RESEARCH PAPER

Ameliorative Effects of Moringa oleifera Leaf Extract on High-Fat Diet-Induced Obesity and Metabolic Dysregulation in Experimental Rats

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Received: 5th January, 2026 | **Revised:** 11th Feb, 2026 | **Accepted:** 10th March, 2025 | **Available Online:** 5th April, 2025

ABSTRACT

Type 2 diabetes mellitus (T2DM) is a complex metabolic condition characterized by impaired insulin sensitivity, progressive β-cell dysfunction, abnormal lipid metabolism, and long-term complications affecting multiple organs. The present investigation aimed to evaluate the therapeutic potential of a methanolic extract of Moringa oleifera leaves (MOE) in an experimental model of T2DM induced by a high-fat diet (HFD) combined with streptozotocin (STZ) administration. Male Wistar rats were subjected to HFD feeding followed by a low-dose STZ injection to establish diabetic conditions. Subsequently, the animals received MOE at a dose of 300 mg/kg/day via oral administration for a duration of three weeks. Various physiological and biochemical parameters, including body weight, food intake, fasting blood glucose, lipid profile, and markers of renal and hepatic function, were systematically assessed. Rats fed with HFD demonstrated a significant increase in body weight irrespective of changes in food consumption, indicating its strong obesogenic effect. Diabetic control animals exhibited marked hyperglycaemia, altered lipid profile, and elevated biochemical indicators of renal and hepatic impairment. Treatment with MOE resulted in a significant reduction in fasting blood glucose levels and improvement in lipid parameters, notably total cholesterol and triglycerides. Furthermore, MOE administration mitigated diabetes-associated renal dysfunction, as reflected by decreased serum urea levels, while showing no adverse impact on hepatic function. Overall, the findings suggest that the methanolic extract of Moringa oleifera leaves exhibits potent antihyperglycaemic and hypolipidaemic effects, along with moderate renoprotective activity, highlighting its potential role as an adjunct therapeutic option for the management of type 2 diabetes mellitus.

Keywords: Moringa oleifera; Type 2 diabetes mellitus; High-fat diet; Streptozotocin; Dyslipidaemia

How to cite this article: Babu JD, Dave PC, Chauhan J, Landge AD, Singh J, Agrawal SKL, Kumar A, Nagaraj. Ameliorative Effects of Moringa oleifera Leaf Extract on High-Fat Diet-Induced Obesity and

Ameliorative Effects of Moringa oleifera Leaf Extract on High-Fat Diet-Induced Obesity and Metabolic Dysregulation in Experimental Rats

Metabolic Dysregulation in Experimental Rats. Int J Drug Deliv Technol. 2026;16(23s): 363-370. DOI: 10.25258/ijddt.16.23s.38
Source of support: Nil.
Conflict of interest: None

1. Introduction

Type-2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction. The global burden of T2DM has increased dramatically due to lifestyle changes, including consumption of calorie-dense high-fat diets and reduced physical activity, leading to obesity, dyslipidaemia and metabolic syndrome [1,2]. Long-term hyperglycaemia is associated with oxidative stress, inflammation and vascular complications, emphasizing the need for safer and more effective therapeutic strategies [3].

Experimental models combining a high-fat diet (HFD) with low-dose streptozotocin (STZ) are widely accepted for mimicking human T2DM. HFD feeding induces peripheral insulin resistance and lipid abnormalities, while subsequent administration of low-dose STZ causes partial β-cell damage, closely resembling the pathophysiology of T2DM in humans [4,5]. This model is therefore considered suitable for evaluating antidiabetic agents that target multiple metabolic defects.

Moringa oleifera Lam., commonly known as the drumstick tree, is a medicinal plant extensively used in traditional medicine. Its leaves are rich in bioactive constituents such as flavonoids, phenolic acids, vitamins, minerals and glucosinolates, which contribute to its antioxidant, anti-inflammatory and hypoglycaemic properties [6,7]. Several experimental studies have demonstrated that *M. oleifera* improves glucose tolerance, enhances insulin sensitivity and ameliorates dyslipidaemia in diabetic models [8].

