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Review on Antidiabetic Effect of Bitter Gourd (Momordica Charantia)

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Abstract: *One of the most prevalent illnesses in both developed and developing nations, diabetes, is also spreading quickly throughout the globe. It is estimated that one-third of diabetics take some form of medication, including supplements. The potential of this plant, also called bitter Gourd or bitter Gourd (Momordica charantia), to prevent diabetes has drawn a lot of interest. The fruits of this plant are also used by people in different countries like East Africa, Asia, South America, and India to treat conditions like diabetes. Numerous preclinical studies have confirmed that Bitter melon exerts hypoglycemic and antidiabetic effects through various putative mechanisms. However, data from human clinical trials are limited and biased due to inadequate study design and low statistical power. To help comprehend the possible therapeutic effects of bitter gourd on diabetes, this review highlights the plant's antidiabetic activity along with phytochemical and pharmacological findings. He is also in favor of carrying out clinical trials with more caution*

Keywords: Mother-disease Charantia hypoglycemic medications, Diabetes Insulin, glucose metabolism, bitter melon, medicinal plant, and bioactive compounds

I. INTRODUCTION

Diabetes mellitus (DM), a metabolic disorder with several etiologies, is typified by a continuous rise in blood sugar levels and anomalies in the metabolism of fats, proteins, and carbohydrates brought on by deficits in either the action or secretion of insulin, or both. Organ failure, malfunction, and long-term damage are all possible consequences of diabetes mellitus. Three main classifications exist for diabetes mellitus. Insulin-dependent diabetes mellitus, another name for type 1 diabetes, is an autoimmune disease that arises when the body's pancreatic cells that create insulin are damaged, resulting in a decrease in the amount of insulin that the pancreas can produce. Insulin must be taken daily by a person with insulin-dependent diabetes in order to survive. Children and young adults are usually the ones who encounter it. Known colloquially as "insulin-independent diabetes mellitus," type 2 diabetes was found in over 90% of adult cases of DM. Insulin resistance is a condition that occurs when the pancreas produces enough insulin, but the body does not use it all. Children and young adults are usually the ones who show it. Almost 90% of adult instances of diabetes mellitus were found to be type 2 diabetes, also referred to as "insulin-independent diabetes mellitus." When the body cannot properly use the insulin that the pancreas produces, even though it produces enough of it, insulin resistance is diagnosed. A kind of glucose intolerance known as gestational diabetes mellitus (GDM) usually manifests in the second or third trimester of pregnancy. One of the two main causes of GDM is either an excess of pregnancy hormone or a deficiency of insulin. One of the metabolic disorders linked to pregnancy is gestational diabetes mellitus (GDM). Damage to the kidneys, eyes, nerves, heart, and blood vessels can result from hyperglycemia. According to the 9th edition of the International Diabetes Federation (IDF) Diabetes, 463 million persons worldwide, aged 20 to 79, have diabetes in 2019, and by 2030, that number is expected to increase to 578 million. (Picture 1). This atlas was published by the Israel Defense Forces. Every six seconds, a diabetic person dies. This mortality rate is higher than the combined death rates from HIV (1.5 million), TB (1.5 million), and malaria (600,000).

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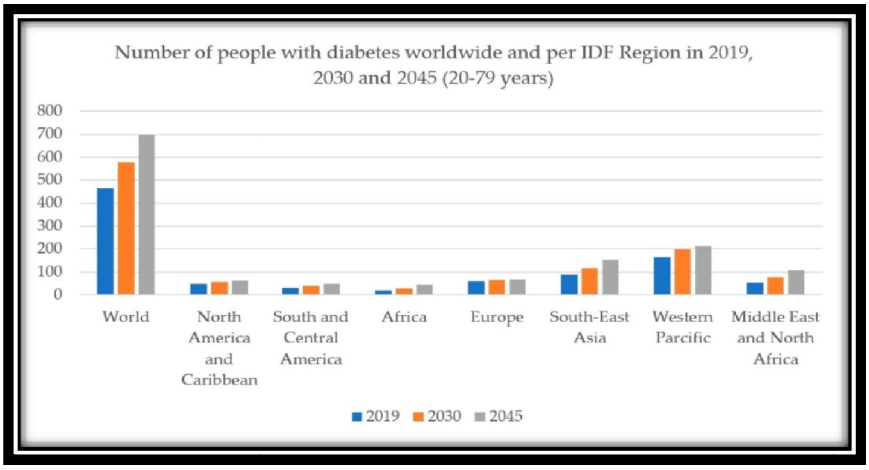

