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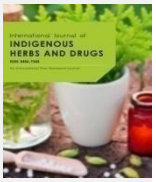

A REVIEW ON: ANTI-HYPERGLYCEMIC ACTIVITY OF POTENTIAL HERBAL PLANTS

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Abstract

Diabetes mellitus is a chronic, multifactorial metabolic disorder that has emerged as one of the most formidable public health challenges of the 21st century. Current therapeutic regimens, though effective in the short term, are often constrained by adverse effects, high cost, and diminished efficacy over prolonged use, highlighting the urgent need for complementary or alternative approaches. Traditional systems of medicine, including Ayurveda, Unani, and Traditional Chinese Medicine, have long emphasized the role of herbal plants in managing hyperglycemia. These natural agents are rich in diverse bioactive compounds-such as alkaloids, flavonoids, terpenoids, saponins, and glycosides that act synergistically through multiple mechanisms. They can stimulate insulin secretion, enhance pancreatic β-cell regeneration, improve peripheral glucose uptake, inhibit carbohydrate-digesting enzymes, modulate hepatic metabolism, and protect against oxidative stress-induced complications.

Keywords: Diabetes mellitus, Ayurveda, Unani, bioactive compounds, alkaloids, flavonoids, terpenoids, saponins.

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Introduction

Diabetes mellitus (DM) is among the most prevalent metabolic disorders of the 21st century, affecting millions worldwide and placing a substantial burden on healthcare systems. It is characterized by persistent hyperglycemia resulting from either impaired insulin secretion, insulin resistance, or both. Prolonged elevation of blood glucose levels contributes to complications in the cardiovascular, renal, neural, and ocular systems, making diabetes not only a metabolic disorder but also a leading cause of disability and premature mortality. According to the International Diabetes Federation (IDF), the number of people living with diabetes is expected to rise from 537 million in 2021 to nearly 643 million by 2030, underscoring the urgent need for effective and sustainable therapeutic strategies. Although several conventional therapies such as sulfonylureas, biguanides, thiazolidinediones, DPP-4 inhibitors, and insulin analogues are widely used, their long-term application is often associated with side effects, high costs, and reduced efficacy [1]. Hypoglycemia, gastrointestinal disturbances, weight gain, and cardiovascular risks are common adverse outcomes, while lifelong dependence on synthetic drugs remains a challenge for many patients. Consequently, the exploration of safer, cost-effective, and holistic treatment alternatives has become a priority in diabetes research. Herbal plants, with their deep-rooted history in traditional medical systems such as Ayurveda, Traditional Chinese Medicine (TCM), and Unani medicine, have emerged as promising therapeutic options. Historical texts described conditions resembling diabetes-referred to as “Madhumeha” in Ayurveda and “Xiao Ke” in TCM-and recommended plant-based formulations to alleviate excessive thirst, frequent urination, and weight loss. Contemporary pharmacological studies have validated these practices, showing that plants contain diverse phytochemicals including alkaloids, flavonoids, terpenoids, saponins, and glycosides with significant antihyperglycemic potential. These bioactive compounds exert their effects

through multiple mechanisms such as stimulating insulin secretion, regenerating pancreatic β-cells, enhancing glucose uptake, inhibiting carbohydrate-digesting enzymes, modulating lipid metabolism, and protecting against oxidative stress. Diabetes mellitus is a non-infectious endocrine disorder that occurs when the body is unable to produce sufficient insulin, as in type 1 diabetes, or fails to use insulin effectively, as in type 2 diabetes. This results in disturbances in the metabolism of carbohydrates, fats, and proteins, ultimately leading to hyperglycemia [2]. It is a chronic and widespread condition that affects individuals across both developed and developing nations. If left untreated, diabetes is associated with severe microvascular complications such as neuropathy, retinopathy, and nephropathy, as well as macrovascular complications including peripheral vascular disease and coronary heart disease. The increasing recognition of diabetes as a global health crisis has led to its frequent comparison with other major non-communicable diseases such as cancer and cardiovascular disorders. Thus, a comprehensive understanding of diabetes mellitus is essential for its effective prevention and management.

