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Review Article

Moringa oleifera: A promising candidate for managing metabolic syndrome

Mahboobeh Ghasemzadeh Rahbardar[1,2], Sercan Karav[3,*], Amirhossein Sahebkar[4,5,6,*]

[1]*Department of Emergency Medicine, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran*
[2]*Clinical Research Development Unit, Shahid Hasheminejad Hospital, Mashhad University of Medical Sciences, Mashhad, Iran*
[3]*Department of Molecular Biology and Genetics, Canakkale Onsekiz Mart University, Canakkale 17100, Turkey*
[4]*Applied Biomedical Research Center, Mashhad, University of Medical Sciences, Mashhad, Iran*
[5]*Centre for Research Impact & Outcome, Chitkara College of Pharmacy, Chitkara University, Rajpura, Punjab 140401, India*
[6]*Biotechnology Research Center, Pharmaceutical Technology Institute, Mashhad University of Medical Sciences, Mashhad, Iran*

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**** Corresponding Author:***
Tel: +985131801239
Fax: +985138823251
amir_saheb2000@yahoo.com

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Abstract

Objective: Metabolic syndrome, as defined, is not a disease in and of itself but rather a collection of metabolic risk factors that frequently coexist. Abdominal obesity, hyperglycemia, hypertriglyceridemia, hypertension, and low amounts of high-density lipoprotein cholesterol (HDL-C) are among the risk factors. *Moringa oleifera* possesses hypotensive, anti-obesity, hypolipidemic, and anti-diabetic properties. Our study aims to determine if *M. oleifera* and its main constituents are potentially beneficial in treating metabolic syndrome and its accompanying complications.

Materials and Methods: This narrative review was conducted on the effects of *M. oleifera* and its main constituents against metabolic syndrome by searching different electronic databases.

Results: *M. oleifera* exerts multifaceted effects on metabolic syndrome through distinct molecular pathways. It improves glucose homeostasis by enhancing glucose tolerance, lowering fasting glucose and glycated hemoglobin (HbA1c), and modulating α-glucosidase activity and glucose transporters. Its antioxidant properties restore hepatic enzyme balance, protect pancreatic islets, and reduce oxidative damage. Anti-obesity actions include inhibiting adipogenesis, reducing triglyceride storage, increasing adipocyte apoptosis and adiponectin levels. Lipid-modulating effects involve elevating HDL, reducing high-density lipoprotein (LDL) and total cholesterol, and regulating lipid-related genes and receptor activity. For blood pressure control, it promotes vasodilation via nitric oxide enhancement and calcium influx inhibition.

Conclusion: Based on previous investigations, there is evidence suggesting that *M. oleifera* has the potential to control metabolic syndrome and mitigate its complications. However, it is important to note that further preclinical and clinical studies are necessary to validate and confirm the beneficial effects of this herb on metabolic syndrome.

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Introduction

Metabolic syndrome does not represent a single disease entity; instead, it encompasses a cluster of interrelated metabolic abnormalities that tend to occur together. These include central (abdominal) obesity, elevated blood glucose levels, increased triglycerides, high blood pressure, and reduced concentrations of high-density lipoprotein cholesterol (HDL-C). The presence of these risk factors raises the probability of type 2 diabetes and cardiovascular disease considerably (Ghasemzadeh Rahbardar et al. 2024; Jalali and Ghasemzadeh Rahbardar 2022). It is crucial to keep in mind that the reported prevalence of the metabolic syndrome can vary widely depending on a number of variables, including the precise criteria used to diagnose it, the gender and age distribution of the study population, the socioeconomic status of the participants, and the ethnic background of the cohorts being studied. The complexity of metabolic syndrome and the impact of numerous variables on its frequency in distinct populations are highlighted by these differences. Furthermore, inactivity, excessive food consumption, and the development of abdominal obesity are identified as the main variables contributing to the onset of metabolic syndrome (Wang et al. 2020). The specific pathophysiological process driving the development of metabolic syndrome is unknown. However, it is highly proposed that abdominal obesity and insulin resistance are important variables in its etiology (Oskouei et al. 2023). As a result, as the first step in the prevention and treatment of metabolic syndrome, lifestyle changes and weight loss should be emphasized (Ghasemzadeh Rahbardar et al. 2024). In addition, it is critical for people with metabolic syndrome to carefully control additional cardiovascular risk factors (Bozkurt et al. 2016). Due to the complicated interplay of multiple factors involved in metabolic syndrome, there is a growing need to investigate innovative treatment approaches that can address its diverse and complex physiological mechanisms.