Fig No.1: Global and regional numbers of people with diabetes as reported by the International Diabetes Federation (IDF).

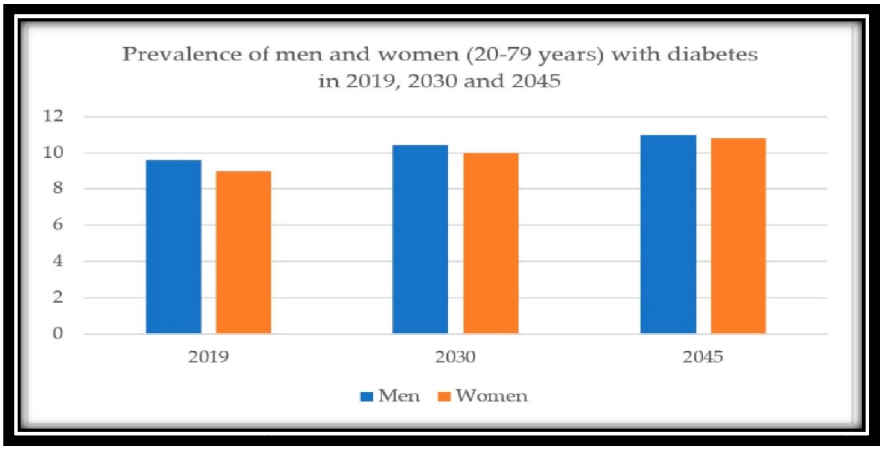

Fig No.2: Diabetes prevalence in men and women (20–79) in 2019, 2030, and 2045.

Herbal remedies derived from medicinal plants continue to be an important therapeutic technique for treating human disease. Diabetes has historically been treated with herbs. Plants and plant extracts have been used to treat diabetes since ancient times. Many commonly used traditional medicines are made from organic materials, minerals, and medicinal plants. A list of 21,000 plants that are used medicinally throughout the world has been compiled by the World Health Organization (WHO). 150 of these are heavily utilized for commercial purposes. Diabetes mellitus is a well-known clinical disorder that can lead to late complications such as neuropathy, nephropathy, and retinopathy. It is common knowledge that natural ingredients play a crucial role in pharmacological biology. Plant expertise can provide valuable perspectives on strategic and sustainable use. Understanding the
active substances found in plant species is currently driving alternative medicine systems. Because M. Charnita possesses strong hypolipidemic and antidiabetic properties, it can be used in conjunction with allopathic medication as an adjuvant to treat diabetes and delay the onset of its consequences. This review has elucidated M. Charnita's possible antidiabetic effect as well as the pharmacological efficacy behind the hypoglycemic action.

Momordica Charantia-

Momordica Charantia, a tropical vegetable that is a member of the Cucurbitaceae family, is grown extensively in Asia, Africa, and the Caribbean for its tasty fruit. Other names for it include balsamic pear fruit, bitter melon, bitter gourd, and bitter apple. Fruit varieties differ greatly in terms of bitterness and form.

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• **Monograph of Bitter Gourd (M.Charantia)-**

Fig No.3-Bitter Gourd (Momordica Charantia)

• **Synonym:** Momordica, Carilla fruit, bitter gourd, and bitter melon.

• **Biological source:** This is a green fruit that is fresh and comes from the **Momordica Charnita Linn** plant.

• **Family:**Cucurbitaceae.