Epidemiology

The term "diabetes" is derived from the Greek word diab, meaning "to pass through" or "a siphon," reflecting the characteristic excessive urination, while "mellitus" originates from the Latin word for "honey-sweet," referring to the sugar-laden urine associated with the disease. Although recognized for thousands of years, the modern understanding of its pathophysiology and management has advanced considerably [3].

Globally, the prevalence of diabetes has risen at an alarming rate. In 2003, the World Health Organization (WHO) projected that by 2030, the number of adults living with diabetes would nearly double, rising from 177 million in 2000 to 370 million. Experts have further estimated that by 2025, the incidence may increase by as much as 64%, affecting approximately 53.1 million individuals. In 2010, the worldwide prevalence of diabetes among adults was around 285 million (6.4%), and this number is expected to reach 439 million (7.7%) by 2030.

Diabetes mellitus, often referred to simply as diabetes, is a group of metabolic diseases characterized by elevated blood glucose levels resulting either from inadequate insulin production, improper cellular response to insulin, or a combination of both. Antihyperglycemic drugs are specifically developed to regulate blood glucose levels and prevent the progression of complications. In 2013, it was estimated that over 382 million people worldwide were living with diabetes, emphasizing its status as a critical global health challenge.

Historical Background

The use of herbal plants in the management of diabetes dates back thousands of years. Long before the discovery of insulin in the 20th century, ancient civilizations recognized the symptoms of excessive urination, thirst, and weight loss, and sought remedies in nature. In Ayurveda (India, around 5000 years ago), diabetes was described as Madhumeha ("honey urine), with detailed classifications of its causes and symptoms. Classical texts such as Charaka Samhita and Sushruta Samhita recommended plant-based treatments including Gymnemasylvestre (Gurmar), Momordica charantia (Bitter melon), Trigonella foenum-graecum (Fenugreek), and Ocimum sanctum (Tulsi). These plants were traditionally used to control sugar levels, improve digestion, and restore metabolic balance.In Traditional Chinese Medicine (TCM), diabetes-like symptoms were referred to as Xiao Ke ("wasting and thirsting syndrome"). Herbal preparations using plants such as Panax ginseng, Astragalus membranaceus, and Coptis chinensis (Huang Lian) were prescribed to regulate qi, nourish yin, and reduce thirst and excessive urination. In the Unani system of medicine* (Arab–Persian tradition), diabetes, known as Ziabetus Shakri, was managed with herbs like Syzygiumcumini (Jamun), Cinnamomum verum (Cinnamon), and Aloe vera. These remedies were believed to correct imbalances of the humors and improve overall metabolism [4]. In medieval Europe, physicians of the Greco-Roman and medieval periods often relied on bitter herbs and dietary modifications. Preparations from *Galega officinalis (Goat's rue) were commonly used, and this plant later inspired the development of metformin, one of the most important modern oral antidiabetic drugs.

Types of Diabetes Mellitus

- Type 1 (Insulin Dependent)
- Type 2 (Non- Insulin Dependent)
- Gestational Diabetes

Type 1 (Juvenile Diabetes Mellitus)

Type 1 diabetes is characterized by the autoimmune destruction of insulin-producing beta cells in the pancreas, resulting in little or no insulin secretion, which is essential for regulating blood glucose levels. Often referred to as "Juvenile Diabetes Mellitus," this form of diabetes is considered hereditary and accounts for about 5–10% of all diabetes cases globally. According to the American Diabetes Association (2001), approximately 20 million people worldwide were affected by type 1 diabetes at that time. This condition is most commonly diagnosed in children and young adults, often with a sudden onset of symptoms that can be severe and potentially life-threatening. Individuals with type 1 diabetes also have a higher risk of developing other autoimmune disorders due to the genetic and immunological factors underlying autoimmunity. Careful and continuous management is therefore required to prevent serious complications such as cardiovascular disease, kidney disease, retinopathy, neuropathy, foot problems, and stroke, especially if blood glucose levels are not properly controlled [5].

