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"Review on: Anti-hyperglycemic effect of banana flower"

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ABSTRACT:
Postprandial hyperglycaemia is characterized as the earliest symptom of diabetes & its management attenuates several of the associated secondary complications. In this context, we investigated the role of ethanol extract of banana flower (EF) for its antihyperglycemic effects. The effect showed a strong inhibition towards a-glucosidase of pancreatic amylase which play a vital role in clinical management of postprandial hyperglycaemia. The major active Compound present in effect were identified as a umbelliferon (CI) & Lupeol (C2) using various spectroscopic methods. were found to inhibit a-glucosidase is a non-competitive mode of inhibition with low key values. Further, in vitro glycation assays showed that Ef & its compounds prevented each Stage of protein glycation & formation of it□s a Intermediary compounds. Ef, C1 & C2 also exhibited a potent inhibition on aldose reductase. Our results suggest that, the observed potential of EF in antihyperglycemic activity via inhibition of a-glucosidase & in anti diabetetogenic effect by inhibition of polyol pathway & protein glycation is more likely to be attributed to the presence of type 2 diabetes .Therapeutic options for diabetes are diet, exercises Oral hypoglycaemic drugs, & insulin therapy Treatment with oral hypoglycaemic agent associated with side effects related to kinetic properties, Secondary failure rates, hypoglycaemia, gastrointestinal disturbances, Skin pains, dermatological disorders, & rise in hepatic enzyme level. Metabolic abnormalities in Carbohydrates, lipids & proteins result from importance of insuring anabolic hormone. Low levels to achieve adequate response & for insulin resistance of target tissues, mainly skeletal muscles, adipose tissue, & to a lesser extent, liver, at the level of insulin receptors, signal transduction system & or effector enzyme or genes are responsible for these metabolic abnormalities .

□ **Keywords** □ Diabetes mellitus; ethanol extract; alpha-glucosidase inhibitors; Glycation; Aldose reductase; Umbelliferone; Lupeol,etc.

INTRODUCTION

Diabetes mellitus is a chronic disorder Characterized by hyperglycaemia, elicited due to a disfunctional carbohydrate, lipid & protein metabolism colth absolute / relative deficiencies the insulin secretion or its action.[1] Diabetes is a gap of lifelong metabolic disorders Caused by defective insulin secretion or impaired insulin action. The International Diabetes Federation estirnapes that there are 463 million people .Cgs.1% confidence interval: 869-601 million living with diabetes in 2019, half of however, remain undiagnosed due to diabetes.[2]

1.1.History

•*DIABETES IN INDIA*

Diabetes is one of the morbidity & mortality problem : leading causes worldwide & a major Vin India In 2012, 60.1% of all death in India were due to non communicable diseases (NCDs).[5] The report of national Urban Diabetes Survey Conducted in metropolitan cities in India confirmed that the prevalence of T2DM was 16.6% in Hyderabad, 13.5% in Chennai, 12.4% in Bangalore, 11.7% in Kolkata, 11.6% in New Delhi, 9.31% in Mumbai and 6.1% in Kashmir valley. The urban-rural differences in the prevalence of diabetes have been consistently reported.[9] As per the International Diabetes Federation (IDF) Atlas, 2019, the prevalence of diabetes in India was 77 million in 2019. India also ranks second in the world for no. of people aged >65 years with DM (12.1 million & this number is expected to rise to 27.5 million by 2045).[7] Alarmingly, the estimated number of people with undiagnosed diabetes is 43.9 million, Traditionally, the dietary interventions derived From natural resources have been Identified for its potential in the management of diabetic Complications. Through the mode of action is not scientifically explained pertaining to mast of the traditional medicinal formulas, their effectiveness in improving the condition have justified their uses in Indian traditional system of medicines.[3] Diabetes es is also well characterized by peripheral insulin resistance, which in turn Jesuits in reduced glucose uptake by the tissues, leading to persistently high postgrandial blood glucose level.[4]

accounting For 5710 of the case load [6] The no. of people living with diabetes in predicted to rise to log million by 2035.[7]

Therapeutic options for diabetes are diet, exercises Oral hypoglycaemic drugs, & insulin therapy Treatment with oral hypoglycaemic agent associated with side effects related to kinetic properties, Secondary failure rates, hypoglycaemia, gastrointestinal disturbances, Skin pains, dermatological disorders, & rise in hepatic enzyme level.[8] Fervid absorption of dietary carbohydrates aided by glycoside hydrolases (a-glucosidases, d-amyler) Converts them into simpler monosaccharide units, which results in elevated blood glucose levels Characterized as postprandial hyperglycaemia. This is distinguished as the curtest symptom of & the use of glycoside diabetes, inhibitors is widely accepted as an efficient method in restraining postprandial, hyperglycaemia by inhibiting the release of free glucose units facilitating a smooth

glucose profile.[9] Diabetes mellitus (DM) is rising to an alarming epidermal level.

