Moringa Potential Mechanisms to Control Hyperglycemia in Type 2 Diabetic Rats Induced by HFD and Low-Dose of STZ

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Abstract:

Diabetes is projected to be the 7th leading cause of death globally by 2030 and is a known risk factor for COVID-19. While Moringa has shown potential in managing type 1 diabetes, its effect on type 2 diabetes may need more studies. This study evaluates Moringa oleifera's ability to reduce glucose levels in type 2 diabetic rats by improving insulin sensitivity and inhibiting digestive enzymes like α-amylase and α-glucosidase, along with its anti-inflammatory and antioxidant activities. Type 2 diabetes was induced in rats using a high-fat diet (22% fat) for 4 weeks and single low-dose streptozotocin (35 mg/kg B.W. STZ), then divided into control and diabetic groups, with varying Moringa dosages (2.5%, 5%, 10%) added to the diabetic rat's diet for 4 weeks. Results indicated that Moringa has significant levels of fiber, proteins, phenols, flavonoids, and antioxidant activity. Moringa improved diabetic symptoms by inhibited digestive enzymes, reducing blood glucose, insulin resistance, and inflammation while enhancing antioxidant properties in serum and pancreatic tissues. It also improved lipid profiles, and protected liver and kidney functions. Additionally, sensory evaluation of bread and pizza with Moringa supplementation showed good acceptability, although higher doses affected color. The study suggests Moringa's potential in managing type 2 diabetes and recommends further research to detect ideal safe dose, usage duration and methods in humans.

Keywords: Moringa oleifera, glucose level, insulin, HOMA-IR.

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INTRODUCTION

Diabetes mellitus is regarded as one of the main epidemics in human history **(Zimmet, 2017)**, and it is between the 10 top reason of adult mortality worldwide, with a prevalence of around 10% **(IDF, 2019; Zheng *et al*., 2018).** It is forecasted to be the 7th worldwide reason of death by 2030 plus total deaths by diabetes are predictable to increase at next ten years greater than 50% **(Mathers and Loncar, 2006)**. Additionally, it is a well-known risk factor for COVID-19, increasing the likelihood of critical illness and death in affected patients, according to epidemiological studies. **(Pratiwi *et al.*, 2021)**. The complications associated with diabetes contribute significantly to morbidity among those affected **(Ghasemi, 2019).** This chronic metabolic condition is defined by persistent hyperglycemia and insulin dysregulation, which are frequently associated by overweight and obesity **(WHO, 2022; IDF (2021)**. Type 2 diabetes (T2D) is a complex, diverse, and polygenic disease described by insulin resistance in target organs and reduced insulin generation by pancreatic beta cells **(Rahmati Najarkolaei *et al*. 2017).**

Animal models are critical for studying type 2 diabetes and evaluating new treatments. One extensively used model involves establishing obesity in mice with a high fat diet (HFD) and then providing a low dose of STZ (streptozotocin) to cause diabetes. This model accurately matches the essential aspects of human type 2 diabetes, including insulin resistance and beta-cell failure. Research has demonstrated that feeding rodents an HFD for 2-7 weeks results in steady insulin resistance, and that combining an HFD with modest dose of STZ causes diabetes in rats **(Mansor *et al*., 2013)**. High-fat diets with fat content ranging from 20-60% are known to cause obesity and diabetes in a variety of rodent strains, including Wistar and Sprague-Dawley rats **(Bradley *et al*., 2017).** These models help study the pathophysiology of T2D and assess potential treatments **(Chen *et al*., 2024; Cao *et al*., 2024).**

Moringa oleifera, sometimes known as the "wonder tree," "life tree," or "horseradish tree," is one of the 14 species in the Moringaceae family

Fuglie (1999). Is renowned for its numerous nutritional and therapeutic properties **(Himanshu *et al*., 2018).** This plant includes a wide range of bioactive elements, such as important minerals, vitamins, dietary fiber, amino acids, beta-carotene, anti-inflammatory agents, antioxidants, phytochemicals, as well as omega-3 and omega-6 fatty acids **(Amjad *et al*., 2015).** Its high concentration of beneficial minerals and bioactive components has earned it a reputation as a cure-all for various diseases **(Daba, 2016).**

Moringa seed powder has been shown to have strong antidiabetic efficacy in rat models. It has been proven to lower glucose levels, reduce lipid peroxide levels, and lower inflammatory markers including IL-6 and immunoglobulin A when compared to diabetic controls **(Gopalakrisnan *et al*., 2016).** Epidemiological investigations have found insulin-like protein in Moringa which correlate to its hypoglycemic properties **(Vargas-Sanchez *et al*., 2019).** In addition, Moringa leaves have been shown to be useful in diabetes management due to their high antioxidant content and ability to decrease pro-inflammatory mediators **(Bamagous *et al*., 2018).** The underlying mechanisms remain elusive, prompting the current investigation to elucidate the multiple pathways involved in type 2 diabetes.

This study aims to look into how *Moringa oleifera* influences glucose levels and insulin resistance in T2D rat's model. It will also investigate its effects as antioxidant and anti-inflammatory activities related to improve health and reduce complications in type 2 diabetic rats.

Material and Methods

Materials

Moringa (*Moringa oleifera)* leaves powder were got from specific herbal store of "ElkhairWelbaraka", Shebin El-Kom, Menoufia Governorate, Egypt which were packed by Dar altyssir for food and perfumery packaging; and defined by the Agriculture Crops Department, Agriculture Faculty, Menoufia University.

Chemicals

Streptozotocin (STZ) was obtained from SIGMA (USA) to induce diabetic mellitus in rats. El-Gomhoriya Company for Trading Drugs, Chemicals and Medical Instruments, Cairo, Egypt offered casein, vitamin mixture, salt mixture, cellulose, L-Cystine, choline chloride, and methanol. Gamma Trade Company, which is based in Cairo, Egypt, supplied biochemical examination kits.

Experimental Animals

Thirty mature male albino rats of the Sprague-Dawley strain, weighing 150 ± 10 grams, were obtained from the Medical Insects Research Institute in Dokki, Cairo, Egypt.

Methods

Proximate chemical Composition, total Phenolic and Flavonoid Content, and Antioxidant Activity.

Main chemical composition involving moisture, ash, crude fiber, fats, and proteins was detected as **AOAC (2019)** methods. Carbohydrate content was estimated by difference after subtracting moisture, protein, fat, fiber and ash from the sample. Total phenolic content was detected as explained by **Limmongkon *et al.*, (2017)**. Total content of flavonoid was measured by a method developed by **Munhoz *et al*., (2014).** Antioxidant activity was evaluated using the DPPH radical scavenging test **(Gülçin *et al*., 2010).**

Biological Experiment

Ethical Approval

All animal experiments were approved by the Animal Care and Use Committee of the Faculty of Home Economics, Menoufia University, Shebin El-Kom, Egypt (Approval No. [MUFHE /F/ NFS /24/24]).

Preparing the Basel Diet (BD)

The base diet was developed according to the formulation guidelines provided by **AIN (1993).**

Induction of type2 diabetes Mellitus.

Type 2 diabetes was induced in rats via feeding on high fat diet (HFD) consisting of 22% fat, 20% protein, and 48% carbohydrates for four weeks**.** The HFD formulation was prepared according to the protocols described by **Zhang *et al.*, (2008)** and **Srinivasan *et al.*, (2005).** Subsequently, rats received single dose of streptozotocin (STZ) intraperitoneal injection at a dose of 35 mg/kg body weight. Diabetes was confirmed 72 hours later by measuring non-fasting plasma glucose levels. Animals with glucose levels exceeding 300 mg/dl were considered diabetic according to established criteria and used for further experiments.