The antidiabetic potential of *M. oleifera* has been attributed to multiple mechanisms, including protection of pancreatic β-cells from oxidative damage, modulation of insulin signalling pathways, inhibition of intestinal carbohydrate-digesting enzymes and improvement of lipid metabolism [9]. Its strong antioxidant activity further helps in reducing oxidative stress, a key contributor to insulin resistance and diabetic complications [10].

Considering the multifactorial nature of HFD- and STZ-induced T2DM, *Moringa oleifera* represents a promising natural therapeutic agent. Therefore, the present study was designed to evaluate the ameliorative activity of *Moringa oleifera* against high-fat diet and streptozotocin-induced type-2 diabetes, with particular emphasis on glycaemic control, lipid profile, oxidative stress parameters and pancreatic histopathology.

2. Materials and Methods

2.1. Chemicals and Materials

Streptozotocin (STZ), diethyl ether (98% purity), and all other chemicals and solvents used in the study were of analytical reagent grade. These included methanol (97%), distilled water, trisodium citrate dihydrate, and citric acid. A 0.1 M citrate buffer (pH 4.5) was freshly prepared using trisodium citrate dihydrate and citric acid for the dissolution of STZ immediately prior to administration. All reagents were used as received without further purification.

2.2. Preparation of *Moringa oleifera* Extract (MOE)

Fresh leaves of *Moringa oleifera* were collected from a local source and authenticated by a qualified taxonomist. The leaves were shade-dried at ambient temperature and subsequently ground into a fine powder.

Approximately 500 g of the powdered material was subjected to maceration in 2 L of methanol (97%) for 48 hours at room temperature. Following extraction, the mixture was filtered to remove plant residues.[11]

The filtrate was concentrated under controlled conditions, and the resulting semi-solid mass was further dried on a water bath to yield a viscous, dark green extract. The dried extract (yield approximately 45 g) was stored in an airtight glass container until further use.

2.3. Preparation of Normal and High-Fat Diets

Two types of experimental diets were utilized: a normal control diet (CON) and a high-fat diet (HFD). The detailed composition of both diets is provided in Table 1. The primary difference between the diets was the proportion of fat content and overall caloric density.

In the HFD formulation, a portion of cornstarch present in the control diet was replaced with animal fat to increase lipid content. The inclusion of animal fat, which is rich in saturated fatty acids, was intended to induce obesity and metabolic disturbances associated with type 2 diabetes in experimental animals.[12]

2.4. Experimental Animals

Male Wistar rats weighing between 170 and 200 g were used in the present study. All experimental procedures were conducted in accordance with the guidelines of the Institutional Animal Ethics Committee (IAEC) and the Committee for the Control and Supervision of Experiments on Animals (CCSEA), India.

Prior to initiation of the experimental protocol, animals were acclimatized to standard laboratory conditions for one week. The total duration of the study was two months, during which animals were maintained under controlled environmental conditions with free access to food and water.

2.5. Experimental Induction of Type 2 Diabetes Mellitus

During the initial phase of the study, animals were maintained on a high-fat diet for a period of four weeks to induce peripheral insulin resistance. Subsequently, partial pancreatic β-cell dysfunction was induced through administration of streptozotocin (STZ).

Animals were fasted beginning at 06:00 h prior to STZ administration. A single intraperitoneal injection of STZ (40 mg/kg body weight) was administered at 13:00 h. The STZ solution was freshly prepared in ice-cold 0.1 M sodium citrate buffer (pH 4.5) immediately before use. To preserve its stability, the solution was protected from light and maintained under cold conditions throughout preparation and administration. Control and non-diabetic animals received an equivalent volume (0.5 mL) of citrate buffer alone.