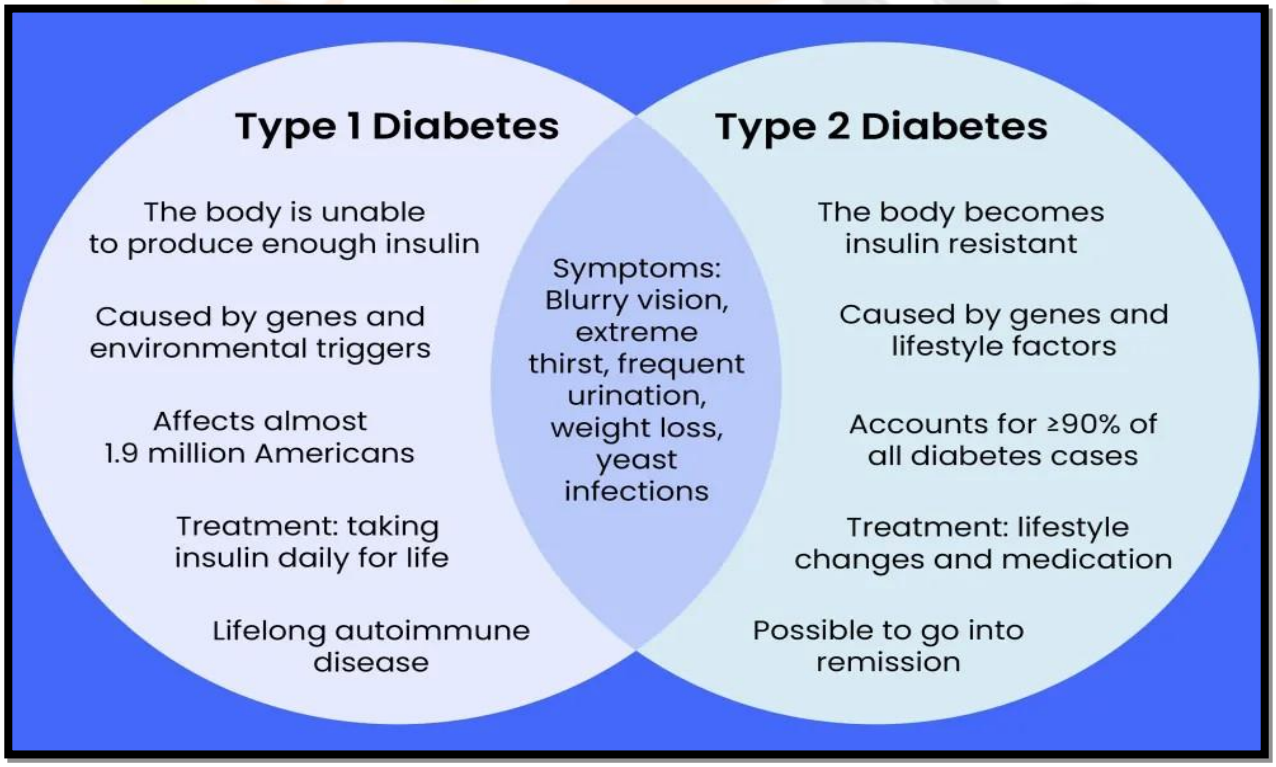
DM is a group of metabolic diseases Characterized by chronic hyperglycaemia resisting From defects in insulin secretion, insulin action, or both. Metabolic abnormalities in Carbohydrates, lipids & proteins result from importance of inswing anabolic hormone. Low levels to achieve adequate response & for insulin resistance of target tissues, mainly skeletal muscles, adipose tissue, & to a lesser extent, liver, at the level of insulin receptors, signal transduction system & or effector enzyme or genes are responsible for these metabolic abnormalities The severity of symptoms is due to by & duration of diabetes.[10]