Experimental animals

The study was conducted at the Faculty of Home Economics, Menoufia University, Shebin El-Kom, Egypt. Rats were housed in cages under standard laboratory conditions with a 12-hour light-dark cycle. All animals underwent a one-week acclimatization period on a baseline diet (BD). Subsequently, 30 rats were randomly divided into 2 main groups: negative control group in which six rats fed the BD and diabetic group in which twenty-four rats fed a high-fat diet (HFD, 22% fat) for four weeks, followed by a single intraperitoneal injection of streptozotocin (STZ) at 35 mg/kg body weight to induce diabetes. The diabetic group was continued on HFD until the end of the experiment and was divided into 4 groups (n=6/group) as following: Group (2): diabetic control, served as a positive control group and fed HFD while Groups 3,4, and 5 were fed HFD supplemented with 2.5%, 5% and 10% *Moringa oleifera* (MO) respectively for 4 weeks. All rats had ad libitum access to water and food throughout the four-week experimental period.

Blood and tissues sampling collection:

At the finish of the four-week treatment period. After a 12-hour fast, blood was obtained via portal vein puncture and was allowed to clot, followed by centrifugation at 3000 rpm for 15 minutes to isolate serum. Serum samples were stored at -20°C for subsequent analysis **(Drury and**

Wallington, 1980). The pancreas tissues were separated and quickly saved on ice for biochemical assays.

Biochemical analysis

Blood Glucose, Serum Insulin Levels, and HOMA-IR Valuation

Serum glucose levels were measured using the method described by **Kaplan (1984).** Serum insulin concentrations were measured according to **Wilson and Miles (1977).** Homeostasis Model Assessment (HOMA-IR) was detected depended on the guidelines of **the Oxford Centre for Diabetes (2009) and Matthews *et al*., (1985).** Insulin sensitivity was assessed with the homeostasis model assessment (HOMA-IR): (fasting glucose [mg/dL]) × [fasting insulin {µU/mL}] ÷ 405) and HOMA-B (fasting plasma insulin [µU/mL] × 360 ÷ [fasting plasma glucose {mg/dL} - 63]).

Serum lipid profile

Triglycerides were quantified using the method described by **Fassati and Prencipe (1982)**. Total cholesterol was detected rendering to **Allain (1974).** HDL (high-density lipoprotein) cholesterol levels were measured using the methods of **Lopez (1977)**. LDL (low-density lipoprotein) plus VLDL (very-low-density lipoprotein) were detected by the next formulas: VLDL-cholesterol = Triglycerides / 5 **(Lee and Nieman, 1996),** The AI (atherogenic index) was calculated as described by **Nakabayashi *et al*., (1995).**

Liver Function Markers

The ALP (alkaline phosphatase) concentration was determined using the **IFCC (1983)** technique. **Yound (1975)** was used to measure ALT (alanine aminotransferase) levels, and **Henry *et al*., (1974)** presented a procedure for determining AST (aspartate aminotransferase) levels.

Kidney Function

The serum creatinine, urea, and uric acid levels were detected by the methods given by **Schirmeister (1964), While *et al*., (1970), and Malhotra (2003),** respectively.

Antioxidant Enzyme Activity and MDA Concentration

The levels of antioxidant enzymes SOD, CAT, and GSH-Px were determined using the modified coupled techniques by **Ohkawa *et al*., (1979), Nishikimi *et al*., (1972), and Paglia and Valentine (1967),** respectively. Malondialdehyde (MDA) was measured using an **Eze *et al*., (2009)** assay. All of these were determined in serum and pancreas tissues .

α-Amylase and α-Glucosidase determination

α-amylase plus α-glucosidase levels were assessed by the methods described by **Ying et al., (1998) and Kim *et al*., (2005),** respectively in serum and pancreas tissues.

Determination of serum protein fractions: total protein, albumin, and globulin

Detected of total protein was done by the procedures explained by **Henry (1964) and Reinhold (1953)**. Albumin levels were tested using the **Doumas *et al*., (1971)** procedure using Boehringer kits. The total globulin fraction was measured using the procedures of **George (2009) and Autenrieth (1917).**

Inflammatory parameters as TNF-α and IL-6 Levels

Tsai *et al*., (2000) and Miller *et al*., (2017) discussed the methods used to test interleukin-6 (IL-6) levels. TNF-α levels were assessed with methods from **Doe *et al*., (2021) and Swaroop *et al*., (2012).**

Sensory Evaluation of Bread and Pizza

Bread and pizza samples were prepared according to **Ihekoronye and Ngoddy (1985)** and **Pacheco de Delahaye *et al*., (2005),** using standardized methods. Twenty staff members from the Department of Nutrition and Food Science, Faculty of Home Economics, Menoufia University, participated in sensory evaluation of bread and pizza. Panelists rated samples based on taste, flavor, color, appearance, texture, compressibility plus overall acceptability. Water was provided for palate cleansing between samples. Bread samples were provided on circular, coded white plates, and pizza was presented similarly. A 1-10 rating system was

employed, with 10 representing "great" and 1 signifying "dislike excessively". The evaluations were conducted two hours after baking. The sensory examination was carried out in a well-lit controlled sensory evaluation laboratory at temperature (25°C).

Statistical Analysis:

Data were examined by one-way ANOVA analysis of variance with SPSS statistical software. Results are given as mean ± SD (standard deviation). Changes among groups were reflected statistically significant at $p< 0.05$ **(SPSS, 1998).**

Results and Discussion

Chemical Composition, Total phenols, Total flavonoids and Antioxidant Activity of Moringa

Table 1 reveals that Moringa contains 6.18% moisture, 5.54% fat, 25.16% protein, 6.32% ash, 56.80% carbohydrates, and 16.74% fiber, indicating its rich content of crude fiber, carbohydrates, and protein. It also has 37.10 mg/kg phenolics, 19.44 mg/kg flavonoids, and 79.67% antioxidant activity. Moringa is nutrient-dense, offering essential amino acids, protein, iron, calcium, vitamin A, and antioxidants **(Azlan *et al.*, 2022; Abd ElHack *et al*., 2018).** Its potent flavonoid antioxidant activity **(Abo-Elsoud *et al.*, 2022)** enhances its nutritional value. Moringa also improves the nutritional profile of cereal products by increasing protein, minerals, and fiber **(Milla *et al.*, 2021).** Moringa oil is rich in monounsaturated fatty acids, especially oleic acid, and contains omega-3, omega-6, tocopherols, calcium, magnesium, phenolics, and flavonoids **(Wu *et al.*, 2020).**

Table (1): The chemical constituents, total phenolic and flavonoid content, and antioxidant capacity of *Moringa oleifera*

Chemical analysis (%)	Moringa powder
Moisture	6.18 ± 0.51
Protein	25.16 ± 2.1
Ash	6.32 ± 0.55
Fat	5.54 ± 0.45
Carbohydrates	٤٠.٠٦ ± ٤.0
Fiber	16.74 ± 1.3
Total phenol (mg gallic acid. g^{-1})	37.10 ± 3.2
Total flavonoids (mg catachin. g^{-1})	19.44 ± 1.5
Antioxidant activity (DPPH %)	79.67 ± 7.1

Each value in the table is the Mean ± Standard Deviation of three replicates.