In recent years, there has been growing interest in the use of herbal products as complementary or alternative strategies for managing different disorders (Ghasemzadeh Rahbardar and Hosseinzadeh 2023; Hosseini et al. 2011; Iranshahi et al. 2010; Iranshahi et al. 2009; Mohammadi Zonouz et al. 2024; Parsamanesh et al. 2021) including metabolic syndrome (Francini-Pesenti et al. 2019; Sahebkar 2013; Sankar et al. 2025). This interest stems from the limitations of conventional pharmacological therapies, which may be associated with high costs, adverse effects, and limited long-term efficacy. Herbal medicines, with their multi-targeted actions offer a promising avenue for intervention. Previous studies have shown that some plants or their main constituents such as *Solanum melongena* (Yarmohammadi et al. 2021), *Portulaca oleracea* (Jalali and Ghasemzadeh Rahbardar 2022), *Nigella sativa* (Fadishei et al. 2021), *Elettaria cardamomum* (Yahyazadeh et al. 2021), Dendrobium (Oskouei et al. 2023), zeaxanthin (Salehsari et al. 2024), curcumin (Panahi et al. 2016), alpha-lipoic acid (Najafi et al. 2022), and alpha-mangostin (Ardakanian et al. 2022) are effective in managing metabolic syndrome and its comorbidities. These botanicals often act through modulation of oxidative stress, inflammation (Sankar et al. 2025), and metabolic signaling pathways such as adenosine monophosphate-activated protein kinase (AMPK) (Sangouni et al. 2021), peroxisome proliferator-activated receptor (PPAR) (Vezza et al. 2021), and Sirtuin 1 (SIRT1) (Alla et al. 2025).

Moringa oleifera Lam. (moringa, miracle tree, or drumstick tree) is a member of the Moringaceae family (SWATIą et al. 2018). This subtropical tree is native to Asia and Africa, mostly in the Sub-Himalayas. However, due to its multiple beneficial applications, its cultivation has

spread globally. *M. oleifera* grows quickly in areas with high temperatures and low water supplies (Nouman et al. 2014). Traditionally, *M. oleifera* has been utilized for various purposes, including the treatment of diabetes mellitus, diarrhea, venomous bites, rheumatism, cardiac stimulation, and as a diuretic (Umar et al. 2018). The nutritional and therapeutic significance of numerous components of this tree, including its leaves, bark, roots, flowers, fruit, and seeds, has been well recognized. The plant has received a lot of attention since the first international conference on *M. oleifera* in 2001 and has been dubbed the "mother's best friend", "miracle tree", or "natural gift" (Chukwuebuka 2015; Mahmood et al. 2010). *M. oleifera* is recognized as one of the most nutrient-rich food plants, containing high amounts of essential amino acids, minerals, polyphenols, proteins, and vitamins. It is abundant in phytochemicals, including alkaloids, anthocyanins, anthraquinone, cardiac glycosides, essential oils, flavonoids, isothiocyanates, saponins, steroids, tannic acid, and terpenoids (Anzano et al. 2021). Several therapeutic properties have been reported for various parts of this plant, including antioxidant, anti-inflammatory (Xu et al. 2019), analgesic (Abdul Haseeb et al. 2021), anti-allergic (Ouattara-Soro et al. 2022), anticonvulsant (Alam et al. 2023), antidiabetic (Wang et al. 2022), antimicrobial (Abd El-Hack et al. 2022), antiulcer (Adji et al. 2022), antiviral (Biswas et al. 2020), anticancer (Adam et al. 2023), and cardioprotective (Patintingan et al. 2023) effects.

This study intends to emphasize the potential effectiveness of *M. oleifera* and its main components in treating metabolic syndrome and its consequences while summarizing the pharmacological characteristics of this plant. We hope to bridge the gap between fundamental scientific understanding and its application in patient care by combining the data and bringing it to the attention of scientists and researchers. The probable *M. oleifera* health advantages in relation to metabolic syndrome can help open the door for more study and clinical testing, which will eventually help those who suffer from these disorders.