There are two types of diabetes mellitus:-

Type 1 diabetes mellitus and

Type 2 diabetes mellitus

Fig1: Types of diabetic mellitus



2.Sign and symptoms of diabetes mellitus

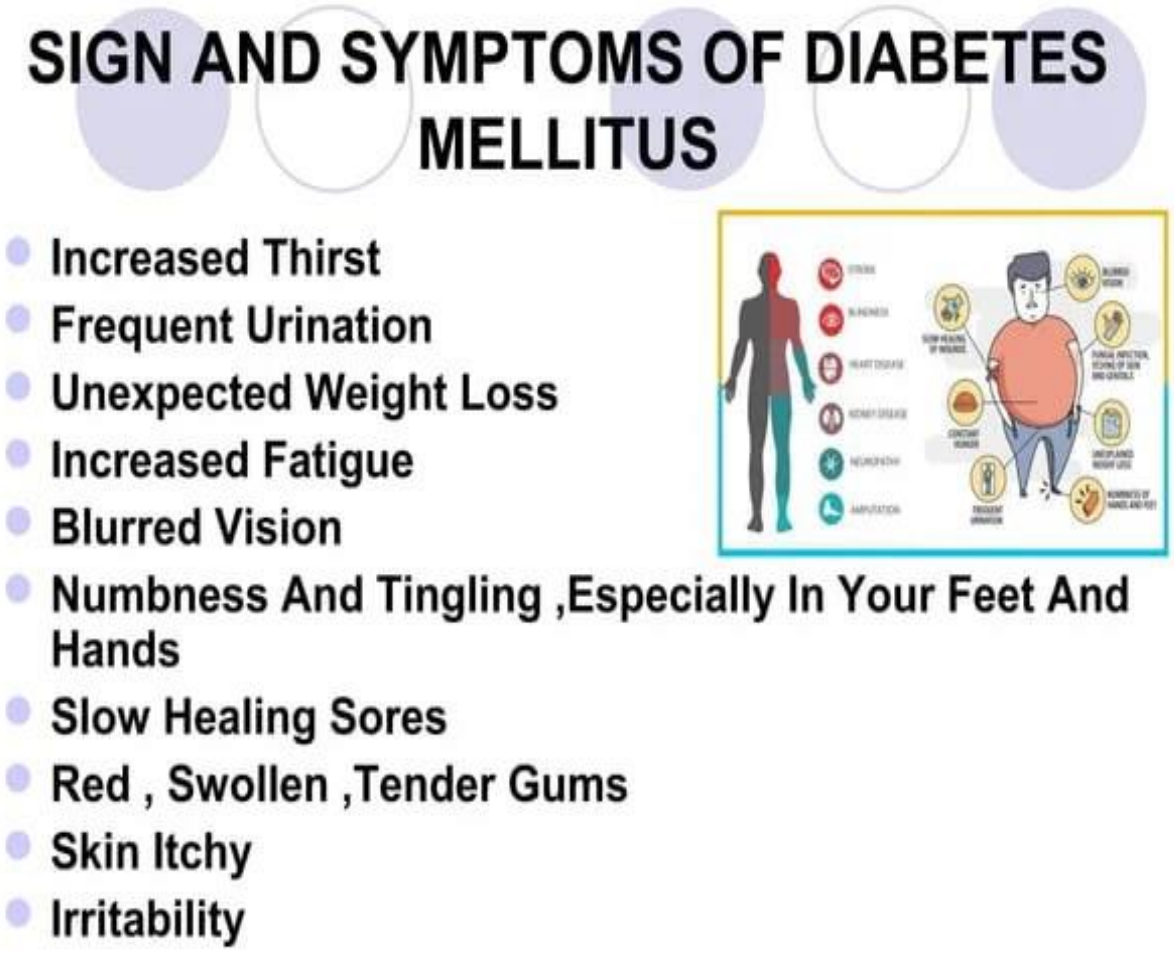


Fig2:Signs and symptoms of diabetes mellitus

3.Prevention of Diabetic Mellitus

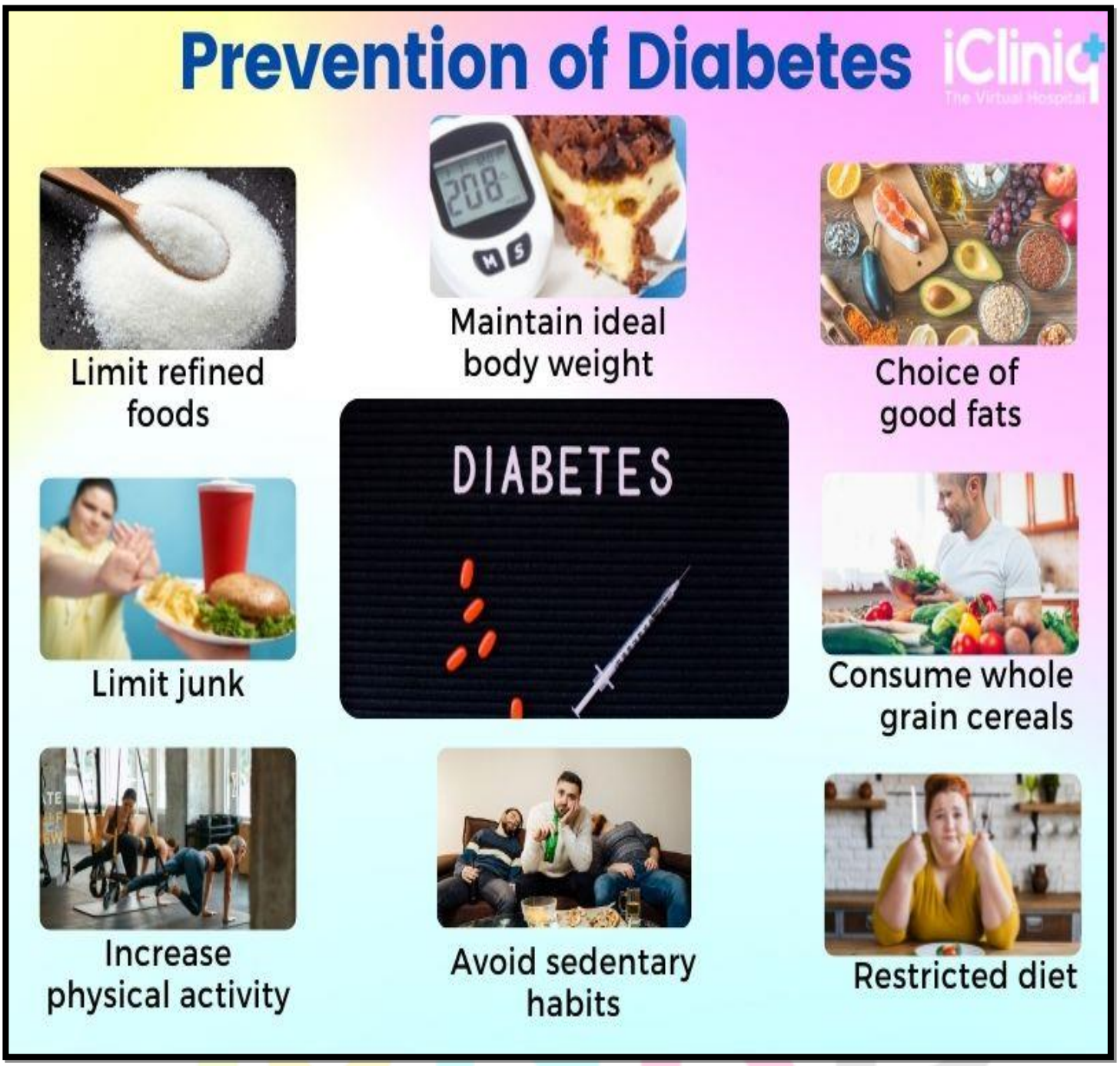
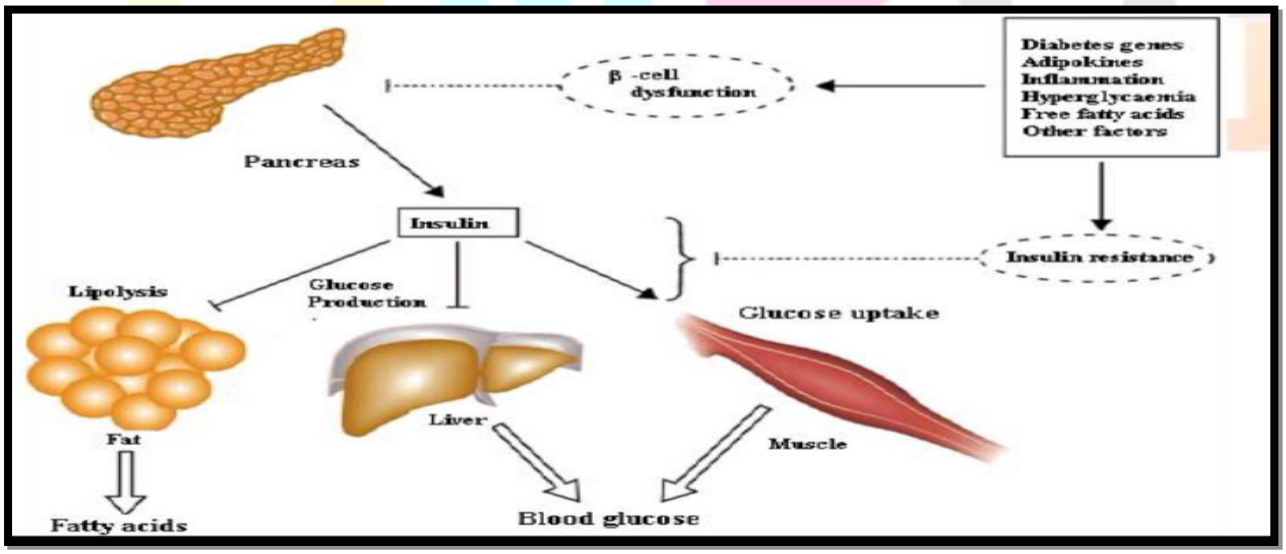