Recognized for its role in blood glucose homeostasis and diabetes management, Moringa's beneficial effects are attributed to its rich composition of proteins, amino acids, polysaccharides, fatty acids, minerals plus vitamins **(Abdelazim *et al.*, 2024)**. Research has confirmed the significant health benefits of Moringa leaf extract's antioxidant properties **(Khalid *et al.*, 2023).**

Effects of Moringa on Biochemical Parameters Related to type 2 diabetic rats in Serum and Pancreatic Tissue

Table (2) presents the effects of Moringa supplementation on glycemic control, insulin sensitivity, beta-cell function, BWG% and the levels of α-amylase plus α-glucosidase enzymes for the serum and pancreas of diabetic rats, as well as pancreatic protein levels. The data demonstrate that the positive control group had considerably greater levels of glucose, insulin, insulin resistance, and digestive enzyme activities, as well as lower beta-cell function and pancreatic protein levels than the negative control group. Various previous studies as, **De Magalhaes *et al.*, 2019 and Huang *et al.*, 2024** have demonstrated that low-dose streptozotocin (STZ) injections (not less than 25mg/kg b.w., and not more than 45mg/kg b.w.) administered

to high-fat diet (HFD)-fed rats induce severe hyperglycemia, hyperinsulinemia, impaired glucose tolerance, dysfunction of β-cell and insulin resistance, which served as type 2 diabetic rat model. This model considered from the few types 2 diabetic animal models that exactly repeat the natural disease progression and metabolic feature in human. Since previous STZ injection, rats fed HFD is well known to cause obesity, hyperinsulinemia and insulin resistance by impaired insulin receptor signaling in muscle, liver plus adipose tissue then pancreas secret more insulin causing hyperinsulinemia, but may can't elevating fasting glucose level and the level of fat and period of HFD that induced fasting hyperglycemia didn't exactly detected by searches, and this feature is same to compensation stage of type 2 diabetes in human. However, low dose of STZ in HFD-fed rats caused a decompensated diabetes stage with insulin resistance which led to plain hyperglycemia plus ketosis. It's important to notice in this experimental model that the effect of HFD on body weight gain (BWG) was abolished by the low-dose STZ administration, ensuing similar pattern to commonly non-obese type 2 diabetes persons. The observed of total BWG% reduction in this model with long period (more than 16th day after STZ injection) is attributed to muscle atrophy and decreased fat and glycogen deposits due to lack insulin action and insufficient glucose and amino acid availability, then strong lipolysis and proteolysis as described by various searches as **Zeidan *et al*., 2019 and De Magalhaes *et al*., 2019**.

Table (2): Effects of Moringa on Biochemical Parameters Related to type 2 diabetic rats in Serum and Pancreatic Tissue

Groups / Parameters	Control (-)	Control (+)	2.5% M	5% M	10% M
Serum					
Glucose (mg/dl)	102.76^{c}±7.85	240.00^{a}±21.52	137.00^{b}±12.48	125.00^{bc}±11.26	110.00^{c}±9.49
Insulin (ng/ml)	4.75^{b} ± 0.43	7.28^{a} ± 0.73	4.70^{b} ± 0.44	4.50^{b} ± 0.45	4.00^{b} ± 0.36
HOMA-IR	1.20^{b} ±.12	4.30^{a} ±0.43	1.63^{b} ±0.16	1.53^{b} ±0.15	1.30^{b} ±0.12
HOMA-B	44^{a} ±4.05	14.8^{d} ±1.15	22.86^{c}±2.01	26.12^{bc}±2.06	30.6 ±2.34
α-amylase (U/l)	26^{c} ±2.09	62.50^{a}±19.61	44^{b} ±4	33^{bc} ±3.04	24.30^{c} ±1.68
α-glucosidase (ng/mg)	45^{d} ±3.67	98^{a} ±9.01	74.56^{b}±6.43	66^{bc} ±5.66	56.5^{c} ±5.25
BWG%	51^{a} ± 4.58	36^{c} ± 3.32	40.6^{bc}±3.03	45.7^{ab} ± 3.90	49.5^{a} ±4.27
Pancreas					
α-amylase (U/g)	29.00^{c} ± 2.71	60.00^{a} ±4.36	42.00^{b} ±3.58	32.00^{c} ±2.78	26.00^{c} ± 1.99
α-glucosidase (ng/mg)	30.00^{d} ±2.65	72.00^{a} ±6.64	66.00^{a} ± 6.07	51.00^{b} ± 4.52	42.00^{c} ± 3.68
protein (g/100mg)	3.26^{a} ±0.48	1.82^{d} ±0.09	2.16^{cd} ±0.19	2.37^{bc} ±0.16	2.84^{ab} ±0.27

Results are presented as mean ± (SD). Means with dissimilar superscript letters (a, b, c, d, e). Indicate statistically significant differences ($p \leq 0.05$) among treated groups as determined by one-way ANOVA followed by Duncan's multiple range test. Moringa is denoted by "M," while HOMA-IR and HOMA-B represent the Homeostatic Model Assessment of Insulin Resistance and Beta-cell Function, respectively.

In contrast, supplementation with various Moringa doses resulted in significant decreases in glucose, insulin, insulin resistance, and digestive enzymes as α-amylase and α-glucosidase levels, as well as significant increases in beta-cell function, BWG% and pancreatic protein levels compared to the diabetic control group. So Moringa can be used by any these experimental doses as personal acceptation. Moringa treatment resulted in significant improvements in body weight gain because improved glucose utilization and insulin sensitivity leading to enhanced muscle's biosynthetic ability **(Zeidan *et al.*, 2019).** MO polysaccharides improved diabetic rats' glucose levels and metabolism **(Lopez-Rodriguez *et al.*,**

2023). MO improves blood glucose management by many activities as indicated by some searches as modifying Hsp70 (heat shock protein 70) and ILP2 (insulin like peptide 2) and increasing glucose absorption in adipocytes **(Oyeniran *et al*., 2022; Vasanth *et al*., 2022).** Also, MO leaf extract reduces blood hyperglycemia, oxidative stress, and fibrosis by decreasing TGF-β1 (Transforming Growth Factor Beta-1) and type IV collagen gene expression **(Thongrung *et al*., 2023).** The flavonoids and polysaccharides in MO leaves slow glucose transport, digestion, and starch hydrolysis **(Yang *et al*., 2022).** MO's insulin-like proteins may also help to reduce blood glucose levels **(Khan *et al*., 2017).** MO's high fiber content slows stomach emptying **(Ndong *et al*., 2007),** while flavonoids and phenolic components contribute to hypoglycemic effect **(Siahaan *et al*., 2022).** Moringa supplementation has also been linked to decreased insulin resistance and visceral obesity **(Irfan *et al*., 2022).** Niazirin, a Moringa phenolic glycoside, reduces insulin resistance, as demonstrated by a reduced HOMA-IR **(Bao *et al*., 2020).** Other studies also have found that MO leaf extract reduces blood glucose levels and improves insulin sensitivity **(Monraz-M'endez *et al*., 2022; Aljazzaf *et al*., 2023; Melebary and Elnaggar, 2023 and Cortes-Alvarez *et al*., 2024).** Streptozotocin-induced diabetic rats demonstrated improved health outcomes following Moringa seed powder supplementation, characterized by lowered diabetic nephropathy plus restored pancreas and renal tissues architecture. Additionally, Moringa ethyl acetate leaf extract administration for one month ameliorated hyperglycemia and reduced glycosylated hemoglobin (HbA1c) levels in diabetic rats **(Kou *et al*., 2018).**

Obese female Wistar rats supplemented with 600 mg/kg/day of ethanolic Moringa leaf extract for 12 weeks showed improved insulin sensitivity and glycemic control. Moringa leaves enhance insulin sensitivity and excretion, increase glucose uptake in muscles and the liver, inhibit glucose-raising enzymes, and reduce intestinal glucose absorption, helping maintain blood glucose levels **(Ahmad *et al*., 2019).** These effects are attributed to Moringa's antioxidant and antihyperglycemic properties **(Tuorkey, 2016).** Rich in flavonoids and polyphenols, Moringa inhibits

starch-hydrolyzing enzymes, contributing to its hypoglycemic effects **(Adisakwattana et al., 2011).** Moringa's ethanol extracts also inhibit α-amylase and α-glucosidase, reducing glucose absorption **(Shaikh *et al.*, 2020).** Its anti-diabetic effects are linked to delayed gastric emptying and enzyme inhibition **(Alegbeleye, 2022),** aligning with its hypoglycemic properties in type 2 diabetes **(Jaiswal *et al*., 2013).**

Moreover, Moringa's effect on pancreatic protein clearly indicates to preventing protein from oxidative damage which come in agree with **Gupta *et al.*, (2012)** who discussed that free radicals which contribute to diabetes development and its complications are well-known to abnormally change proteins and produced proteins carbonyl product that a marker of proteins oxidative damage.