• **Scientific Classification -**

Kingdom:	Vertebratae
Group:	Tracheophytes
Group:	Agiosperm
Group:	Audicots
Group:	The Rosids
Arrangement:	Curbitales
Family:	Cucurbitoideae
Class:	Momordica
Type:	Momordica Charantia

Bionomical Name: M.Charantia

Extraction of Bitter Gourd

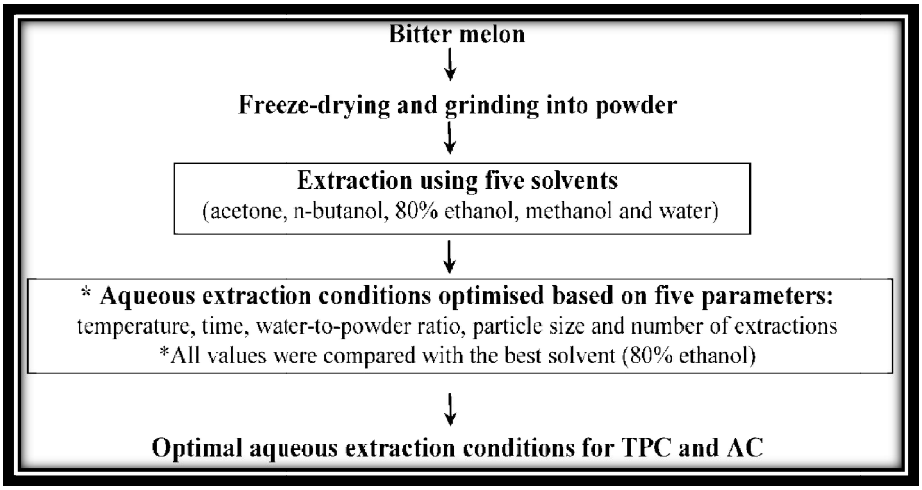

Fig No. 5- Extraction Of Bitter Melon.

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• Storage-

Fruits should be stored at room temperature (25–30°C) for three days. Bitter gourd fruits can be stored for up to six days at room temperature (25–32°C) or ten days at 12°C when packaged in a modified environment with an ethylene absorbent. If the product is placed in a 100-gauge polyethylene bag, stored at 12°C, and treated with 1-MCP (250 pb) after harvest, its storage life can be extended to 14 days, or almost two weeks.

• Nutritional Profile of Bitter Gourd-

The bitter melon is a robust, high- nutrient factory made up of a wide range of profitable substances. These include antioxidants, vitamins, minerals, and bioactive composites, each of which enhances its exceptional stiffness in managing a variety of illnesses. Vitamins C, A, E, B1, B2, and B3 are abundant in fruits, as is vitamin B9, sometimes known as folate. Fruits, seeds, and leaves each had 176.61 Kcal/100 g of calories. In addition, the fruit is rich in minerals, iron, potassium, calcium, zinc, magnesium, and phosphorus, and it contains a high concentration of healthy fiber (bitter melon "causerie," 2008). The medicinal qualities of bitter melon have been linked to its high antioxidant content. Fruit components such as phenols, flavonoids, isoflavones, terpenes, anthraquinones, and glucosinolates are partly responsible for the fruit's bitter taste.

•Nutritional Composition of Bitter Gourd-

The nutritional makeup of M. Charantia from Sorifa (2018) and Gayathry and John (2022) is provided in the table below.

