 14th ed. Philadelphia: Elsevier; 2021.
3. American Diabetes Association. Classification and diagnosis of diabetes: Standards of Medical Care in Diabetes—2024. *Diabetes Care*. 2024;47(Suppl 1):S16–S33.
4. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels: IDF; 2021.
5. DeFronzo RA, Ferrannini E, Groop L, et al. Type 2 diabetes mellitus. *Nat Rev Dis Primers*. 2015;1:15019.
6. Atkinson MA, Eisenbarth GS, Michels AW. Type 1 diabetes. *Lancet*. 2014;383(9911):69–82.
7. Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. *Nat Rev Immunol*. 2011;11(2):98–107.
8. Kahn SE, Cooper ME, Del Prato S. Pathophysiology and treatment of type 2 diabetes. *Lancet*. 2014;383(9922):1068–1083.
9. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2010;33(Suppl 1):S62–S69.
10. World Health Organization. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia. Geneva: WHO; 2006.
11. Brunton LL, Hilal-Dandan R, Knollmann BC. Goodman & Gilman's The Pharmacological Basis of Therapeutics. 13th ed. New York: McGraw Hill; 2018.
12. Katzung BG. Basic and Clinical Pharmacology. 15th ed. New York: McGraw Hill; 2021.
13. Rang HP, Ritter JM, Flower RJ, Henderson G. Rang & Dale's Pharmacology. 9th ed. Elsevier; 2020.
14. Modak M, Dixit P, Londhe J, Ghaskadbi S, Devasagayam TP. Indian herbs and herbal drugs used for the treatment of diabetes. *J Clin Biochem Nutr*. 2007;40(3):163–173.
15. Patel DK, Kumar R, Laloo D, Hemalatha S. Diabetes mellitus: An overview on its pharmacological aspects and reported medicinal plants having antidiabetic activity. *Asian Pac J Trop Biomed*. 2012;2(5):411–420.
16. Subapriya R, Nagini S. Medicinal properties of neem leaves: A review. *Curr Med Chem Anticancer Agents*. 2005;5(2):149–156.
17. Basch E, Ulbricht C, Kuo G, Szapary P, Smith M. Therapeutic applications of fenugreek. *Altern Med Rev*. 2003;8(1):20–27.

18. Mulvihill EE, Huff MW. Citrus flavonoids and metabolic health. *J Nutr Biochem*. 2012;23(7):849–859.
19. Mahluji S, Attari VE, Mobasseri M, et al. Effects of ginger on glycemic status. *Int J Food Sci Nutr*. 2013;64(6):682–686.
20. Leone A, Spada A, Battezzati A, et al. Moringa oleifera seeds and oil: Characteristics and uses. *Int J Mol Sci*. 2016;17(12):2141.
21. Jurenka JS. Therapeutic applications of pomegranate. *Altern Med Rev*. 2008;13(2):128–144.
22. Ayyanar M, Subash-Babu P. Syzygium cumini and its antidiabetic properties: A review. *Asian Pac J Trop Biomed*. 2012;2(3):240–246.
23. Akhtar MS, Ramzan A, Ali A, Ahmad M. Effect of Amla fruit on blood glucose. *Int J Food Sci Nutr*. 2011;62(6):609–616.
24. Chattopadhyay RR. A comparative evaluation of some blood sugar lowering agents. *J Ethnopharmacol*. 1999;67(3):367–372.
25. Vuksan V, Sievenpiper JL. American ginseng and glycemic control. *J Am Coll Nutr*. 2005;24(6):389–395.
26. Desai SA, Khulbe P, Dange YD, Kadam S, Jaiswal M. Antioxidants-rich food sources of nutraceutical and functional foods. InAntioxidants as Nutraceuticals 2025 Jan 14 (pp. 265-281). Apple Academic Press.

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