Sub-Types

Type 1a (Autoimmune) develops as a result of an autoimmune response and is often associated with other autoimmune disorders such as Addison's disease, Graves' disease, and Hashimoto's thyroiditis.

Type 1b (Idiopathic) accounts for approximately 10% of type 1 cases. In this form, patients may experience significant insulin deficiency and are at risk for ketoacidosis.

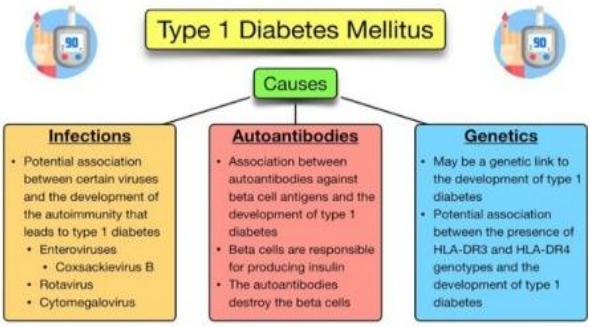

Fig 01: Causes of Type-1 Diabetes Mellitus.

Type 2 (Adult Type)

Type 2 diabetes is a multifactorial metabolic disorder that arises from a combination of genetic, environmental, and lifestyle factors which disrupt metabolic pathways, particularly those involved in glucose regulation. It is most common in elderly individuals and is characterized by both impaired insulin secretion and insulin resistance. Insulin resistance, a key feature of the disease, is influenced by several factors including oxidative stress, down regulation of insulin receptors, and a reduction in the number of insulin receptors. According to the World Health Organization (WHO), type 2 diabetes is a major global health concern, currently affecting over 422 million people worldwide and contributing to approximately 1.6 million deaths each year. In this condition, the body's cells become resistant to the action of insulin, making it difficult to regulate blood glucose levels effectively. As a result, individuals with type 2 diabetes are at risk of developing serious complications such as kidney disease, eye disorders, nerve damage, and cardiovascular problems [6]. Multiple risk factors contribute to the development of type 2 diabetes. While many are related to lifestyle choices such as poor diet, physical inactivity, and obesity, others are linked to genetic predisposition and mental health conditions.

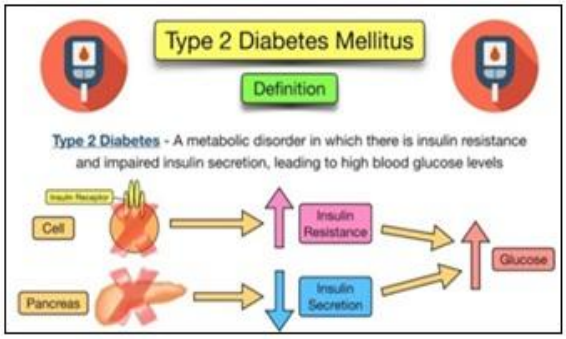

Fig 02: Type-2 Diabetes Mellitus.

Gestational Diabetes

Gestational diabetes mellitus is a form of glucose intolerance that is first diagnosed during pregnancy, typically developing in the second or third trimester. During this stage, the body produces higher levels of certain hormones, which can interfere with insulin function and lead to insulin resistance. When the pancreas is unable to produce sufficient insulin to compensate for this resistance gestational diabetesoccurs.