Fig3: Prevention of diabetic mellitus

4 .Patho-physiology of diabetes mellitus

Insulin resistance in muscle and liver and beta cell failure represent the core pathophysiologic defects. In type 2 DM (TRDM). In the liver, the Insulin resistance is manifested by an overproduction of glucose during the basal state despite the presence of fasting hyperinsulinemia , as occurs following a meal. In the muscle, the insulin resistance of manifest by impaired glucose uptake following ingestion of a carbohydrate and resultant postprandial hyperglycaemia.[11] Insulin secretion from the pancreas normally seduce glucose output by the liver, enhances glucose uptake by skeletal muscle & supresses Fatty and release from fat tissue. The various Factor□s Shown that contribute to the pathogenesis Of T2DM affect both insulin secretion & insulin action. Decreased insulin secretion will reduce Insulin signalling in its target tissues. Insulin resistance pathways affect the action of insulin in each of the major target tissues ,leading to Increased circulating Fatty acids & the hyperglycaemia of the major larger tissue, leading to increased Circulating fatty acids and the hyperglycaemia of diabetes. In turn, the raised concentration of glucose and fatty acids in the bloodstream will feed back to worsen both insulin secretion and insulin resistance (Figure2).[12]In addition to the muscle, liver of B-cell (triumvirate) the fat cell ,(accelerated lipolysis), gastrointestinal tract (incretin deficiency/resistance), alpha cell hyperglucagonemia), kidney (increased glucose reabsorption) & brain (insulin resistance) all play imp roles in the development of glucose intolerance in T2DM Individuals. Collectively these eight players comprises. The Ominous octet & dictate that: 1) multiple drugs used in combination will be required to correct the multiple pathophysiological defect, 2) treatment should be based upon reversal of known pathogenic abnormalities and not simply on reducing HbA1c, & (3) therapy must be started early to prevent/ show the progressive beta-cell failure that already is well established in impaired glucose tolerance (ZAT) patients.[13]

Fig4: Pathophysiolgy of hyperglycemia and increased fatty acids in Type 2 diabetic mellitus



5.Complications of diabetes

DM may induce several complications (microvascular and macrovascular)can co-exist with other diseases The macrovascular complications, which are more severe, are coronary disease (stroke of peripheral neuropathy. The microvascular affect in the long-term may lead to macrovascular complications such as diabetic retinopathy, diabetic nephropathy 5 diabetic foot.[14]

Diabetic retinopathy is common in both TLDM (40%~ & T2DM (200%). Diabetic nephropathy is one of the most imp. Causes (61%) of end-stage renal disease that requires renal replacement. Diabetic neuropathy is a common complication of diabetes that results in damage to the nerves due to high blood sugar level for a long period of time. Spontaneous intracerebral haemorrhage (slCH), defined as Spontaneous bleeding into the brain, accounts for 10 to 2010 of all strokes, & to associated with diabetes .In terms of macrovascular complications, patients with DM have a 2 to 4 times higher size of cardiovascular disease & up to a 3 times increase in mortality than non-diabetics.[15]

In the context of the ongoing COVID-19 pandemic ,DM is known to affect outcomes. The reason for worse. Prognosis in people with diabetes is likely to be multifactorial, wherein comorbidities Such as hypertension & cardiovascular disease, obesity, and a pro-inflammatory and pro-coagulative state probably contribute to the risk of worse outcomes. The infection itself might represent a worsening factor for people with diabetes, as it can precipitate acute metabolic complications through direct negative effects on B-cell Function.[16]

There is a higher prevalence of diabetes among severe patients than non-severe patients (10.8% VS 5.4.10), & the mortality rare of COVID 19. Among diabetic patients is 7.310, higher than overall mortality rate (2.3%).[17]

6. Diabetes Management : Diet, Lifestyle & Medication

The management of diabetes include lifestyle changes that involves strict diet and exercise along with oral hypoglyemic There is strong & Consistent evidence that obesity management can delay the progression from pre diabetes toT2DM & iI beneficial in the treatment of T2DM.[18]

Recombinant macronutrient intake-

Macronutrient	Cardian guideline [19]	AOH [20]	ICMR [21]
Carbohydrate	• > 130 g/day • % total daily energy from carbon hydrate, >45%; 60% if low Glycemic index, High fiber foods.	60-70% of energy intake	55-60% energy from carbohydrate
Dietary fiber	Age 19-50 years; • 25 g/day (Women) • 38 g/day (Men)	25-50 g/day	40 g/day
Fat	PUFA Age 19-50 years; • 12 g/day (Women) • 17g/day (Men) Age > 51 years; • 119 g/day (Women) • 149 g/day (Men)	< 30% of daily energy intake	20-30% of total energy intake
Protein	0.8 g/kg body wt. for adult men & women>18 years of age.	15-20% of daily energy intake	12-13% of total energy