To minimize the risk of acute hypoglycaemia resulting from transient insulin release, animals were provided with 10% glucose solution in place of drinking water for 48 hours, commencing 6 hours after STZ injection. Diabetic status was confirmed after 72 hours by measuring fasting blood glucose levels using tail vein samples and a handheld glucometer. Animals with blood glucose levels exceeding 300 mg/dL were considered diabetic and included in subsequent experimental procedures.

Body weight was recorded on a weekly basis, and food intake was monitored throughout the study by calculating the difference between the amount of feed provided and the residual feed remaining in each cage.[13,14]

2.6. Experimental Design

A total of twenty-four male Wistar rats were randomly allocated into four experimental groups (n = 6 per group) and maintained under their respective dietary and treatment conditions throughout the study period. The normal control group (CON) received a standard laboratory diet for two months, whereas the high-fat diet (HFD) group was fed a high-fat diet for the entire duration of the experiment.

For induction of type 2 diabetes mellitus, animals in the diabetic control group (DM) were initially subjected to a high-fat diet for four weeks, followed by administration of streptozotocin. After confirmation of the diabetic state, these animals continued on the high-fat diet until the end of the study.

The treatment group (DM–MOE) underwent an identical diabetic induction protocol and subsequently received methanolic extract of *Moringa oleifera* leaves. The extract was freshly prepared in distilled water and administered orally at a dose of 300 mg/kg body weight once daily for three weeks using an oral gavage technique. All treatments were administered at 21:00 h to ensure consistency in dosing and minimize circadian variability.[15,16]

Accordingly, the experimental groups were defined as follows:

1. **CON group**: Rats fed a normal control diet for the entire study period.
2. **HFD group**: Rats fed a high-fat diet throughout the study duration.
3. **DM group**: Rats rendered diabetic by high-fat diet feeding followed by STZ injection and maintained on HFD until study completion.
4. **DM–MOE group**: Diabetic rats treated orally with *M. oleifera* methanolic leaf extract (300 mg/kg/day) for three weeks.

2.7. Biochemical Analysis

At the end of the study, animals were anesthetized and blood samples were collected via cardiac puncture. The blood was allowed to clot and centrifuged at 3000 rpm for 15 min to separate serum. Serum glucose was measured to evaluate glycemic status, while lipid profile parameters included triglycerides, total cholesterol, and HDL. [17]

2.8. Statistical Analysis

All data were analysed using SPSS software (version 22) and expressed as mean ± SD. Statistical comparisons were performed using one-way ANOVA followed by Tukey's post hoc test. A value of $P < 0.05$ was considered statistically significant.

3. Results and Discussion

3.1. Changes in Body Weight and Food Consumption

Rats subjected to a high-fat diet (HFD) exhibited a significant increase in body weight compared with the normal control (CON) group ($p < 0.001$). Moreover, body weight gain in the HFD group was significantly greater than that observed in diabetic control (DM) animals ($p < 0.001$). Treatment with *Moringa oleifera* extract did not produce a statistically significant change in body weight in the treated diabetic group (DM–MOE) relative to untreated diabetic rats.

Analysis of food intake revealed that CON animals consumed significantly higher quantities of feed compared with HFD-fed rats ($p < 0.001$). Despite greater food consumption, CON rats maintained lower body weights, underscoring the higher obesogenic efficiency of the high-fat diet. No significant differences in food intake were observed among the HFD, DM, and DM–MOE groups, indicating that variations in body weight were primarily attributable to metabolic alterations rather than differences in caloric intake.

These findings suggest that chronic intake of a fat-enriched diet promotes excessive weight gain independent of food consumption, thereby highlighting its critical role in the development of obesity-associated insulin resistance.