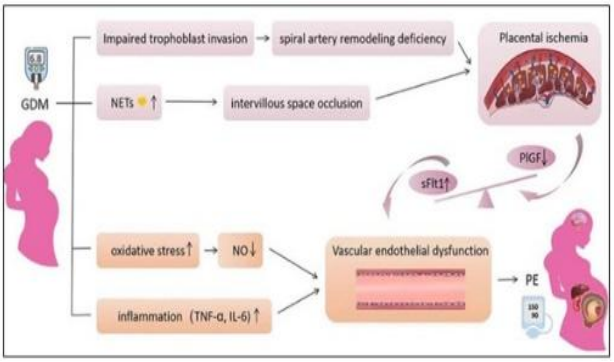

Fig 03: Gestational Diabetes Mellitus

Pathophysiology

1. Pathophysiology of Type 1 Diabetes Mellitus

The development of type 1 diabetes mellitus results from a combination of genetic predisposition and environmental triggers. These factors lead to the formation of autoantigens on insulin-producing pancreatic beta cells, which then enter the bloodstream and lymphatic system. The autoantigens are processed and presented by antigen-presenting cells (APCs), initiating an immune response [7].

During this process, T helper 1 (Th1) lymphocytes become activated and release interferon-gamma (IFN-γ). This, in turn, stimulates macrophages to secrete interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α). Th1 cells also promote the activation of autoantigen-specific cytotoxic T (CD8+) cells by releasing interleukin-2 (IL-2). Simultaneously, T helper 2 (Th2) lymphocytes are activated and release interleukin-4 (IL-4), which stimulates B lymphocytes to produce islet cell autoantibodies and anti-GAD65 antibodies.

The combined immune assault, involving macrophages, cytotoxic T cells, and autoantibodies, ultimately destroys pancreatic beta cells. This progressive destruction results in reduced insulin secretion and the onset of type 1 diabetes mellitus [8].

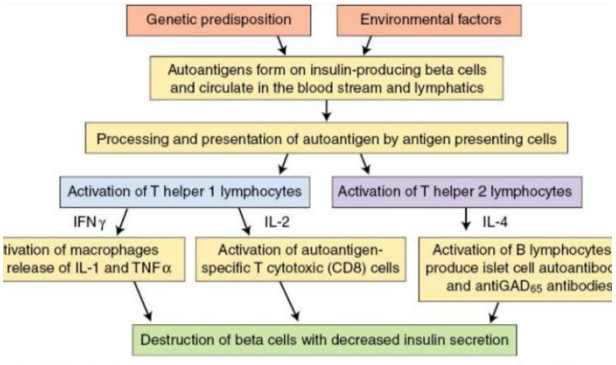

Fig 04: Pathophysiology of Type 1 Diabetes Mellitus

2. Pathophysiology of Type 2 Diabetes Mellitus

The process begins with impaired insulin secretion, where the pancreas gradually loses its ability to release sufficient insulin. This impairment may result from a combination of genetic predisposition and lifestyle-related factors. As insulin secretion declines, the liver responds by increasing glucose production, which leads to elevated blood glucose levels (hyperglycemia). Normally, insulin suppresses hepatic glucose output, but when insulin is deficient or ineffective, glucose release becomes unregulated. Persistent hyperglycemia and prolonged exposure to insulin resistance

then cause defects in insulin receptors and the signaling pathways that follow insulin binding, known as post-receptor defects. These abnormalities reduce the responsiveness of cells to insulin. Consequently, peripheral tissues such as skeletal muscle and adipose tissue, which are the major sites for glucose uptake, become increasingly resistant to insulin's action. This diminished glucose uptake further worsens hyperglycemia. Ultimately, the interplay of impaired insulin secretion, excessive hepatic glucose production, and insulin resistance in peripheral tissues leads to the onset of Type 2 Diabetes Mellitus, a chronic condition characterized by persistently high blood sugar levels [9].