Fig5: Nutritional requirements of diabetes

•Nutrition as a component of diabetes care

6.1.American Diabetes Association (ADA)

In patients with T2DM who also have overweight or obesity, modest and sustained weight loss has been shown to improve glycemic control and to reduce the need for glucose-lowering medications. Diet, physical activity and behavioral therapy designed to achieve and maintain 25% weight loss is recommended for patients with T2DM who have overweight or obesity and are ready to achieve weight loss. To achieve weight loss of >5%, short-term (3-month) interventions that use very low-calorie diets ($800 kcal/day) and meal replacements (MR) may be prescribed for carefully selected patients. The ADA confirms

that studies of reduced calorie interventions show reductions in HbA1c of 0.3 to 2.0% in adults with T2DM, as well as improvements in medication doses and quality-of-life. Sustaining weight loss can be challenging but has long-term benefits. Maintaining weight loss for 5 years is associated with sustained improvements in HbA1c and lipid levels.[22]

6.2.Diabetes Canada Clinical Practice Guidelines Expert Committee [23]

•People with diabetes should receive nutrition counselling by a registered dietitian.

•Nutrition therapy can reduce HbA1c by 1.0 to 2.0% and, when used with other components of diabetes care, can further improve clinical and metabolic outcomes.

•Reduced caloric intake to achieve and maintain a healthier body weight should be a treatment goal for people with diabetes with overweight or obesity.

•The macronutrient distribution is flexible within recommended ranges and will depend on individual treatment goals and preferences.

•Replacing high-glycemic-index carbohydrates with low-glycemic-index carbohydrates in mixed meals has a clinically significant benefit for glycemic control in people withT1DM and T2DM.

•Weight loss programs for people with diabetes may use partial MR plans. Commercially available, portion-controlled, vitamin- and mineral-fortified meal replacement products usually replace 1 or 2 meals per day in these plans.

7.Diet and diabetes: The Indian context

Asian Indians appear genetically predisposed to DM, most likely through the interplay of multiple susceptible genes. However, recent environmental and behavioral change appears to be the primary driver of the increasing prevalence of obesity, insulin resistance and T2DM.

India□s rapidly developing economy has driven the expansion of urbanized zones, altered social and employment patterns, and provided greater access to high-fat processed foods that are often consumed outside of the home.

In recent decades, refined vegetable oils have become more plentiful and affordable and an increasing number of meals and snacks are being consumed out of the home.

Fast- and snack-foods are generally high in fat, and also commonly contain trans fats, both of which can contribute to insulin resistance.

The average daily intakes of fruits and vegetables are generally low.

There is generally a low level of knowledge related to the healthy food choices and the basic concept of □simple carbohydrates□ and dietary fiber.[24]