Parameters	Amount
Protein (gram)	0.94
Fat grams	0.15
grams of carbohydrates	0.22
Consumption of Fiber (g)	3.33
Total Sugar Content (%)	3.0
Chlorophyll (mg)	10.8
Thiamine (milligrams)	0.07
riboflavin	0.04
Niacin	0.42
Phosphorous (mg)	71.2
Calcium	21.0
potassium	28.0
Iron	0.8
Sodium	3.4
zinc	0.3
Water Percentage (%)	84.2-91.4
Lipids (%)	0.2-2
Calcium as a carotenoid	212-230IU

Bitter gourd's Antidiabetic Effect: Mechanism of Action

Numerous researchers have examined the impact of various Momordica charantia concentrates and compounds on hypoglycemia and antiglycemia in human and animal models. Bitter gourd and its many extracts and concentrates have hypoglycemic properties that are mediated by a variety of pharmacological, physiological, and biochemical pathways. The primary glycolytic pathway biocatalyst is activated, the primary gluconeogenic enzymes are hidden, islet β cells and their activity are safeguarded, intestinal glucose absorption is restricted, reduce in blood sugar level activity, glucose incitation to the skeletal and peripheral muscles, suppression of adipocyte differentiation and the absence of information about the main gluconeogenic enzymes. This review clarifies the mechanisms underlying Momordica charantia's anti-diabetic effects. Specifically, the plant inhibits glucose metabolism enzymes (fructose-1,6-bisphosphate and glucose-6-phosphatase) and activates AMP-activated protein kinase (AMPK) in pancreatic cells; additionally, the

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plant stimulates fatty acid absorption, increases insulin production, improves insulin resistance, and promotes glucose and fatty acid catabolism. Figure No. 6

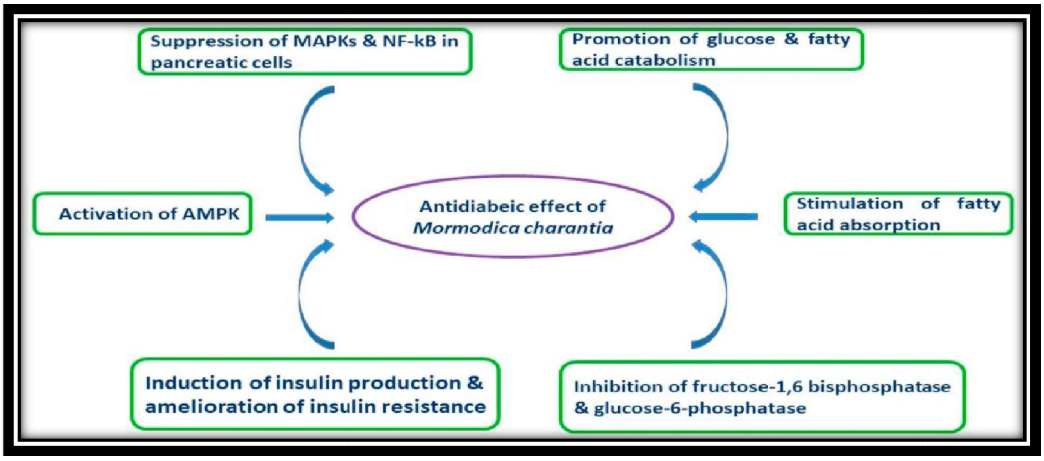

Fig No.6- The mechanism behind bitter gourd's anti-diabetic properties.

• NF-kB and MAPKs are suppressed in pancreatic β-Cells-

A pivotal point in the pathophysiology of type 1 and type 2 diabetes is the loss of pancreatic β-cells. Apoptosis, a systemic process, is brought on by the cytokines interleukin-1β (IL-1β), interferon-gamma (IFN-γ), and tumor necrotic factor-alpha (TNF-α). These cytokines activate a variety of MAPKs, including NF-κB, p38, p44/42, extracellular-regulated protein kinases (ERKs), and stress-activated protein kinase/c-Jun N-terminal kinases (SAPK/JNKs). The result is the β-cells in the pancreas dying (Figure 7). IL-1β causes cell death by stimulating the MAPKs p38, p44/42, and SAPK/JNK. Comparatively, p38 causes pancreatic β-cells to undergo apoptosis, and mitochondrial cytochrome is released when Bcl-2 is phosphorylated by SAPK/JNKs. The combination of TNF-α and IFN-γ also activates SAPK/JNK. Cytokines can also induce cell death by stimulating NF-κB, which in turn triggers caspase-3 activity.