Materials and Methods

A comprehensive literature search was conducted to identify relevant studies evaluating the potential of *M. oleifera* in managing components of metabolic syndrome. The search was performed across three major databases—PubMed, Scopus, and Google Scholar—covering publications from January 2013 to July 2025.

Search strategy

A combination of Medical Subject Headings (MeSH) and free-text terms was used to maximize retrieval sensitivity. The following keywords and their Boolean combinations were employed: "anti-diabetic", "anti-hyperglycemic", "anti-hyperlipidemic", "anti-hypertensive", "anti-obesity", "atherosclerosis", "blood glucose", "blood pressure", "diabetes", "drumstick", "dyslipidemia", "high cholesterol", "high triglyceride", "hypercholesterolemia", "hyperglycemia", "hyperlipidemia", "hypertension", "hypertriglyceridemia", "hypoglycemic", "hypotensive", "insulin", "metabolic syndrome", "miracle tree', "Moringa", "*Moringa oleifera*", "obesity", "overweight", "serum lipids", and "weight loss".

Inclusion criteria

Studies were included if they met the following criteria:

- Published in English (at least the abstract) between 2013 and August 2023
- Published in peer-reviewed journals
- Investigated the effects of *M. oleifera* on one or more components of metabolic syndrome (e.g. hyperglycemia, dyslipidemia, hypertension, and obesity)

- Included *in vitro*, *in vivo* (animal), or human clinical studies
- Reported relevant biochemical, physiological, or clinical outcomes
- Used *M. oleifera* in any form (e.g. leaf extract, seed oil, powder, isolated compounds)

Exclusion criteria

The following types of studies were excluded:

- Review articles, editorials, commentaries, and conference abstracts
- Studies not focused on metabolic syndrome or its components
- Articles lacking sufficient methodological detail or outcome data
- Studies using multi-herbal formulations where the specific effect of *M. oleifera* could not be isolated
- Non-English publications

Study selection and data extraction

Titles and abstracts were screened for relevance, followed by full-text review of potentially eligible articles. Data were extracted on study design, model (*in vitro*, *in vivo*, and clinical), dosage and form of *M. oleifera*, duration of intervention, and key outcomes related to metabolic syndrome.

Quality considerations

Although this is a narrative review, methodological rigor and relevance were prioritized. Preference was given to studies with appropriate controls, validated outcome measures, and clear reporting of statistical significance. Clinical trials were assessed for sample size, randomization, and blinding where applicable.

Results

Effect of *M. oleifera* on Diabetes

Diabetes mellitus refers to a group of diverse metabolic conditions characterized primarily by persistently elevated blood glucose levels. This condition arises due to impaired insulin production, varying degrees of insulin resistance, or a combination of both factors (Schleicher et al. 2022). The diagnostic criteria for diabetes mellitus are based on the recommendations of leading international bodies, including the American Diabetes Association (ADA), the European Association for the Study of Diabetes (EASD), the International Diabetes Federation (IDF), and the World Health Organization (WHO). Diagnosis can be established by one of the following: a random venous plasma glucose level of ≥ 11.1 mmol/l (200 mg/dl), a fasting plasma glucose (FPG) level of ≥ 7.0 mmol/l (126 mg/dl) after 8–12 hr of fasting, a 2-hr plasma glucose level of ≥ 11.1 mmol/l (200 mg/dl) during an oral glucose tolerance test (OGTT), or a glycated hemoglobin (HbA1c) value of ≥ 48 mmol/mol (6.5%) (Schleicher et al. 2022). Diabetes mellitus is triggered by a number of genetic and environmental factors (Bai et al. 2016). It is characterized by disturbed protein and lipid metabolism, high blood glucose levels, and impairments to the nervous system, retina, liver, and kidneys (Chugh 2019). This syndrome is identified by insufficient insulin secretion and/or insulin resistance due to β-cell dysfunction (Ghasemzadeh Rahbardar et al. 2025b; Jalali and Ghasemzadeh Rahbardar 2022). The global burden of diabetes, especially type 2 diabetes, is escalating due to a combination of factors including population aging, lack of physical activity, poor nutritional habits, and increased obesity rates (Sobhani et al. 2025).