It could be concluded from results of this study and previous investigated findings that the Moringa has hypoglycemic effects. Moringa's mechanisms to exhibit glycemic control may come from different actions as influence some metabolic ways, slow gastric emptying, slow glucose transport, digestion, and starch hydrolysis as inhibition enzymes of carbohydrate-digesting, including α-amylase plus α-glucosidase, hence reducing glucose absorption, decreased insulin resistance and enhancing insulin sensitivity which increasing glucose uptake by muscles and the liver and MO's insulin-like protein/peptide may also help which mimetic insulin effect and can become innovative medicine. Moringa can be useful for obese and non-obese diabetic patients. Anywise, additional studies are needed to employ on variety of experimental models and human to prove and detect the perfect and safe dose and period toward these respects.

Effects of Moringa on α -TNF and IL-6 as inflammatory parameters in type 2 diabetic rats

Table (3) shows the benefits of adding Moringa at 2.5%, 5%, and 10% to the diets of rats with type 2 diabetes. In comparison to the diabetic group, adding Moringa resulted in significant reductions in TNF-alpha and IL-6 (interleukin-6) levels in both serum and pancreas at all concentrations. In contrast, the diabetic group had considerably higher levels of these

cytokines than the negative control group, indicating increased inflammation caused by raised blood glucose levels. Under diabetes disease condition of high glucose, fatty acids accumulation and ROS (reactive oxygen species), inflammatory pathways enhance involving inflammatory injury to many tissues like liver as found in HFD-STZ caused type 2 diabetic mice model by **Leu *et al*., (2024)**. According to searches as **Safhi *et al*., (2018)** and **Zeidan *et al*., (2019)**, inflammatory pathways in diabetes result in elevated levels of TNF-α, IL-6 and IL-1β that are stimulating insulin resistance, oxidative stress and development of diabetes and its complication as kidney injury. Also, **Huang *et al*., (2024)** revealed that type 2 diabetes is known as chronic inflammatory illness that considered plus an unhealthy feeding one of its caused factors.

The study conducted by **Safhi *et al*., (2018)** indicated that daily injections of *M. oleifera* extract significantly reduced these cytokines, highlighting its anti-inflammatory properties. *Moringa oleifera* (MO) has demonstrated anti-inflammatory properties in various models. It has been shown to reduce pro-inflammatory cytokines, including IL-6, TNF-α, IL-1β, and IL-12, in models of diabetic and chemically induced liver and kidney damage (**Abdel Fattah *et al*., 2020; Abd-Elhakim *et al*., 2021; Abou-Zeid *et al*., 2021).** Additionally, MO has been found to inhibit macrophage-derived cytokines IL-6, TNF-α, and IL-8 **(Jaja-Chimedza *et al*., 2017).** Further studies have revealed that MO can lower the levels of IL-6, TNF-α, IL-1β, and IFN-β, while also inhibiting nuclear factor-κB (NF-κB) activation and reducing autophagy **(Xiong *et al*., 2021)**. In the context of colorectal cancer, MO leaf extracts have been shown to decrease inflammation by reducing TNF-α, IL-2, and IL-6 levels, and by improving overall inflammatory cytokine profiles **(Cuellar-Nunez *et al*., 2021).**

Table (3): Effects of Moringa on α -TNF and IL-6 as inflammatory parameters in type 2 diabetic rats

Parameters	Serum		Pancreas	
Groups	TNF-α (pg/ml)	IL-6 pg/ml	TNF-α (pg/mg)	IL-6(pg/mg)
Control (-)	$47.99^{c} \pm 3.97$	$75.00^{d} \pm 6.45$	$35.00^{d} \pm 3.36$	$68.00^{d} \pm 6.62$
Control (+)	$126.00^{a} \pm 11.39$	$187.00^{a} \pm 16.52$	$150.00^{a} \pm 13.53$	$230.00^{a} \pm 21.52$
M (2.5%)	$101.00^{b} \pm 7.54$	$156.00^{b} \pm 13.52$	$120.00^{b} \pm 10.15$	$180.00^{v} \pm 15.53$
M (5%)	$88.00^{b} \pm 7.20$	$118.00^{c} \pm 10.14$	$100.00^{c} \pm 8.19$	$126.00^{c} \pm 11.7$
M (10%)	$53.33^{c} \pm 6.02$	$81.00^{d} \pm 5.56$	$50.00^{d} \pm 3.61$	$78.00^{d} \pm 6.80$

Reaults are presented as mean ± (SD). Values with dissimilar superscript letters (a, b, c, d, e). Indicate statistically significant changes ($p < 0.05$) among treatment groups as determined by one-way ANOVA followed by Duncan's multiple range test. Moringa, TNF-α, and IL-6 represent Moringa oleifera, tumor necrosis factor-alpha, and interleukin-6, respectively.

Effects of Moringa on antioxidant parameters in type 2 diabetic rats

Table (4) summarizes the effects of Moringa supplementation on antioxidant enzyme activities and lipid peroxidation in the serum and pancreas of type 2 diabetic rats. Constricted to control group, diabetes significantly increased MDA (malondialdehyde) level while decreasing activities of SOD (superoxide dismutase), catalase (CAT), and GPX (glutathione peroxidase). The same results of reduction antioxidant enzymes and increased MDA in both serum and liver tissues of HFD-STZ induced type 2 diabetic mice model were reported by **Leu *et al.*, (2024)** who discussed that the antioxidant enzymes of body (SOD, CAT plus GSH-Px) consider a marker for oxidative stress. In which SOD found in organisms to modify the conversion of superoxide anion radical to produce H_2O_2 and O_2; also, CAT and GSH-Px act a detoxification role by adjust toxic peroxides into non-toxic ones and preventing cells of organs from peroxidase injury. Metabolic dysregulation often escorted type 2 diabetes via insulin resistance which resulting in reduce glucose uptake then raise blood glucose, insulin and fatty acids and these metabolic dysregulations can aggravate the (ROS)

reactive oxygen species accumulation in cells. ROS react with body macromolecules and generate terminal products of lipid oxidation as MDA, which can cause organ injury indirectly. Hyperglycemia-caused oxidative stress is concerned in the beginning and development of diabetes plus, it can result in danger complications if remain untreated **(Omodanisi *et al*., 2017).**