Pancreatic β-cell death is caused by cytokines in two different ways. (1) MAPKs (p38, p44/42, and SAPK/JNKs) are activated by TNF-α, IFN-γ, and IL-1β. These MAPKs then phosphorylate BCl-2. Following the activation of cytochrome C by the phosphorylated Bcl-2, Apaf 1 is recruited and procaspase 9 is converted to caspase 9, which in turn transforms procaspase 3 to caspase 3, finally leading to cell death. On the other hand, caspase 3 is released in response to cytokines activating NF-κB, which causes cell death.

• Promoting the assimilation of fatty acids and the degradation of glucose

Peroxisome proliferator-activated receptor gamma (PPAR-γ) gene expression in adipose tissue, blood glucose levels, and hepatic and serum lipid profiles are all elevated by bitter gourd seeds, according to one study. In M. charantia, PPAR-γ is activated by the phytochemical compound 9c,11t,13t-CLN (Figure). PPAR-γ belongs to the nuclear hormone receptors superfamily, which is a subfamily of ligand-activated transcription factors known as PPARs.

The coordination of numerous cellular and metabolic processes, including energy homeostasis, glucose metabolism, de novo lipogenesis, lipoprotein and triglyceride metabolism, and the uptake, storage, oxidation, and transport of fatty acids, is dependent on PPARs. Additionally, PPARs bind to numerous genes. By functioning as a PPAR-γ ligand activator and promoting the activation of genes involved in lipid catabolism and glucose consumption, M. charantia seed decreases hyperglycemia and hyperlipidemia (Figure 8). By encouraging lipoprotein lipase to break down plasma triglycerides and free fatty acids, activating PPAR-γ has been demonstrated to lower these levels. Moreover, PPAR-γ stimulates fat accumulation, encourages cellular differentiation, and regulates insulin activity in adipose tissue. PPAR-γ activators also increase adipogenesis by encouraging the storage of triacyl glycerides and fatty acids in adipocytes after mean increase insulin sensitivity.

Adipose tissue is stimulated by Bitter Gourd to release PPAR-γ, which has three mechanisms of action against diabetes: (1) increasing rates of glycolysis; (2) upregulating the expression of lipoprotein lipase enzymes, which degrades TAG;

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and (3) increasing adipogenesis and enhancing TAG storage, which increases insulin sensitivity, lowers lipid levels, and increases absorption of fatty acids.

• Insulin resistance is lessened, and insulin production is increased

In the pancreas of STZ-initiated diabetic rodents, Ahmed et al. investigated the effects of daily oral administration of M. charantia natural product juice on the activity of α, β, and δ cells. Jeewathayaparan et al. demonstrated that M. charantia might induce the release of insulin from endocrine pancreatic β cells when taken orally. When M. charantia alcohol concentration was administered to diabetic rats treated with alloxan, significant improvements in islets of Langerhans and strong hypoglycemic effects were seen. Additional research has demonstrated that M. charantia can cause the liver's glucose-absorbing machinery to activate and the endocrine pancreas to release insulin (Figure 10). It has been proposed that the GLUT-4 transporter's recruitment serves as the mechanism for achieving the aforementioned effects.

• Enzyme-dependent protein kinase alpha activation -

Nutrients derived from Bitter Gourd fruits have shown demonstrated potential in improving glucose absorption by cells, inducing insulin release, and amplifying the effects of insulin. The bioactive ingredient in bitter melon stimulates AMPK (AMP-activated protein kinase α), a protein that controls how food is metabolized for energy and improves the uptake of glucose, which is impaired in diabetics. The AMPK anti-diabetes action pathways in muscle and liver tissues are well understood.

AMPK suppresses the synthesis of crucial genes such as CREB-regulated transcription co-activator 2 (CRTC2) and fork head Box O1 (FOXO), which in turn prevents the liver from producing glucose. Additionally, the liver's AMPK activity inhibits the synthesis of cholesterol, de novo fatty acid synthesis, and fatty acid catabolism (Figure 11). In muscle tissue, M. charantia has the ability to activate AMPK, primarily resulting in an increase in the mitochondrial and cytoplasmic fatty acid oxidation.