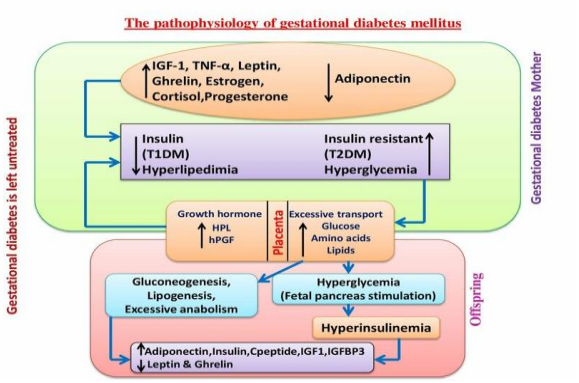

Fig 05: Pathophysiology of Type 2 Diabetes Mellitus

1. Pathophysiology of Gestational Diabetes Mellitus

The process begins in the mother, especially when GDM is left untreated. On the maternal side, hormonal changes play a central role. Levels of insulin-like growth factor I (IGF-I), tumor necrosis factor-alpha (TNF-α), leptin, ghrelin, estrogen, cortisol, and progesterone are elevated, while adiponectin levels are reduced. These alterations drive two key metabolic pathways: a decrease in insulin secretion, which mimics a type 1 diabetes–like effect and leads to hyperlipidemia, and the development of insulin resistance, which resembles a type 2 diabetes–like effect and causes hyperglycemia. Both pathways collectively contribute to a metabolic environment that affects the placenta.The placenta functions as a mediator, further influencing maternal metabolism while also transferring nutrients and hormones to the fetus. It secretes growth hormone (GH), human placental lactogen (HPL), and human placental growth factor (HPGF), all of which exacerbate maternal insulin resistance. At the same time, the placenta facilitates excessive transport of glucose, amino acids, and lipids from the mother's circulation into the fetus.As a result, the fetus experiences hyperglycemia, which stimulates the fetal pancreas to produce excessive insulin, leading to fetal hyperinsulinemia [10]. This condition drives enhanced anabolic processes, including gluconeogenesis and lipogenesis, causing abnormal metabolic activity in the fetus. Fetal hyperinsulinemia is also associated with altered levels of regulatory substances, including increased insulin, C-peptide, IGF-1, and IGFBP-3, along with decreased adiponectin, leptin, and ghrelin.

Major Mechanisms of Antihyperglycemic Activity

1. **Enhancing Insulin Secretion (Insulin Secretagogues) Mechanism**
   Stimulate pancreatic β-cells to increase insulin release.
   Drug Examples:Sulfonylureas (e.g., glipizide, glyburide), Meglitinides (e.g., repaglinide) Target:Pancreatic β-cell ATP-sensitive K^+ channels.
2. **Increasing Insulin Sensitivity**
   Mechanism:Improve the ability of tissues (muscle, fat, liver) to respond to insulin. Drug examples:Biguanides (e.g., metformin)↓ hepatic gluconeogenesis, ↑ peripheral glucose uptake, Thiazolidinediones (TZDs) (e.g., pioglitazone), Activate PPAR-γ to enhance insulin sensitivity
3. **Delaying Carbohydrate Absorption**
   Mechanism:Inhibit enzymes that digest carbohydrates in the gut, leading to slower glucose absorption [11].
   Drug examples:α-glucosidase inhibitors (e.g., acarbose, miglitol) Target: Enzymes in the intestinal brush border (maltase, sucrase, etc.)
4. **Increasing Renal Glucose Excretion**
   Mechanism:Inhibit glucose reabsorption in the kidney, promoting glucosuria.
   Drug examples: SGLT2 inhibitors (e.g., canagliflozin, dapagliflozin), Target:Sodium-glucose co-transporter 2 in the proximal tubule
5. **Enhancing Incretin Effect**
   Mechanism: Mimic or boost GLP-1 (glucagon-like peptide-1), a gut hormone that: ↑ insulin secretion (glucose-dependent) ,↓ glucagon secretion, Slows gastric emptying Promotes satiety
   Drug examples: GLP-1 receptor agonists (e.g., liraglutide, exenatide), DPP-4 inhibitors (e.g., sitagliptin, saxagliptin), Prevent breakdown of GLP-1
6. **Direct Insulin Supplementation**
   Mechanism: Replaces or supplements insulin to promote: Glucose uptake by tissues (especially muscle and fat) Inhibition of hepatic glucose production. Drug examples: Insulin analogs (e.g., insulin glargine, insulin lispro)
7. **Suppressing Hepatic Gluconeogenesis**
   Mechanism: Directly inhibit the liver's glucose production. Drug example: MetforminInhibits mitochondrial respiratory chain (complex I), Activates AMPK → ↓ gluconeogenesis
8. **Reducing Glucagon Secretion**
   Mechanism: Lower glucagon levels to prevent hepatic glucose release.
   Drug examples: GLP-1 receptor agonists, DPP-4 inhibitors [12].