Moringa supplementation effectively reversed these effects by enhancing antioxidant enzyme activities and reducing MDA levels in both serum and pancreas tissues. These findings suggest that Moringa supplementation improves the cellular antioxidant defense system and attenuates oxidative stress. Notably, Moringa polysaccharides have also been reported to increase CAT, SOD, and GPX activities while decreasing MDA and reactive oxygen species (ROS) **(Gu *et al*., 2022).** Prior studies have similarly stated the ability of Moringa leaf extract to enhance intracellular antioxidant defenses and reduce oxidative damage **(Mthiyane *et al*., 2022; Tian *et al*., 2021)**. Moringa's potential to increase SOD levels in diabetic patients is due to its active components, which include phenols and terpenoids **(Vergara-Jimenez *et al*., 2017).** The extract may also help birds cope with heat stress by modifying the antioxidant system, resulting in better antioxidant profiles and lower lipid peroxidation indicators in pigeons exposed to high temperatures **(Jimoh *et al*., 2022).** Moringa extract can also protect the kidneys by increasing antioxidant enzyme activity (SOD, CAT, and GSH) and lowering pro-inflammatory cytokines **(Aju *et al*., 2019).** Moringa has been shown in studies to reduce MDA production, which has been linked to a decrease in ROS **(Lukiswanto *et al*., 2022; Ebrahem *et al*., 2022; Cortes-Alvarez *et al*., 2024),** Because of its high polyphenol content, it exhibits strong anti-free radical action and protects against oxidative damage **(Elgamily *et al*., 2016).** Pre-administration of Moringa hydro-ethanol extract provided effective hepatotoxicity protection, with regulated lipid peroxidation, GST, GPX, and GR levels **(Giaccoppo *et al*., 2015).** Furthermore, Moringa can lower MDA levels and acetylcholinesterase (AChE) activity while increasing SOD and catalase activities **(Kou *et al*., 2018).** So, it could be concluded that Moringa improves the cellular antioxidant defense system, attenuates oxidative

stress, and has anti-inflammatory properties which prevent development of diabetes and its complications.

Table (4): Effect of Moringa on Antioxidant Status and Malondialdehyde as Indicators of Lipid Peroxidation

parameters	Serum				Pancreas			
Groups	MDA (nmol/ml)	SOD (U/ml)	GP (U/ml)	CAT (ng/ml)	MDA (nmol/mg)	SOD (U/mg)	GPX (ng/mg)	CAT (ng/mg)
Control (-)	2.60^{b}±0.24	143.50^{a}±11.71	136.5^{a}± 11.71	5.09^{b}±0.49	0.59^{d}±0.05	117^{a}±10.05	178^{a}±15.95	6.81^{a}±0.69
Control (+)	8.20^{a}±0.73	81.50^{c} ± 7.45	99.00^{b} ± 7.49	2.30^{d}±0.23	10.07^{a}±0.99	37^{c}±3.64	40^{e} ± 2.65	1.25^{e}±0.13
M (2.5%)	4.30^{b}±0.36	112.5^{b} ± 9.77	121.50^{a}±10.82	3.65^{c}±0.36	7.34^{b}±0.74	85^{8}±8.85	71^{d} ± 5.57	2.66^{d}±0.27
M (5%)	2.48^{b}±0.20	135.00^{a}±11.53	132.00^{a}±10.54	5.28^{b}±0.52	2.13^{c}±0.21	100^{b}±8.19	105^{c} ± 9.85	3.50^{c}±0.35
M (10%)	3.54^{b}±2.99	147.5^{a} ± 13.39	140.00^{a}±11.53	6.65^{a}±0.60	0.74^{d}±0.11	120^{a}±10.15	155^{b}±13.11	4.87^{b}±0.49

Data are presented as mean ± (SD). Significant differences among treatment groups ($p \leq 0.05$) as determined by one-way ANOVA followed by Duncan's multiple range test. Dissimilar lowercase letters (a, b, c, d, e). Indicate significant differences between group means. Moringa is abbreviated as M, while MDA, SOD, GPX, and CAT represent malondialdehyde, superoxide dismutase, glutathione peroxidase, and catalase, respectively.

Effects of Moringa on serum lipids profile in type 2 diabetic rats

Table (5) presents the impacts of Moringa supplementation on lipids profile in type 2 diabetic rats. Diabetic rats exhibited significantly increased levels of triglycerides (TG), TC (total cholesterol), LDL (low density lipoprotein), VLDL (very low-density lipoprotein), atherogenic index (AI), and decreased HDL (high-density lipoprotein) contrasted to healthy controls. Many searches as **Pratiwi *et al.*, (2021)** revealed that the type 2 diabetic rats model by (HFD) high-fat diet plus (STZ)streptozotocin injection which is popular due to its simple, low cost and similar to diabetic pathogenesis in human showed changes in lipids profile by elevated TG, TC and LDL plus declined HDL which considered from important diabetic complications. And **Zeidan *et al.*, (2019)** discussed that indicated

dyslipidemia caused by excessive lipolysis via raised blood lipids with abnormally metabolism.

Moringa leaves' nutrient-rich composition, including vitamins, minerals, and amino acids, supports its hypolipidemic effects **(Olayaki *et al.*, 2015).** MO leaf extracts inhibit pancreatic lipase and α-glucosidase, lowering TC, HDL-C, and LDL cholesterol **(Chen *et al.*, 2020).** Similar effects were seen when MO was added to ice cream and fed to rats **(Ademosun *et al.*, 2022).** MO improves metabolic conditions in obese mice and protects against hypertriglyceridemia and hypercholesterolemia **(Zunica *et al.*, 2021; Watanabe *et al.*, 2021).** Fermented MO leaf extracts reduced hepatic fat production and improved lipid profiles and adipokines **(Awad *et al.*, 2021)**. MO leaf extract also resisted metabolic syndrome and inhibited fat absorption **(Alkhudhayri *et al.*, 2021).** Studies show significant reductions in triglycerides, LDL-C, and cholesterol, with improved HDL-C levels **(Monraz-M'endez *et al.*, 2022; Aljazzaf *et al.*, 2023; Cortes-Alvarez *et al.*, 2024).** MO's phenolic components, including flavonoids, and plant sterols help block fat absorption, inhibit cholesterol reabsorption, and enhance cholesterol excretion, lowering lipid levels **(Melebary and Elnaggar, 2023).**

Table (5): Effects of Moringa on serum lipids profile in type 2 diabetic rats

Parameters Groups	TG (mg/dl)	TC (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)	AI
Control (-)	70.29^{d}±5.67	86.30^{c}± 7.50	46.09^{ab} ± 4	25.88^{d}±2.79	14.06^{d}±1.20	0.87^{d}±0.087
Control (+)	164.20^{a}±14.58	175.50^{a}±16.40	40.56^{b}±3.46	102.10^{a}±8.90	32.84^{a}±2.91	3.33^{a} ± 0.34
M (2.5%)	107.45^{b}±9.07	132.90^{b}±12.85	44.82^{ab}±3.45	66.59^{b}± 6.01	21.49^{b}±1.57	1.97^{b} ± 0.19
M (5%)	89.22^{c} ± 7.76	113.71^{b}±10.57	48.32^{a}± 4.55	47.55^{c} ± 3.64	17.84^{c}±1.59	1.35^{c} ± 0.13
M (10%)	73.64^{cd} ± 6.20	91.62^{c} ± 8.50	48.07^{ab}±3.90	28.82^{d}± 1.96	14.73^{cd}±1.23	0.91^{d} ± 0.08

Results are given as mean ± (SD). Significant changes ($p < 0.05$) between treated groups as determined by one-way ANOVA followed by Duncan's multiple range test. Change lowercase letters (a, b, c, d, e). Indicate significant changes between group means. M represents Moringa. TC, LDL-C, VLDL-C, HDL-C, TG and AI denote total cholesterol, low-

density lipoprotein cholesterol, very low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, total triglycerides, and atherogenic index (TC/HDL-C), respectively.