3.2. Effects of *Moringa oleifera* Extract on Blood Glucose and Lipid Profile

Table 2 summarizes the impact of *Moringa oleifera* leaf extract on fasting blood glucose and serum lipid parameters across experimental groups. The diabetic control (DM) group exhibited a significant elevation in fasting blood glucose levels compared with the normal control (CON) group (**p < 0.001), confirming successful induction of type 2 diabetes. Administration of *Moringa oleifera* extract (DM–MOE) resulted in a significant reduction in blood glucose levels relative to untreated diabetic rats (#p < 0.05). However, glucose levels remained higher than those of the CON group, indicating a partial yet meaningful antihyperglycaemic effect.

In terms of lipid parameters, the DM group demonstrated a significant increase in total cholesterol (TC) ($p < 0.05$) along with elevated triglyceride (TG) levels compared with CON animals, indicative of diabetes-associated dyslipidaemia. Treatment with *Moringa oleifera* extract led to a reduction in TC and TG levels in the DM–MOE group compared with the DM group, suggesting an improvement in lipid metabolism, although these changes did not reach statistical significance. High-density lipoprotein cholesterol (HDL-C) levels did not differ significantly among the experimental groups.

Overall, these results indicate that *Moringa oleifera* extract exerts a significant antihyperglycaemic effect and demonstrates a favorable trend toward amelioration of diabetes-induced lipid abnormalities.

3.3. Influence of *Moringa oleifera* Extract on Renal and Hepatic Biochemical Parameters

Table 3 presents the effects of *Moringa oleifera* extract on renal and hepatic biochemical markers across the study groups. The diabetic control (DM) group showed a significant increase in serum urea levels ($p < 0.05$) compared with the normal control (CON), indicating the onset of diabetes-associated renal dysfunction. Treatment with *Moringa oleifera* extract (DM–MOE) significantly reduced serum urea levels relative to untreated diabetic rats (#p < 0.05), suggesting a renoprotective effect. Serum creatinine levels remained statistically comparable across all groups, indicating preservation of glomerular filtration function within the study period.

Regarding hepatic parameters, both HFD and DM groups exhibited elevated activities of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) compared with the CON group, reflecting hepatic stress associated with high-fat feeding and hyperglycaemia. Although AST and ALT levels remained elevated in the DM–MOE group, no further significant increase was observed, indicating that *Moringa oleifera* treatment did not exacerbate hepatic dysfunction.

Collectively, these findings suggest that *Moringa oleifera* extract provides partial protection against renal impairment while maintaining hepatic safety under diabetic conditions.

4. Discussion

The present investigation evaluated the protective potential of the methanolic extract of *Moringa oleifera* leaves (MOE) in a high-fat diet (HFD) and streptozotocin (STZ)-induced type 2 diabetes mellitus (T2DM) rat model, with a focused assessment of metabolic, glycaemic, lipid, renal, and hepatic parameters. The experimental design effectively reproduced hallmark characteristics of human T2DM, including persistent hyperglycaemia, dyslipidaemia, alterations in body weight dynamics, and organ-specific biochemical perturbations, thereby providing a reliable platform for assessing the therapeutic efficacy of MOE.[18]

Chronic consumption of an HFD resulted in a significant increase in body weight despite comparatively reduced food intake relative to normal controls, indicating the high obesogenic efficiency of dietary fat. This observation is consistent with previous findings suggesting that HFD-induced adiposity is primarily driven by increased energy density and metabolic imbalance rather than absolute caloric intake. Notably, diabetic animals exhibited relatively lower weight gain compared with HFD-fed non-diabetic counterparts, which may be attributed to insulin deficiency–associated catabolic processes, including enhanced proteolysis and lipolysis. Administration of MOE did not produce a statistically significant effect on body weight in diabetic rats, indicating that its pharmacological action is predominantly metabolic rather than related to appetite suppression or weight regulation.[19,20]