• Bioactive Compound of Momordica Charantia Act as insulin-

Throughout the tropical world, especially in East Africa, South and North Asia, Vietnam, India, China, and Central and South America, Momordica charantia (MC) is one of the most widely grown vegetables. It belongs to the Cucurbitaceae family and is frequently referred to as bitter gourd and bitter melon (Figure 14). In addition to being eaten as a vegetable, MC is also believed to have traditional therapeutic applications as a herbal remedy. Its bioactivities include anti-inflammatory, antioxidant, antiviral, anti-cancer, and antibacterial properties; its primary characteristic is its ability to prevent diabetes.

Bioactive compounds	Distribution	Pharmacological effects	References
Triterpenoids	Leaves, stem, fruits	Cancer chemo protective, anticancer antioxidant, antidiabetic	[23-26]
Peptides and proteins	Seed	Antiviral, anti-tumour, immune suppressant, antimicrobial	[27-30]
Phenolics	Fruit and seed	Antioxidant, anti-inflammatory, immunostimulant	[31-34]
Saponin	Fruit, root, seed	Antihyperglycemic, hypolipidemic, antiviral, bacteriostatic	[35-39]
Polysaccharide	All parts of plant	Antioxidant, antidiabetic, immune enhancement, neuroprotective, antitumor	[40-44]
Lipid	Seed	Anti-tumor, antioxidant	[45,46]
Steroids	Fruit and pericarp	Antimicrobial	[47-49]

Because bitter melon can cure so many different medical conditions, researchers are becoming more and more interested in learning more about the bioactive compounds in bitter melon and how they affect the body. Nonetheless, as numerous studies documents, Much attention has been paid to the hypoglycemic properties of the anti-diabetic compounds. various published clinical trials have demonstrated the hypoglycemic effects of various bioactive compounds present in the fruit, seeds, and leaves of bitter melon extract in both human and animal models of diabetes

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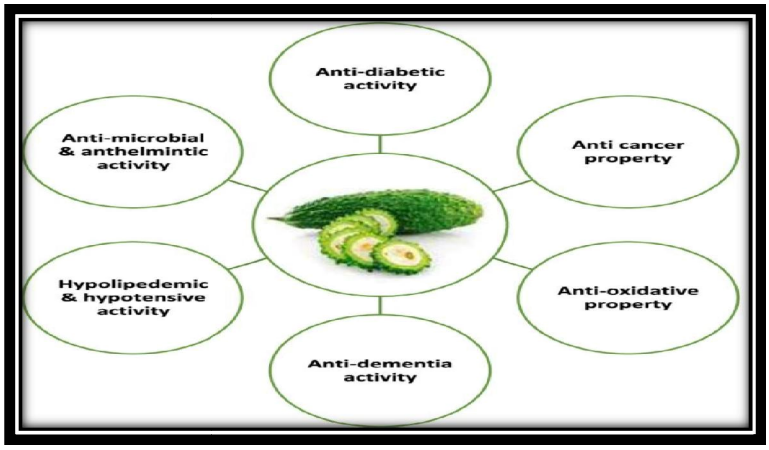
Potential Mechanisms of Bitter Gourd Extract's Action

It is believed that Bitter Gourd and its many extracts and components create their hypoglycemic effects through a variety of physiological, pharmacological, and biochemical pathways. Several mechanisms could be responsible for M. charantia's hypoglycemic actions, including the preservation of islet cells and their functions, the stimulation of glucose utilization in skeletal and peripheral muscle, the inhibition of intestinal glucose uptake, the inhibition of adipocyte differentiation, the suppression of important gluconeogenic enzymes, and the stimulation of a key enzyme of the HMP pathway. The key enzyme of the HMP pathway is activated, significant gluconeogenic enzymes are blocked, and islet β cells and their functions are preserved. Globally, more than 140 studies have looked at the anti-hyperglycemic and anti-hypoglycemic qualities of different bitter gourd extracts and components using both human and animal models.