Effects of Moringa on serum levels of liver enzymes and protein fraction in type 2 diabetic rats

Table (6) presents the effects of Moringa supplementation on liver enzyme profiles and protein parameters in type 2 diabetic rats**.** Compared to the healthy control group, diabetic rats exhibited significantly elevated levels of AST (aspartate transaminase), ALT (alanine transaminase), ALP (alkaline phosphatase), and globulin, while albumin, total protein, and A/G ratio (albumin-to-globulin) were significantly lowered. Albumin levels are a critical indicator of liver function, and their decline is often associated with hepatic dysfunction **(Guria *et al*., 2014)**. Also, elevation of liver enzymes in HFD-STZ caused type 2 diabetic mice model reported by **Leu *et al*., (2024)** who discussed that (NAFLD) Non-alcoholic fatty liver disease signifies a harsh type 2 diabetes complication. The liver acts a fundamental role to regulate levels of glucose and maintain lipid homeostasis. Metabolic dysregulation often escorted type 2 diabetes, resulting in hepatic steatosis and development of many liver pathologies, in which insulin resistance in type 2 diabetes reduce glucose uptake then raise insulin and lipids causing hyperglycemia plus hypertriglyceridemia and extreme accumulation of fat lead to liver injury and NAFLD. Also, these metabolic dysregulations can aggravate the (ROS) reactive oxygen species accumulation in cells. ROS react with body macromolecules and generate terminal products of lipid oxidation as MDA, which can cause organ injury indirectly. So hypoglycemic, hypolipidemic and antioxidant effects are an important reason for hepatoprotective action.

Moringa supplementation significantly reduced the levels of these liver enzymes and restored protein parameters toward normal values. These findings align with previous research demonstrating the hepatoprotective effects of Moringa in reducing elevated serum AST and ALT levels in diabetic rats (**Omodanisi *et al*., 2017)**. The hepatoprotective properties of

Moringa leaves extract, which contains quercetin and kaempferol, account for the reduction in ALT, AST, and ALP levels in Moringa-treated mice **(Toppo *et al.*, 2015).** Moringa leaves had a substantial influence on ALP, AST, and ALT levels, lipid reduction, and lipid peroxidation in rat liver, resulting in decrease plasma AST, ALT, ALP, plus creatinine levels, as reduced drug-caused hepatic and renal damage **(Miltonprabu *et al.*, 2017)**. Moringa seed extract also helped regulate amino transferase activity and globulin levels in rats, lowering liver damage and fibrosis **(Hamza, 2010).**

Moringa oleifera has demonstrated hepatoprotective effects in multiple animal studies. **Liu *et al.*, (2022)** attributed Moringa's liver protection to its bioactive compounds and underlying mechanisms**. Ahmad *et al.*, (2020)** reported that Moringa leaf extract prevented liver damage in high-fat diet-fed rats by reducing lipid peroxidation (LPO), enhancing glutathione (GSH) levels, and decreasing liver enzyme activities (AST, ALP, ALT). Moringa supplementation enhanced hepatic amino acid synthesis, helping to heal liver inflammation. Moringa extract includes phenolic compounds with strong antioxidant and redox properties, which neutralize free radicals and reduce oxidative stress **(Adewusi and Afolayan, 2010).**

Similarly, **Hussein *et al.*, (2019)** demonstrated Moringa's hepatoprotective properties and ability to raise serum albumin and total protein levels. Also, Moringa supplementation (200 mg/kg/BW) significantly increased antioxidant enzyme concentrations and serum proteins after 8 weeks **(El-bakry *et al.*, 2016).** In addition to **Mousa *et al.*, (2017)** who reported a considerable raise in total protein levels after utilizing *Moringa oleifera*. **Omodanisi *et al.*, (2017a)** discovered that methanolic Moringa leaf extract (250 mg/kg body weight) significantly elevated albumin and total protein levels in diabetic rats. **Gupta *et al.*, (2012)** found equivalent results, with significant increases in these parameters after Moringa treatment. These findings are consistent with **Hamza (2010)** report that Moringa seed extract reversed CCl_4-induced blood albumin declines while increasing serum globulin levels. **Sadek *et al.*, (2017)** discovered that ethanolic Moringa leaf extract (500 mg/kg b.wt)

alleviated the reduction in total protein and albumin in (diethylenitrosamine) DEN-intoxicated rats, and **Gayathri *et al.*, (2020)** found that Moringa leaf meal supplementation increased blood protein and albumin levels.

Table (6): Effects of Moringa on serum levels of liver enzymes and protein fraction in type 2 diabetic rats

Parameters Groups	ALT (U/L)	AST (U/L)	ALP (U/L)	Total protein (g/dl)	Albumin (A) (g/dl)	Globulin (G) (g/dl)	A/G Ratio
Control (-)	58.23^{c}±3.65	80.32^{d} ± 6.27	83.62^{c} ± 7.85	7.50^{a}± 0.68	4.63^{a}± 0.49	2.87^{bc}±0.29	1.61^{a}±0.14
Control(+)	100.42^{a}±7.7	144.90^{a}±13.86	169.16^{a}±13.97	4.90^{d}± 0.29	1.26^{d}± 0.12	3.64^{a}± 0.37	0.34^{c}±0.04
M (2.5%)	82.40^{b}±9.94	131.10^{ab}±10.99	144.44^{b}± 9.40	5.50^{cd}±0.50	2.40^{c}± 0.19	3.10^{b}± 0.18	0.77^{b}±0.08
M (5%)	71.17^{bc}±7.56	122.20^{b} ± 9.99	92.99^{c} ± 7.62	6.40^{bc}±0.61	3.80^{b}± 0.49	2.60^{c}± 0.15	1.46^{a}±0.14
M (10%)	67.94^{c}± 4.48	100^{c} ± 7.61	87.40^{c} ± 6.29	7.10^{ab}±0.27	4.30^{ab}±0.30	2.80^{bc}±0.19	1.54^{a}±0.16

Results are given as mean ± (SD). Statistical significance among treated groups as determined by one-way ANOVA followed by Duncan's multiple range test. Means with different lowercase letters (a, b, c, d, e). Indicate significant differences ($p < 0.05$). Moringa is abbreviated as M; ALT, alanine aminotransferase; AST, aspartate aminotransferase; and ALP, alkaline phosphatase.

Effects of Moringa on kidney function in type 2 diabetic rats

Table (7) presents the impacts of Moringa supplementation on renal function in type 2 diabetic rats. Contrasted to control group, diabetic rats exhibited significantly increased levels of urea, creatinine, and uric acid, indicative of renal dysfunction. Serum creatinine and urea levels, as established by **Kakalij *et al.*, (2014)**, serve as reliable biomarkers for nephrotoxicity. The HFD-STZ induced type 2 diabetic rats showed insulin resistance, hyperglycemia, hyperinsulinemia, hyperlipidemia, proteinuria plus hypertension, that significantly developed diabetic nephropathy more than type 1 diabetic rats as found by **Danda *et al.*, (2015)** and revealed that various animal experiments and human studies showed a direct relation between adiposity, insulin resistance plus hypertension and renal disease; also lipids act a valuable role in renal injury pathogenesis which may be by

raise glomerular macrophages, chemical mediators and LDL-contingent oxidative stress that analogous atherosclerosis plus hypertension helps also in this pathogenesis, but exact mechanism remain unclear. Also, **Kundu *et al.*, (2022)** supported these findings since HFD caused diabetic nephropathy.