Marked hyperglycaemia observed in diabetic control animals confirmed the successful establishment of insulin resistance and pancreatic β-cell dysfunction. Treatment with MOE resulted in a significant reduction in fasting blood glucose levels, demonstrating a pronounced antihyperglycaemic effect. This activity may be associated with the presence of bioactive phytoconstituents such as flavonoids, phenolic compounds, isothiocyanates, and quercetin, which are known to enhance insulin sensitivity, promote peripheral glucose utilization, inhibit intestinal glucose absorption, and confer cytoprotective effects on pancreatic β-cells through antioxidant mechanisms. Although normoglycaemia was not fully restored, the observed improvement underscores the potential utility of MOE as an adjunctive therapeutic agent in diabetes management.[21-22]

Diabetes-induced dyslipidaemia was evident in the diabetic control group, as indicated by elevated levels of total cholesterol and triglycerides. These alterations are commonly associated with insulin resistance and enhanced hepatic lipid synthesis. Treatment with MOE produced a favorable trend toward normalization of lipid parameters, particularly total cholesterol and triglycerides, suggesting an improvement in lipid homeostasis. The hypolipidaemic action of *Moringa oleifera* may be attributed to modulation of lipid-metabolizing enzymes, increased bile acid excretion, and antioxidant-mediated inhibition of lipid peroxidation. High-density lipoprotein cholesterol (HDL-C) levels remained relatively unchanged across experimental groups, indicating that the extract predominantly influenced atherogenic lipid fractions.[23-24]

Evaluation of renal function revealed a significant elevation in serum urea levels in diabetic animals, indicative of early renal impairment associated with chronic hyperglycaemia. Treatment with MOE significantly reduced serum urea concentrations, suggesting a renoprotective effect, potentially mediated through improved glycaemic control and attenuation of oxidative stress-induced renal injury. The absence of significant changes in serum creatinine levels across groups suggests that glomerular filtration remained largely preserved within the study duration.[25-26]

Assessment of hepatic enzymes demonstrated increased activities of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) in HFD-fed and diabetic groups, reflecting hepatic stress and possible steatotic changes associated with insulin resistance and lipid accumulation. Although MOE administration did not significantly normalize these enzyme levels, it did not exacerbate hepatic injury, indicating a favorable hepatic safety profile at the tested dose. Previously reported hepatoprotective effects of *Moringa oleifera* are largely attributed to its antioxidant and anti-inflammatory properties, which may require extended treatment duration or dose optimization to achieve more pronounced outcomes.[27,28]

Collectively, the results of the present study demonstrate that the methanolic extract of *Moringa oleifera* leaves exerts significant anti-hyperglycaemic effects, improves diabetes-associated dyslipidaemia, and provides partial protection against renal dysfunction without inducing hepatotoxicity. These findings substantiate its traditional use in the management of diabetes and suggest its potential as a complementary therapeutic agent for mitigating metabolic complications associated with T2DM. Further investigations focusing on mechanistic pathways, dose optimization, and long-term efficacy are warranted to validate its clinical applicability[29-43].

5. Conclusion

The findings of the present study indicate that the methanolic extract of Moringa oleifera leaves exhibits notable antihyperglycaemic activity in a type 2 diabetes mellitus model induced by a high-fat diet and streptozotocin. Administration of the extract led to a significant reduction in elevated blood glucose levels and demonstrated a beneficial effect on diabetes-associated lipid abnormalities, particularly through the reduction of total cholesterol and triglyceride concentrations.

Furthermore, the extract conferred partial protection against diabetes-related renal impairment, as reflected by decreased serum urea levels, while showing no evidence of hepatotoxicity at the tested dose.

Although treatment with Moringa oleifera did not produce a significant effect on body weight or fully restore altered hepatic enzyme levels, the overall improvement in metabolic parameters underscores its potential therapeutic value. These results align with its traditional medicinal use and suggest that Moringa oleifera may serve as a supportive agent in the management of type 2 diabetes and its associated complications.

However, further detailed investigations are required to elucidate the underlying molecular mechanisms, establish optimal dosing regimens, and evaluate its long-term efficacy and safety profile.