Kim and Kim also reported that Bitter Gourd extract inhibited the activation of mitogen-activated protein kinases (MAPKs), such as p38, p44/42, and stress-activated protein kinase/c-Jun N-terminal kinase (SAPK/JNK). The results show that Bitter Gourd inhibits NF-κB and MAPKs in MIN6N8 cells, protecting pancreatic β-cells. Influencing the expression of the PPAR-γ gene, bitter gourd improves blood and liver lipid profiles as well as serum glucose levels (related study). Ragasa et al. found that 5α-stigmasta-7-en-3β-ol and clerostrol, two M. charantia sterols, had significant hypoglycemic effects. In diabetic rats, global cerebral ischemia-reperfusion-induced neuronal loss results in neurological impairments. Charantia was discovered to have a strong neuroprotective effect against these neurological impairments. PTP1B, a negative regulator of insulin signaling, is one possible therapeutic target for the management of type 2 diabetes. Numerous physiological and biochemical pathways are thought to be involved in the hypoglycemic effects of M. charantia, its extracts, and its individual components. Among these are the HMP pathway, which also includes the following: inhibition of intestinal glucose absorption, inhibition of adipocyte differentiation, insulin secretagogue-like effect, stimulation of peripheral cell and skeletal muscle glucose utilization, preservation of pancreatic islet cells and their functions, and suppression of important gluconeogenic enzymes.

Nutraceutical Properties of Bitter Gourd



II. CONCLUSION

One of the main ideas in dietetics and nutritional sciences is the idea of food as medicine. Bitter gourd has been used for centuries in diet supplements and ethnomedicine to treat conditions and symptoms related to what is now known as diabetes. Bitter gourd has received a lot of attention from researchers worldwide because of its potential to treat a wide range of illnesses. Reportedly, this adaptable plant has the ability to treat almost any human ailment. This might be as a result of the plant's more than 225 unique therapeutic ingredients. These many substances may act alone or in concert to produce therapeutic benefits. Charantin, insulin-like peptide, and alkaloid-like extracts are the only extracts having hypoglycemic characteristics comparable to those of the plant or its crude extracts in relation to diabetes. These various chemicals appear to have various functions in the treatment and management of diabetes mellitus.

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REFERENCES

[1]. The hypoglycemic impact of traditional Indian herbs used in diabetes treatment is explained by Joseph B. and Jini D. The journal Res J Med Plant. 2011a;5(4):352–376. [Source: Google Scholar]

[2]. Bandara, R.; Medagama, A.B. Is it safe and effective to continue using complementary and alternative medicines (CAMs) to treat diabetes mellitus? 2014, 13, 102; Nutr. J. [Cross Reference]

[3]. Global Federation for Diabetes. The International Diabetes Federation (IDF) published the 9th edition of the IDF Diabetes Atlas in 2019.

[4]. PMID 15182917 (https://pubmed.ncbi. nlm.nih.gov/15182917).

[5]. Extraction, quantification, and antioxidant activities of phenolics from the pericarp and seeds of bitter melons (Momordica charantia) harvested at three maturity stages (immature, mature, and ripe) Horax R., Hettiarachchy N., Chen P. 2010;58:4428–4433 in J. Agric. Food Chem. The doi is 10.1021/jf9029578. [PubMed] [CrossRef] [Source: Google Scholar]

[6]. The study conducted by Gadang et al. (2011) found that dietary bitter melon seed enhances the expression of peroxisome proliferator-activated receptor-γ gene in adipose tissue, decreases the expression of nuclear factor-κB, and mitigates symptoms related to metabolic syndrome.

[7]. Human beta cell mass and function in diabetes: new insights and technological developments to comprehend the etiology of the disease, C. Chen et al., Mol. Metabol. 6 (9) (2017) 943–957.