8.TREATMENT OF DIABETES MELLITUS

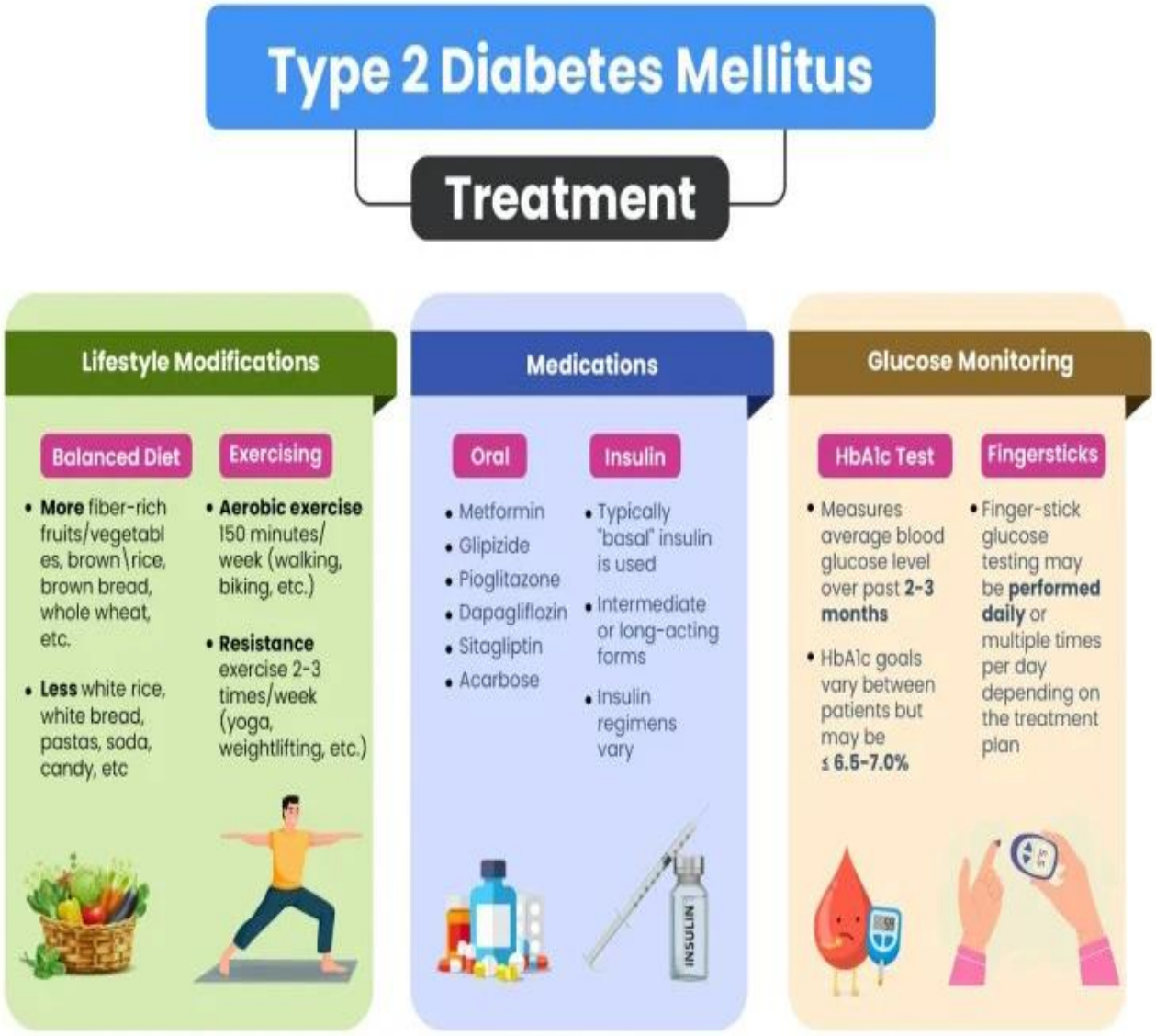


Fig 6: Treatment of diabetic mellitus

9.BANANA PLANT

Banana plant, often erroneously referred to as a □tree□, is the largest herbaceous flowering plant .[25] Banana belongs to the family mucosae, genus muse & is number oε $ is a general term embracing a speeres hybrids in this genus .[26]

Banana (musa sp.) in considered one of the most Important favourable of popular fruits in Egypt & all over the world.[27] Different plants of banana plant are known to be used in Ayurveda & other traditional Folklore medicine for treatment of various disease including diabetes.[28]

The use of other parts of the banana plants, such as leaves & sheaths pseudo Stem, Pith I male bud has been reported recently.[29]

Fig7:Banana plant

10.BANANA FLOWERS

Banana flowers are large, dark purple-red blossoms that grow from the end of a bunch of bananas They key ate also called banana inflorescence banana blossom, banana male bud [30]or banana heart.[31]They are an agricultural by-product that is often consumed as vegetable in many countries speech Malaysia, Indonesia, Srilanka, Philippine□s & other South-East Asian, Countries. In Sri Lanka, It is consumed as a curry as well as a boiled or deep fried salad with rise & Wheat bread.[32]

Besides being consumed fresh banana blossom can also be made various products such as dehydrated, vegetable, pickle & conned food.[33] Also, dehydrated inflorescences (male Flowers & bracts) are a great nutritive complement based on their high content of potassium & fibre. In view of their high nutritional value, inflorescences (male flower bracts) can be used in the diet in the form of demonstrated flour & these easily incorporated into food showed by.[29] Banana, Flowers have, tremendous nutritional value & heathy effects. They are used in treatment of bronchitis, constipation ulcers & good for diabetics. It is traditionally believed to be beneficial as a lactating agent & helps to relieve painful menstruation. Spectrophotometric constants for common haemoglobin derivatives in human, dog and rabbit blood . The banana flower is rich in phytochemicals like vitamins, proteins and

flavonoids.[26] These banana blossoms are usually thrown away by producers in plantations fare one of the agricultural by-products which are getting more attention from many researchers &food manufactures as a food Source.[34] Therefore, the main target of the present Study was to evaluate the therapeutic effect of different concentration & food mag as of dried banana Flower (Banana male bud).

Fig8: Banana flower

11.POWDER DOSAGE FORM

11.1.Banana male bud powder

Preparation of banana male hurts powder :-

Freshly banana (musa acuminate & castivar Williams mare buds were washed with running tap water, cleaned, cut into slices & soaked to came acid 5010 ↓

Then dried at 40°C by hybrid solar convective. Drying system, belonging to the solar energy dept. National Research Centre, Dokki Egypt, powdered, stored at 4° (for further analyses).