Moringa treatment groups demonstrated significant improvements in renal function, as evidenced by reduced creatinine and urea levels, suggesting protection against diabetic nephropathy. The reduced levels of creatinine, uric acid and urea observed in groups treated with *Moringa oleifera* (MO) seed extracts suggest its protective effects against diabetic nephropathy **(Tian *et al.*, 2021)**. **Nabila *et al.*, (2017)** discovered that Moringa therapy effectively reduced serum urea and creatinine. Diabetic nephropathy has been associated to increased blood urea and creatinine levels in rats. Moringa leaf supplementation lowered blood uric acid and creatinine levels **(Abdel-Wareth and Lohakare, 2021),** and restored uric acid levels in diabetic rats **(Elhamalawy *et al.*, 2022).** Eight weeks of co-supplementation with Moringa seed powder significantly decreased serum uric acid and creatinine levels, corroborating the findings of **Al-Malki and El-Rabey (2015).** Further evidence supports MO as a beneficial remedy for kidney disorders **(Saleh, 2019),** with MO effectively reducing diabetic nephropathy in alloxan-treated rats. Similarly, **Kagbo and Abaekwume (2021)** reported that Moringa mitigated hepato-renal damage caused by acetaminophen-induced hyperglycemia. According to **Akter *et al.*, (2021),** the reno-protective effects of Moringa are thought to stem from its anti-inflammatory and antioxidant activities, which may help alleviate oxidative stress and inflammation, both of which are significant contributors to the progression of kidney disease. Methanolic Moringa extracts have also demonstrated efficacy in reducing urea and creatinine levels in kidney ischemia models **(Stephen *et al.*, 2020).** Further support for these findings was provided by **Arafat *et al.*, (2018),** who demonstrated that Moringa has the capability to reduce oxidative stress and tissue damage in both the liver and kidneys.

The reno-protective effects of Moringa are further supported by several studies. **Karthivashan *et al.*, (2016)** highlighted the role of

Moringa's mineral, flavonoid, and phenolic compounds in enhancing kidney protection. **Ahmed *et al.*, (2020)** demonstrated Moringa's efficacy in reducing blood creatinine and BUN levels in patients with chronic renal disease. **Tang *et al.*, (2017)** linked Moringa consumption to decreased LDL, oxidative stress, and atherosclerosis, particularly in kidney infection models. These results align with **Ouédraogo *et al.*, (2011)**, who arrived that Moringa, when co-administered with gentamicin, attenuated renal dysfunction by reducing serum creatinine and urea levels.

Table (7): Effects of Moringa on kidney function in type 2 diabetic rats

Parameters Groups	Serum uric acid (mg/dl)	Serum creatinine (mg/dl)	Serum urea (mg/dl)
Control (-)	$2.50^{d} \pm 0.24$	$0.48^{d} \pm 0.05$	$23.56^{d} \pm 2.12$
Control (+)	$4.53^{a} \pm 0.43$	$1.07^{b} \pm 0.10$	$76.06^{a} \pm 6.39$
M (2.5%)	$3.70^{b} \pm 0.34$	$0.81^{c} \pm 0.08$	$63.40^{b} \pm 4.81$
M (5%)	$3.24^{bc} \pm 0.26$	$0.72^{c} \pm 0.07$	$56.50^{b} \pm 5.14$
M (10%)	$2.70^{cd} \pm 0.23$	$0.57^{d} \pm 0.06$	$31.97^{c} \pm 3.26$

Results are given as mean ± (SD). Statistically significant changes ($p \leq 0.05$) between treated groups as determined by one-way ANOVA followed by Duncan's multiple range test. Dissimilar lowercase letters (a, b, c, d, e). Within each row indicate significant changes between group means. M represents Moringa.

Sensory properties of bread and pizza made with 2.5%, 5%, and 10% Moringa powder in place of wheat flour

Table (8) displays the mean sensory quality of bread and pizza samples made with wheat flour that was partially replaced by 2.5 %, 5% and 10% Moringa powder, as evaluated by twenty staff members. The findings indicated that there are no substantial variances in taste, flexibility, or flavor between the control and 2.5% Moringa powder samples for bread. However, there were notable variances in look, color, texture, and overall acceptability. Bread samples containing 5% and 10% Moringa powder shown substantial variations in all sensory qualities except elasticity. For pizza, significant variations were identified at 5% and 10% Moringa powder

doses, but no significant changes were obtained among control and 2.5% Moringa pizza samples in any sensory attribute. Lower concentrations yielded better results, particularly for bread, but higher concentrations imparted a green color that some consumers found unacceptable. This variability in consumer preference highlights that acceptance can differ based on individual differences. Overall, all Moringa concentrations provided biological advantages, so Moringa can be used by any level as individual acceptance.

Moringa oleifera is used in India to enhance energy bars and oil cakes **(Ijarotimi *et al*., 2013).** Fortification aims to improve food products by adding important nutrients while retaining sensory attributes **(Allen *et al*., 2006). Kalowole *et al*., (2013)** found that adding up to 8% Moringa leaf powder to wheat bread preserves sensory properties. Moringa leaf powder, on the other hand, is less favorable compared to seeds or flowers **(Karim *et al*., 2015).**

In Africa, "Amala" cake benefits from the inclusion of 10% Moringa leaf powder, which boosts protein content by around 48% while also enhancing calcium, potassium, magnesium, salt, and iron. This addition had no discernible impact on the cake's sensory properties **(Abioye and Mo, 2015).** Incorporating Moringa powder from seeds, flowers, or leaves into bread recipes boosts protein and fiber content while also influencing sensory characteristics like color and aroma. To counteract the herbal flavor, a taste-masking chemical could be used **(Chinma *et al*., 2014).**

According to **Bolarinwa *et al*., (2017)**, it was noted that key sensory attributes did not show significant changes between bread enriched with 5% Moringa and traditional wheat flour bread, implying that consumer acceptance of both types was comparable. The study also found that sensory evaluations of bread containing 5% Moringa seed powder were similar to those of wheat flour bread in terms of firmness and texture, indicating that Moringa can enhance the strength and extend the shelf life of wheat bread. Additionally, the elasticity of bread, which measures height recovery between compression cycles, showed no significant change in storage days

for bread with 1.25% Moringa. While **Hayat *et al*., (2018)** found control bread to be superior in terms of flavor, smell, and appearance, the 2.5% Moringa leaf powder bread emerged as a palatable gluten-free alternative. These findings align with **Ighabul *et al*., (2018),** who demonstrated the potential of Moringa leaf for producing nutritious and appealing cookies**. Sengev *et al*., (2013)** found that increasing the amount of Moringa powder affected the acceptable crumb texture. It was indicated by **Dachana *et al*., (2010)** that the addition of dried Moringa leaves to cookie dough led to an increase in dough hardness, alongside a reduction in cohesiveness and spreadability. **Abioye and Mo (2015)** observed that lower viscosities in formulations containing Moringa leaf powder influenced the resulting texture. **Ogunsina *et al*., (2011)** found that adding Moringa powder to bread increased its stiffness. These findings align with the growing body of research exploring the usage of Moringa leaves and seed powders to enhance the nutritional value of various food products, including weaning cereals, biscuits, and bread **(Bolarinwa *et al*., 2017).**

Agreeing to **Abraham *et al*., (2013) and Nagib (2014),** increasing the dose of Moringa leaf powder reduces the acceptability of pizza. Consumer adoption of Moringa-fortified bread may differ by area. Other studies have found that the black color and bitter taste of Moringa powder reduce customer adoption **(Karim *et al*., 2015).** These studies, however, focuses on customer acceptance in South Africa, where the dim color of Moringa may be less troublesome in brown bread than in white bread. Previous research **Obichili and Ifediba,** (**2019)** demonstrated that adding Moringa powder colored bread, particularly white bread, due to Moringa's high chlorophyll content**. Bourekoua *et al*., (2018)** found that higher Moringa concentrations reduced the nimbleness of bread crumb and coating. Although dark colors may influence consumer approval, some people may be willing to tolerate them for health reasons. Adding a lightening agent may boost the appeal of Moringa-fortified bread **(Sengev *et al*., 2013).**

Moringa oleifera can increase the nutritional value of dairy products like yogurt and cheese. Adding 0.5% Moringa leaf extract to yogurt preserves sensory satisfaction, whereas fortifying cheese with Moringa leaf

powder increases protein, carbohydrate, and lipid content. Some consumers may tolerate higher doses despite the herbal flavor **(Lokapirnasari *et al*., 2021)**. Moringa leaves are also used in soup, beverages, and salad, but research on these applications is ongoing, and sensory and nutritional properties have yet to be fully predicted **(Stevens *et al*., 2013; Babayeju *et al*., 2014).**

It could be concluded that Moringa is a versatile ingredient that can be added as it is to a variety of foods, such as yogurt, cheese, or even water. However, these options may not always be well-received by everyone. An exciting alternative is to incorporate Moringa into baked goods like bread or pizza, which could offer a more palatable way to enjoy its benefits. Also, adding Moringa to cooked vegetables as malukhia and spinach etc. may be good suggest. Moving forward, it's crucial to explore how Moringa behaves when subjected to heat. It is essential for specifically future studies to examine how heating affects Moringa's nutritional and therapeutic properties. Research should determine whether Moringa retains its full efficacy after cooking or if its effectiveness is compromised, which could require adjusting the dosage to achieve the desired health benefits.