Table 1. Composition of Experimental Diets

Component	Normal Control Diet (CON) (% w/w)	High-Fat Diet (HFD) (% w/w)
Cornstarch	38.0	10.0
Ground wheat	20.0	5.0
Wheat bran	6.0	14.0
Beef tallow fat	3.0	22.0
Soybean meal	27.0	33.0
Animal protein	3.5	3.5
Calcium source	2.5	2.5
Total	100	100

Abbreviations: CON, normal control diet; HFD, high-fat diet.

Table 2. Effects of *Moringa oleifera* Extract on Blood Glucose and Serum Lipid Profile

Group	Glucose (mg/dL)	TC (mg/dL)	TG (mg/dL)	HDL-C (mg/dL)
CON	158.7 ± 35.2	46.3 ± 10.5	115.6 ± 34.2	30.8 ± 6.9
HFD	210.4 ± 42.8	54.9 ± 11.3	192.7 ± 49.8	32.1 ± 7.5
DM	612.5 ± 138.6***	68.2 ± 13.4*	205.9 ± 70.3	34.5 ± 8.2
DM–MOE	395.3 ± 129.7#	57.8 ± 13.6	172.6 ± 65.4	27.9 ± 12.8

Values are expressed as mean ± SD (n = 6). ***p < 0.001 compared with the normal control group; *p < 0.05 compared with the normal control group; #p < 0.05 compared with the diabetic control group.

Table 3. Effects of *Moringa oleifera* Extract on Renal and Hepatic Biochemical Parameters

Group	Urea (mg/dL)	Creatinine (mg/dL)	AST (U/L)	ALT (U/L)
CON	31.8 ± 7.6	0.60 ± 0.05	122.7 ± 32.4	39.5 ± 11.3
HFD	25.4 ± 3.1	0.67 ± 0.07	198.6 ± 48.9	66.2 ± 21.7
DM	57.2 ± 10.4*	0.71 ± 0.05	245.3 ± 128.7	88.6 ± 40.2
DM–MOE	42.6 ± 12.1#	0.68 ± 0.09	275.9 ± 198.3	81.7 ± 54.6

Values are expressed as mean ± SD (n = 6). *P < 0.05 compared with the normal control group; #P < 0.05 compared with the diabetic control group.

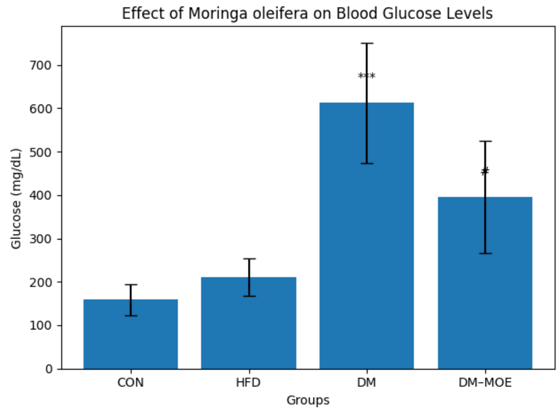

Fig. 01: Effects of high-fat diet and *Moringa oleifera* extract on body weight

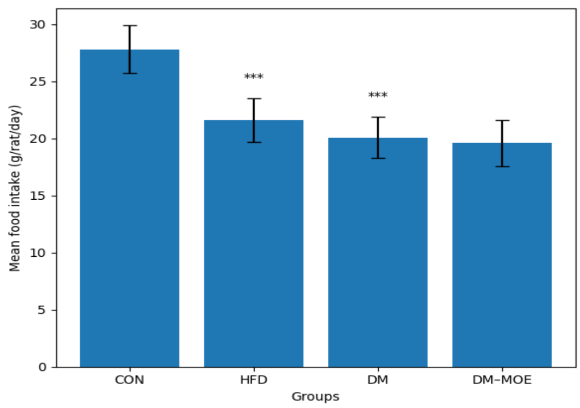

Fig. 02: Effects of high-fat diet and *Moringa oleifera* extract on food intake

6.References

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