Table 01: Main Risk Factors of Antihyperglycemic Therapy

Mechanism of Drug	Drug Class	Potential Risks / Side Effects
↑ Insulin secretion	Sulfonylureas, Meglitinides	Hypoglycemia, weight gain
Insulin replacement	Insulin	Hypoglycemia, weight gain
↓ Hepatic glucose output	Metformin	Lactic acidosis (rare), GI upset, B12 deficiency
↑ Insulin sensitivity	Thiazolidinediones (TZDs)	Fluid retention, heart failure, weight gain, bone fracture

↓ Carbohydrate absorption	α-Glucosidase inhibitors	Bloating, flatulence, diarrhea
↑ Renal glucose excretion	SGLT2 inhibitors	UTIs, genital infections, dehydration, ketoacidosis
↑ Incretin effect	GLP-1 receptor agonists	Nausea, vomiting, pancreatitis, weight loss
↑ Incretin effect	DPP-4 inhibitors	Rare: pancreatitis, joint pain [13]

Prevention Tips

- Don't skip meals
- Take meds as prescribed
- Check blood sugar regularly
- Know the signs of low sugar
- Carry sugar or candy just in case

Potential of herbal plants for antihyperglycemic activity

Gymnemasylvestre: Gymnemasylvestre, commonly known as Gurmar (meaning "sugar destroyer" in Hindi), cowplant, or Australian cow plant, is a medicinal herb native to India, Africa, and parts of Australia. It belongs to the family Apocynaceae (formerly Asclepiadaceae), and its leaves are widely used in traditional Ayurvedic medicine for therapeutic purposes. The plant contains several bioactive compounds, including gymnemic acids, gurmarin, saponins, and flavonoids, which are primarily responsible for its strong antihyperglycemic (blood sugar–lowering) effects. The herb acts through multiple mechanisms. One of its unique properties is the ability to suppress sweet taste perception by blocking sugar receptors on the tongue, thereby reducing sugar cravings [14]. It also stimulates insulin secretion from pancreatic beta cells and may even promote their regeneration, improving overall insulin production. Additionally, Gymnema reduces intestinal absorption of glucose by competing with dietary sugars and enhances glucose uptake in peripheral tissues such as muscle and fat cells, which supports better blood sugar control.Scientific studies have demonstrated that Gymnemasylvestre can lower fasting blood glucose, postprandial (after-meal) glucose, and HbA1c levels, highlighting its potential as an effective herb for diabetes management. Traditionally, it has been used in Ayurveda to treat Madhumeha (diabetes), curb sugar cravings, aid weight loss, and improve digestion.When used appropriately, Gymnema is generally considered safe, although mild side effects such as nausea or stomach upset may occur. Since it can enhance the effects of conventional antidiabetic medications, individuals are advised to monitor blood sugar closely and consult a healthcare provider before use, especially during pregnancy or lactation [15].

Fig 06: Gymnemasylvestre

Momordica Charantia

Momordica charantia, commonly known as bitter melon, is a flowering vine belonging to the Cucurbitaceae family [16]. It is also referred to by several other names, including M. chinensis, M. elegans, M. indica, M. sinensis, and M. operculata. This climbing perennial, which can grow up to 5 meters in length, is extensively cultivated in Asia, India, East Africa, and South America, primarily for its distinctly bitter fruits. While bitter melon is commonly used in cooking, it has long been recognized as a natural remedy, particularly for managing diabetes [17].