Recent research indicates that oxidative stress and inflammation are likely responsible for the loss of β-cells in diabetes. As evidenced by the formation of free radicals, particularly reactive oxygen species (ROS), and decreased glutathione metabolism, oxidative stress is a critical component in the development of diabetes. Auto-oxidation of glucose, the formation of lipid peroxides, changes in antioxidant enzymes, and non-enzymatic protein glycosylation are all factors that contribute

to oxidative stress in diabetes. Furthermore, oxidative stress activates many inflammatory signaling pathways, resulting in inflammation. Pro-inflammatory signal expression can attract local inflammatory cells, improving local inflammation and eventually leading to type 2 diabetes and β-cell death (Samarghandian et al. 2017).

The current pharmacological management of type 2 diabetes includes agents such as metformin, sodium-glucose cotransporter-2 (SGLT2) inhibitors, glucagon-like peptide-1 (GLP-1) receptor agonists, dipeptidyl peptidase-4 (DPP-4) inhibitors, sulfonylureas, and insulin therapy (Choi et al. 2022; Schroeder 2022). While these therapies are effective in glycemic control, limitations such as adverse side effects, high costs, limited accessibility, and reduced long-term efficacy highlight a pressing need for complementary treatment options. In this context, the exploration of plant-based interventions, including *M. oleifera*, offers a promising avenue for enhancing glycemic regulation with potentially fewer side effects.

The following part will include research on the effects of *M. oleifera* on diabetes, insulin resistance, and glucose metabolism.

In vitro

M. oleifera leaf extract enhanced glucose uptake in insulin-resistant HepG2 cells. Network pharmacology and bioinformatics analyses predicted protein kinase B (Akt)1 and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as central molecular targets and suggested that hypoglycemic effects of the extract are closely linked to the phosphoinositide 3-kinase (PI3K)-Akt signaling pathway (Hong et al. 2023).

In vitro evaluations demonstrated that *M. oleifera* leaf extract possesses notable antioxidant and anti-adipogenic properties. At a concentration of 1 mg/ml, the extract achieved approximately 85% 2,2-Diphenyl-1-picrylhydrazyl (DPPH) radical scavenging activity and exhibited substantial ferric-reducing antioxidant power. In adipocyte culture, the extract significantly inhibited lipid accumulation. This effect was accompanied by downregulation of key adipogenic transcription factors—PPARγ, sterol regulatory element-binding protein (SREBP), and CCAAT/enhancer binding protein-α (C/EBPα). Additionally, the extract demonstrated approximately 60% inhibition of both lipase and dipeptidyl peptidase 4 (DPP4) enzymatic activities. It also reduced expression of DPP4, AMPK, and NAD(P)H:quinone oxidoreductase 1 (NQO1) genes. Moreover, it enhanced GLP-1 secretion by 25% in L-cell models (Kumar et al. 2025).

Studies containing both *in vitro* and *in vivo* parts

It has been demonstrated that treating yeast cells with *M. oleifera* leaf aqueous extract resulted in enhanced glucose tolerance and rate of glucose uptake, as well as a reduction in α-amylase and α-glucosidase activities. In the *in vivo* part of the study, the administration of *M. oleifera* leaf aqueous extract to rodents significantly reduced fasting blood sugar (FBS) (Khan et al. 2017).

The bioactive peptide MoHpP-2, isolated from *M. oleifera* seeds, demonstrated potent α-glucosidase inhibition and was further evaluated for its antidiabetic effects *in vitro* and *in vivo*. In insulin-resistant C2C12 myocytes, MoHpP-2 significantly enhanced glucose utilization and antioxidant defense. In type 2 diabetic mice, induced via high-fat diet and streptozotocin, MoHpP-2 administration resulted in improved glycemic control, liver function, and systemic antioxidant capacity. Remarkably, it modulated gut microbiota composition by increasing microbial diversity, reducing the Firmicutes/Bacteroidetes ratio, and suppressing pathogenic taxa. Metabolomic profiling indicated that MoHpP-2 facilitated unsaturated fatty acid biosynthesis, suppressed bile acid

formation, and regulated linoleic and amino acid metabolism, all linked to oxidative stress mitigation and enhanced insulin responsiveness (Fan et al. 2024).