11.2. Chemical Analysis of dried banana male bud

5.2.1. Chemical composition of Banana male bud

Moisture, ash, protein, fat & crude fibre core determined in dried banana male bud according to method described in (A.O.A.C2005). [35] While; Carbohydrate was determined by dinitrosalicylic and method.[36]

5.2.2.Sugar composition

Sugars (glucose, fructose & sucrose) were determined by High Performance Liquid Chromatography (HPLC) on an Agilent model 1100 series system (Agilent,USA)

12.MATERIAL AND METHODS

12.1.Chemicals

Butylated hydroxyl anisole (BHA), 1,1-diphenyl-2-picrylhydrazyl (DPPH), 2,2′-azinobis-3-ethylbenzthiazoline-6-sulfonate (ABTS), phenazine methosulfate (PMS), nitro blue tetrazolium (NBT), gallic acid, dinitro phenyl hydrazine (DNPH), l-cysteine, porcine pancreatic α-amylase (EC 3.2.1.1), α-glucosidase (EC 3.2.1.20) from Saccharomyces cerevisiae, p-nitrophenyl-α-d-glucopyranoside (pNPG), β-

NADPH and aminoguanidine were obtained from Sigma-Aldrich (St. Louis, MO, USA). Thiobarbituric acid (TBA), trichloro acetic acid (TCA), sodium carbonate and nicotinamide adenine dinucleotide-reduced (NADH) were obtained from Sisco Research laboratories (Mumbai, India). Acarbose (Glucobay, 50 mg) was obtained from Bayer India (Thane, India). Reagents and solvents used for extraction and silica-gel for column chromatography were procured from Merck (Mumbai, India). All of the other reagents were of analytical grade.

12.2.Plant Material

inflorescences of Musa sp. Cv. Nanjangud rasa bale were collected from banana cultivating farms of Nanjangud, Karnataka, India. The specimen was identified by the Department of Horticulture, Government of Karnataka, Mysore, India. Flowers were separated from the inflorescence followed by discarding the spathe. The isolated flowers were cleaned, cut into small pieces and dried at 40 °C in an oven. This was powdered using a homogenizer and further stored at 4 °C until use.

13.EXTRACTION

The coarse powder was subjected to hot extraction using ethanol in a Soxhlet apparatus. Extraction was performed twice with 95% ethanol (500 ml) and filtered. The resulting filtrate was concentrated under vacuum using rotary evaporator (Rotavapor R-200, Buchi, Switzerland) and the yield of ethanol extract was recorded. Subsequently, ethanol extract of banana flower (EF) was subjected to preliminary phytochemical screening to identify the EF was estimated as per.[38]

14.METHOD OF PREPARATION

Banana male bud Powder-

Freshly banana (musa aruminate L.) cultivar Williams male buds were washed with running tap water, cleaned, cut into slices and soaked in citric acid solution. Then dried at 40°c by hybrid Solar convective drying system, belonging to the Solar energy dept., National Research centre, Dokk, Egypt, powdered & stored at further analyses) 4° for further analyses).

15.MODE OF ACTION

 Inhibiting a-glucosidase: Banana flower extract (EF) and its constituents inhibit the a-glucosidase enzyme, which decreases the rate of glucose absorption during digestion.[39]

 Inhibiting aldose reductase and glycation: EF and its constituents inhibit aldose reductase and glycation, which may help with secondary complications of diabetes.[40]

 Reducing advanced glycation end- products (AGEs): Banana flower supplements can inhibit the accumulation of AGEs in diabetic rats.[41]

Improving glucose tolerance: Oral administration of chloroform extract of banana flowers (MSFEt) can improve glucose tolerance in experimental diabetic rats.[42]

 Reducing blood glucose: MSFEt can reduce blood glucose and glycosylated haemoglobin.

Increasing total haemoglobin: MSFEt can increase total haemoglobin.[43]

16.CONCLUSION

In conclusion,a EF extendes a remarkable antihyperglycemic effect via inhibition of carbohydrate hydrolysing enzyme as well as ameliorates the diabetic induced complications by inhabiting AR and AGE related pathway. The beneficial effect EF are in agreement with the positive effect of the isolated umbelliferon and Lupeol ,suggesting them as potential antidiabetic agents. Further ,our result are intricately linked to the antioxidant activities of EF and its constitute which suggest its association with the antidiabetic properties.Also,identification of significant amount of umbelliferon (0.38%) and Lupeol(0.36%) in EF provided an insight for a strong chemical basis to the alleged beneficial role in EF in antidiabetic properties. However,in vivo studies to provide a stronger basis for these evidence are essential before it can therapeutic procedures.

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