In addition to its fruit, other parts of the plant have demonstrated notable health benefits, including the ability to alleviate toothache, diarrhea, and furuncles. Research has highlighted its antidiabetic potential; for instance, a study involving male Wistar rats showed that a diet containing 10% dried bitter gourd improved diabetic conditions and helped prevent common diabetes-related symptoms such as polyuria and polydipsia [18].

The medicinal properties of Momordica charantia are attributed to its bioactive constituents, including momordic 1, momordic 2, and cucurbitacin B, which play a significant role in diabetes management. Additionally, plant phenols present in bitter melon have demonstrated hypolipidemic effects, helping reduce cholesterol and triglyceride levels [19]. Studies indicate that oral administration of bitter melon fruit juice or seed powder can lead to a significant reduction in fasting blood glucose levels. These effects are believed to result from a combination of insulin secretagogue activity and insulin-mimetic properties of its active compounds.

Fig 07: Momordicacharantia

Trigonella Foenum-Graecum

Trigonella foenum-graecum L. (Fenugreek/Mentulu) is an annual herb from the Fabaceae family, widely cultivated in India, the Middle East, and North Africa. Both its seeds and leaves have been traditionally used as food supplements and herbal medicine, particularly for diabetes and digestive ailments [20]. The anti-hyperglycemic effect of fenugreek is mainly linked to bioactive compounds such as 4-hydroxyisoleucine, trigonelline, galactomannan, flavonoids, and saponins, which act together to regulate blood glucose and enhance insulin function. Clinical investigations validate these effects, showing that regular consumption of fenugreek seeds-whether as powder, extract, or soaked form-leads to significant reductions in fasting and postprandial glucose, better oral glucose tolerance, and moderate improvement in HbA1c levels in type 2 diabetes. With its safety, affordability, and accessibility, fenugreek serves as a valuable adjunct to conventional anti-diabetic therapies.

Ocimum Sanctum

Ocimum sanctum L. (Tulsi/Holy Basil), a sacred herb of the Lamiaceae family, originates from the Indian subcontinent and has been treasured in traditional medicine for centuries. It is widely recognized for its therapeutic benefits in diabetes, respiratory disorders, and stress-related conditions. The anti-hyperglycemic potential of Tulsi is largely attributed to its rich phytoconstituents such as eugenol, ursolic acid, rosmarinic acid, flavonoids, and tannins, which collectively regulate glucose metabolism and support insulin activity.

The hypoglycemic action of Tulsi is mediated through multiple pathways. It stimulates pancreatic β-cells to enhance insulin release, while also improving insulin sensitivity in peripheral tissues, thus facilitating efficient glucose uptake. Additionally, Tulsi regulates hepatic carbohydrate metabolism by suppressing gluconeogenesis and glycogenolysis, thereby reducing endogenous glucose output. Its strong antioxidant and anti-inflammatory properties safeguard β-cells against oxidative stress and help prevent diabetic complications. Experimental findings further suggest its ability to influence lipid metabolism, offering added protection against diabetes-associated dyslipidemia [21].

Allium Sativum

Allium sativum L., commonly known as garlic or Vellulli, is a perennial herb belonging to the family Amaryllidaceae. It is widely cultivated and utilized both as a culinary ingredient and a medicinal plant. Throughout history, garlic has held a significant place in traditional medicine systems due to its cardiovascular, antimicrobial, and metabolic benefits. The anti-hyperglycemic activity of garlic is mainly attributed to its sulfur-containing bioactive compounds such as allicin, S-allyl cysteine, diallyl disulfide, and ajoene. These compounds exhibit insulin-mimetic, antioxidant, and anti-inflammatory properties that contribute to glucose regulation.22Garlic exerts its blood glucose-lowering effect through multiple mechanisms. It stimulates insulin secretion from pancreatic β-cells, enhances peripheral glucose uptake, and improves insulin sensitivity, thereby promoting efficient glucose utilization. In addition, garlic inhibits hepatic gluconeogenesis and reduces glycogenolysis, leading to a decline in endogenous glucose production. Its strong antioxidant and anti-inflammatory properties help protect pancreatic β-cells from oxidative stress and inflammatory damage, both of which are key factors in the progression of diabetes. Furthermore, garlic supports lipid metabolism by lowering cholesterol and triglyceride levels, providing cardiovascular protection in diabetic individuals [23].