The control group consisted of wheat flour, whereas the Moringa-supplemented groups included 2.5%, 5%, and 10% Moringa, respectively, by substituting various amounts of wheat flour.

Table (8). Sensory examination of bread and pizza made with varying amounts of Moringa instead of wheat flour

	Bread				Pizza			
Parameter	**Control**	**2.5% M**	**5% M**	**10% M**	**Control**	**2.5% M**	**5% M**	**10% M**
Appearance	9.9^{a}±0.316	9.1^{b}±0.738	8^{c} ± 0.943	7.1^{d}±1.1005	10^{a} ± 0.00	9.6^{a}±0.516	8.7^{b} ± 0.483	8^{c} ± 0.943
Color	9.9^{a}±0.316	8.9^{b}± 0.994	7.9^{c}±1.1005	6.7^{d} ± 0.823	10^{a} ± 0.00	9.9^{a}±0.316	8.5^{b} ± 0.707	7.5^{c}±1.080
Taste	9.8^{a}±0.422	9.4^{a}± 0.843	8.6^{b} ± 0.966	7.6^{c} ± 0.699	10^{a} ± 0.00	9.7^{a}±0.675	8.2^{b} ± 1.033	6.8^{c}±0.632
Compressibility	9.1^{a}±1.1005	9^{a} ± 0.943	8.5^{a} ± 1.269	8.4^{a} ± 1.174	9.9^{a}±0.316	9.5^{a}±0.8498	8.3^{b}±0.9486	7.9^{b}±1.197
Flavor	9.7^{a}±0.675	9.4^{a}± 0.699	8.4^{b} ± 1.075	7.1^{c} ± 0.738	9.9^{a}±0.316	9.6^{a}±0.699	8.4^{b}±0.843	6.9^{c}±0.7378
Texture	9.6^{a}±0.516	9.1^{ab}±0.738	9^{ab} ± 0.817	8.5^{b}±0.8498	9.9^{a}±0.316	9.8^{a}±0.632	9^{b}±0.8165	7.7^{c}±1.1595
Overall Acceptability	9.9^{a}±0.316	9.4^{ab}±0.699	8.9^{b} ± 0.876	7.9^{c} ± 0.738	10^{a}±0.00	9.7^{a}±0.483	8.6^{b}±0.699	7.4^{c}±0.516

Results are given as mean ± (SD). Statistically significant changes ($p \leq 0.05$) between treated groups as determined by one-way ANOVA followed by Duncan's multiple range test. Dissimilar lowercase letters (a, b, c, d, e). Within each row indicate significant changes between treatment means. M represents Moringa.

Conclusion

In conclusion, this study and previous findings show that Moringa has natural hypoglycemic effects beneficial for both obese and non-obese diabetic patients. Moringa works through various mechanisms, including slowing gastric emptying, glucose transport, digestion, and inhibiting carbohydrate-digesting enzymes like α-amylase and α-glucosidase. These actions reduce glucose absorption, decrease insulin resistance, and improve insulin sensitivity. Moringa's insulin-like protein may also mimic insulin's effects. Its antioxidant and anti-inflammatory properties help manage diabetes-related complications like renal and liver dysfunction. However, further studies are needed to determine the optimal dosage and duration for human use. Additionally, Moringa is a versatile ingredient with high nutritional value that can be added to foods like yogurt, cheese, bread, and pizza. Although some may not prefer its taste, supplementing baked or

cooked foods such as bread, pizza, or vegetables like malukhia can improve acceptability. Lower supplementation levels maintain good sensory qualities, but higher doses enhance biological effects. Future studies should explore how heating affects Moringa's nutritional and therapeutic properties to determine the ideal doses for achieving health benefits.

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الآليات المحتملة لنبات المورينجا في ضبط مستويات السكر المرتفعة لدى الفئران المصابة بالسكرى من النوع الثاني الناجم عن غذاء عالي الدهون مع جرعة منخفضة من الستربتوزوتوسين

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الملخص العربي:

من المتوقع أن يكون مرض السكري السبب السابع للوفاة عالميًا بحلول عام ٢٠٣٠، وهو عامل خطر معروف لزيادة الإصابة بمضاعفات فيروسCOVID-19 . بينما أظهرت شجرة المورينجا (Moringa oleifera)تطورًا في التحكم فى مرض السكري من النوع الأول، وقد تحتاج تأثيراتها على مرض السكري من النوع الثاني إلى مزيد من الدراسات. تهدف هذه الدراسة إلى تقييم قدرة المورينجا على تقليل مستويات الجلوكوز في الفئران المصابة بمرض السكري من النوع الثاني من خلال تحسين حساسية الإنسولين وتثبيط إنزيمات الهضم مثل-α أميلاز و-αغلوكوسيداز، بالإضافة إلى أنشطتها المضادة للالتهابات والأكسدة. تم تحفيز مرض السكري من النوع الثاني في الفئران باستخدام غذاء غني بالدهون (٢٢٪ دهون) لمدة ٤ أسابيع، ثم تم إعطاؤها جرعة منخفضة واحدة من ستربتوزوتوسين (٣٥ ملغ/كغ من وزن الجسم)، ثم قسمت إلى مجموعات ضابطة ومجموعات مصابة بالسكري، مع إضافة جرعات متفاوتة من المورينجا (٢,٥٪، ٥٪، ١٠٪) إلى غذاء الفئران المصابة بالسكري لمدة ٤ أسابيع. أظهرت النتائج أن المورينجا تحتوي على مستويات كبيرة من الألياف والبروتينات والمواد الفينولية والفلافونويد، بالإضافة إلى نشاطها المضاد للأكسدة. كذلك حسنت المورينجا من أعراض السكري من خلال تثبيط إنزيمات الهضم، وتقليل مستويات الجلوكوز في الدم، ومقاومة الإنسولين، والالتهابات، في حين زادت من الخصائص المضادة للأكسدة في مصل الدم وأنسجة البنكرياس. كما حسنت صورة دهون الدم، وحافظت على وظائف الكبد والكلى. بالإضافة إلى ذلك، أظهرت التقييمات الحسية للخبز والبيتزا المضاف إليها المورينجا قبولًا جيدًا، على الرغم من أن الجرعات العالية أثرت على اللون. توضح الدراسة أن المورينجا لها القدرة على ضبط السكري من النوع الثاني وتوصي بمزيد من الابحاث لتحديد الجرعة المثالية والآمنة، ومدة الاستخدام، وطرق التطبيق على البشر.

الكلمات المفتاحية: مورنغا أوليفيرا، مستوى الجلوكوز، الإنسولين، HOMA-IR.

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