A study investigated the antidiabetic potential of dietary fibers derived from *M. oleifera* leaves (MDFs), emphasizing how particle size influences their metabolic effects. Among the tested variants, MDF200 demonstrated superior *in vitro* hypoglycemic activity by maximizing glucose adsorption, delaying diffusion, and effectively inhibiting digestive enzymes. *In vivo* findings using a type 2 diabetes mouse model (induced via high-fat diet and streptozotocin) revealed that all MDF formulations significantly improved insulin sensitivity, mitigated oxidative stress and dyslipidemia, and reduced tissue damage. MDF80 and MDF200 notably enhanced gut microbiota composition, increasing beneficial strains and reducing pathogenic species. MDF80 elevated levels of acetic and butyric acids, while MDF40 increased propionic acid. All MDF interventions boosted hepatic G protein-coupled receptor 43 (GPR43) expression and GLP-1 secretion. Mechanistically, larger particle MDFs activated AMPK and extracellular signal-regulated kinase (ERK) phosphorylation, whereas smaller particles stimulated GLP-1 and PI3K/Akt signaling (Li et al. 2025).

In vivo

Supplementing *M. oleifera* leaf aqueous extract to normal mice exhibited anti-hyperglycemic properties, but it reduced glucose tolerance impairment in mildly diabetic mice (Luangpiom et al. 2013). It has been reported that the administration of *M. oleifera* leaf aqueous extract could decrease blood glucose levels in both normoglycemic and diabetic rats (Edoga et al. 2013). According to the findings of a study, the aqueous extract of *M. oleifera* leaves has considerable hypoglycemic effects. These results are related to the extract antioxidant properties, which assist in the normalization of increased hepatic pyruvate carboxylase enzyme levels and the regeneration of damaged hepatocytes and pancreatic β–cells (Abd El Latif et al. 2014).

Similarly, the administration of an aqueous extract of *M. oleifera* leaves to diabetic rats elevated the amounts of pancreatic reduced glutathione (GSH) and declined FPG and MDA, as well as damage of islets of Langerhans cells (Yassa and Tohamy 2014). It has been illustrated that the administration of *M. oleifera* leaf ethanolic extract to diabetic rats could significantly decrease their blood glucose levels (Aja et al. 2015). Adding *M. oleifera* seed powder to the diet of diabetic rats enhanced the amounts of serum antioxidant enzymes and lowered lipid peroxide, interleukin (IL)-6, immunoglobulin (Ig) A, IgG, FBS, and HbA_{1c} (Al-Malki and El Rabey 2015).

M. oleifera leaf methanolic extract administration to diabetic rats was associated with increased glucose tolerance, serum insulin level, glycogen amounts, glycogen synthase activities, and glucose uptake (Olayaki et al. 2015). In another investigation, *M. oleifera* leaf ethanolic extract, moringinine, quercetin, and chlorogenic acid were administered to diabetic rats. The results revealed that the *M. oleifera* leaf ethanolic extract reduced blood glucose levels and MDA amounts. Also, quercetin showed the highest antidiabetic potential, followed by chlorogenic acid and moringinine (Ali et al. 2015).

The oral supplementation of *M. oleifera* leaf aqueous extract to diabetic mice resulted in increased total antioxidant capacity as well as interferon gamma (INF-γ), and a reduction in homeostatic model assessment for insulin resistance (HOMA-IR) levels (Tuorkey 2016). The findings of another study revealed that the administration of ethanolic extracts of *M. oleifera* flowers, leaves, roots, seed, and stem to diabetic rats lowered the blood glucose levels. Also, the extracts obtained from the seeds and leaves exhibited

stronger effects in reducing glucose levels and inhibiting α-amylase and α-glucosidase enzymes than the other extracts (Adejoh et al. 2016). The *M. oleifera* callus tissue ethanolic extract decreased blood glucose levels in diabetic mice (Oriabi 2016).

In addition, it has been shown that oral administration of *M. oleifera* leaf aqueous extract to diabetic rats increased glycogen synthase gene expression and attenuated blood glucose levels (Abd Eldaim et al. 2017). An investigation was aimed at evaluating the hypoglycemic effects of *M. oleifera*. Five different aqueous ethanol extracts with varying concentrations (95%, 75%, 50%, 25% v/v, and 100% water) were orally administered to healthy rats. Additionally, the extracts were tested on diabetic rats to assess their acute and sub-chronic anti-hyperglycemic properties. Moreover, the most potent extract was further divided into hexane, chloroform, ethyl acetate, butanol, and water fractions, which were screened for their anti-diabetic properties. The findings revealed that among all the extracts and fractions, the 95% (v/v) ethanol extract at a dose of 1,000 mg/kg, and its butanol fraction at a dose of 500 mg/kg, exhibited the highest activity by significantly reducing blood glucose levels after a single administration in diabetic rats. No significant hypoglycemic effects were observed in healthy rats (Irfan et al. 2017).