[8]. Interleukin-1β induces tissue factor expression in A549 cells through both EGFR-dependent and -independent mechanisms, according to T. Mechelke et al.'s study published in Int. J. Mol. Sci. 22 (12) (2021) 6606.

[9]. Differential mechanisms of Erk-1/2 and p70S6K activation by glucose in pancreatic #2 cells, by.I. Briaud et al., Diabetes 52 (2003) 974–983.

[10]. C. Tournier and colleagues, Need for JNK in stress-induced cytochrome c-mediated death pathway activation, Science 288 (5467) (2000) 870–874. Reference [110] beta-cell apoptosis: triggers and communication, Diabetes 50 (Suppl 1) (2001) S58–63, T. Mandrup-Poulsen.

[11]. W.H. Kim, and colleagues, Joint activation of JNK/SAPK by TNF-alpha and IFN-gamma: apoptosis of pancreatic beta-cells through the p53 and ROS pathway, Cell Signal. 17 (12) (2005) 1516–1532.

[12]. In Signal transduct, Target Ther. 2, 2017, e17023, T. Liu and colleagues.

[13]. Kim, H.Y., "Extract from bitter melon (Momordica charantia) suppresses cytokine-induced activation of MAPK and NF-κB in pancreatic β-Cells," Food Sci. Biotechnol. 20 (2) (2011) 531–535.

[14]. In the bitter gourd (Momordica charantia L.), fractionation and identification of 9c, 11t, and 13t-conjugated linolenic acid as an activator of PPARalpha were reported by Chuang et al. in J. Biomed. Sci. 13 (6) (2006) 763–772.

[15]. 14. Global estimates of the prevalence of diabetes for 2010 and 2030, Shaw JE, Sicree RA, Zimmet PZ. 2010;87:4–14; Diabetes Res Clin Pract. [PubMed] [Scholar Google]

[16]. John, J., and Gayathry, K.S. 2022. An exhaustive analysis of bitter gourds (Momordica charantia L.), a treasure trove of useful bioactive ingredients for therapeutic foods. 4(10):1–14 in Food Production, Processing, and Nutrition.

[17]. Khan, M.U. Lifestyle Modification in the Prevention of Type II Diabetes Mellitus. Oman Med. J. 2012, 27, 170–171. [CrossRef] [PubMed]

[18]. Herbal Antidiabetics: A Review, Int. J. Res. Pharm. Sci. 2011, 2, 30–37, Bhoyar, P.K., Tripathi, A.K., Baheti, J.R., Biyani, D.

[19]. An overview of the phytopharmacological characteristics of Ficus racemosa Linn by Joseph B. and Raj S. J. 2010b;3(2):134–138; Int J Pharm Sci Rev Res. [Source: Google Scholar]

[20]. Joseph B, Jini D, Ajisha SU. Characterization of the phytochemistry of a herbal medication formulation for arthritis. 2012;6(2):54–60; Res J Phytochem. [Source: Google Scholar]

[21]. Phytopharmacol. 2012;2(1):144–169. 9. Singh U, Singh S, Kochhar A. Therapeutic potential of antidiabetic neutraceuticals. [Source: Google Scholar]

[22]. The domestication of plants in antiquity Hopf M., Zohary D., p. 122 Oxford: University Press of Oxford, 2000. [Refer to Google Scholar] 11. Cefalu WT, Ye J, Wang ZQ. The impact of botanical dietary

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supplements on human carbohydrate metabolism. Drug Targets for Endocr Metab Immune Disord. 2008;8:78–81. [PubMed] [Scholar Google]

[23]. Cousens G. The Tree of Life 21-Day Program is a diabetes cure. North Atlantic Books, California, 2008, pp. 191-292. [Scholar Google] 13. Joseph B, Raj SJ. Pharmacognostic and phytochemical properties of *Aleo vera* Linn - An overview. *Int J Pharm Sci Rev Res*. 2010a;4(2):106–110. [Google Scholar]