Fig 08: Allium sativum

Cinnamonum Cassia

Cinnamomum cassia, commonly known as cassia or Chinese cinnamon, is often mistaken for Ceylon cinnamon, though they are distinct in both botanical and chemical characteristics [24]. Cassia has a long-standing history of use in traditional medicine, particularly in Korea, China, and Russia, where it has been widely employed for managing diabetes mellitus. Native to Sri Lanka, cassia thrives in regions of Southeast Asia, favoring environments with temperatures ranging from 10–23°C and altitudes between 100–1200 meters above sea level.25 Belonging to the Lauraceae family, this evergreen tree can grow up to 20–30 feet in height. In addition to its medicinal significance, cassia is a globally popular spice known for its distinctive aroma and versatile culinary applications. Oral administration of cinnamaldehyde has been shown to markedly decrease serum glucose, glycated hemoglobin (HbA1c), total cholesterol, and triglyceride levels. Moreover, it induces a dose-dependent increase in serum insulin, hepatic glycogen, and high-density lipoprotein (HDL) concentrations. These findings support the traditional use of cinnamon as an effective natural agent in regulating blood glucose levels among individuals with type 2 diabetes mellitus [26].

Fig 09: Cinnamonum Cassia

Stevia Rebaudiana

Stevia rebaudiana, commonly known as sugar leaf, candy leaf, or the sweet herb of Paraguay, is a perennial herb belonging to the Asteraceae family. It is native to northeastern Paraguay but is now widely cultivated across Europe, Asia, and North America. Stevia is particularly renowned for its extraordinary natural sweetness, which is approximately 250–300 times greater than that of sucrose [27]. For centuries, it has been traditionally utilized by South American communities as a natural therapeutic agent in the management of diabetes mellitus. This small shrub typically attains a height of about 30 cm, though mature plants may grow up to 80 cm, featuring woody stems and tender green leaves. The plant is abundant in bioactive phytochemicals known for their hypoglycemic, hypolipidemic, and antihypertensive effects. Notably, stevia can reduce postprandial blood glucose levels without stimulating insulin secretion, making it particularly beneficial for individuals with impaired glucose tolerance. In prediabetic women, stevia consumption has been shown to decrease blood glucose levels without significantly affecting 2-hour postprandial glucose concentrations [28].

Conclusion

The escalating prevalence of diabetes mellitus and its associated complications calls for innovative, cost-effective, and safe therapeutic strategies. Although synthetic antihyperglycemic drugs remain the cornerstone of treatment, their limitations-ranging from side effects such as hypoglycemia and gastrointestinal disturbances to lifelong Dependency-underscore the necessity of exploring alternative solutions. Herbal plants, with their diverse phytochemical reservoirs and holistic modes of action, emerge as powerful allies in this regard. Their ability to

enhance insulin secretion, improve insulin sensitivity, delay glucose absorption, reduce hepatic gluconeogenesis, and provide antioxidant protection highlights their multidimensional efficacy. Moreover, their accessibility, affordability, and cultural acceptance make them practical choices, particularly in resource-limited settings where diabetes imposes a severe burden. This integrative potential places them not merely as substitutes, but as complementary interventions capable of augmenting conventional therapies while minimizing their drawbacks.

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Conflict of Interest

Authors are declared that no Conflict of Interest

Informed Consent and Ethical Statement

Not Applicable

Author Contribution

All authors are contributed equally.

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