The administration of *M. oleifera* leaf ethanolic extract to mice with type 2 diabetes could pointedly increase insulin levels and attenuate FBS (Tang et al. 2017). The intraperitoneal injection of *M. oleifera* leaf protein isolate to diabetic mice improved catalase (CAT) amount, decreased MDA and blood glucose levels; however, it had no effect on insulin secretion (C. Paula et al. 2017). *M. oleifera* roots methanolic extract and roots powder reduced blood glucose levels, the amounts of serum superoxide dismutase (SOD), CAT, and glutathione peroxidase (GPx), as well as the damage to the islet of Langerhans in diabetic rats (Umar et al. 2018). Treating diabetic rats with *M. oleifera* leaf methanolic extract considerably increased glucose tolerance, fasting plasma insulin, and reduced FBS (Muzumbukilwa et al. 2019). Supplementing *M. oleifera* leaf methanolic extract to diabetic rats resulted in augmented levels of plasma insulin, SOD, CAT, GPx, glutathione-reductase (GRD), and reduced glutathione, though it lowered serum glucose, HbA_{1c}, thiobarbituric acid reactive substances (TBARS), and hydroperoxides (Aju et al. 2019). Additionally, it has been claimed that the *M. oleifera* crude extract-formulated tablets displayed antidiabetic effects in diabetic rats (Okafo et al. 2019). It has been shown that receiving *M. oleifera* fruit ethanolic extract increased serum insulin levels and reduced blood glucose levels in diabetic mice (Kumari et al. 2021).

Furthermore, receiving *M. oleifera* leaf ethanolic extract pointedly attenuated blood glucose levels, lipid peroxidation, and augmented glutathione, CAT, and SOD (Chuks et al. 2022).

A study was designed to evaluate the therapeutic efficacy of *M. oleifera* nanoparticles in mitigating insulin resistance within a pre-diabetic rat model. Following induction of pre-diabetes through high-fat diet feeding, moringa nanoparticles was administered. The intervention significantly reduced pro-inflammatory cytokines (tumor necrosis factor-alpha (TNF-α), IL-6), triglyceride levels, and HOMA-IR values across treated groups, while concurrently elevating fasting insulin. Particularly, the lowest dose restored fasting blood glucose to normoglycemic levels and achieved a marked reduction in insulin resistance indices (Rusminingsih et al. 2023).

A dose-dependent study in *Drosophila melanogaster*, including both yellow-white controls and insulin receptor heteroallelic mutants, evaluated the metabolic effects of chronic exposure to dietary moringa leaf powder. Low-dose moringa leaf powder supplementation (≤1.5%) improved glucose and carbohydrate metabolism in the

yellow-white strain and reduced lipid accumulation in the insulin receptor mutants. These doses also enhanced survival and maintained body weight. However, prolonged intake of higher insulin receptor doses (>4%) led to elevated triacylglycerides, increased weight, and reduced lifespan across both strains (Lopez-Rodriguez et al. 2023).

In a dexamethasone-induced rat model of acute and chronic insulin resistance, the alcoholic extract and ethyl acetate fraction of *M. oleifera* bark exhibited significant insulin-sensitizing effects. Through bioactivity-guided fractionation, a novel phytochemical was isolated and identified for the first time from moringa bark. Structural characterization using spectroscopic techniques revealed the active compound to be a procyanidin dimer-type polyphenol, which was responsible for restoring insulin sensitivity in the treated animal models (Sholapur et al. 2023).

In a controlled experimental study on diabetic rats, the aqueous extract of *M. oleifera* leaves was shown to mitigate insulin resistance and hepatic damage. After diabetes induction via alloxan, animals receiving the extract exhibited significantly reduced blood glucose and HOMA-IR levels compared to untreated diabetic controls. Antioxidant capacity improved markedly, with decreased lipid peroxidation and elevated enzymatic defenses (CAT, GPx, and SOD) alongside enhanced nuclear factor erythroid 2-related factor 2 (Nrf2) expression. Genes involved in insulin signaling pathways—including Akt, glucose transporter-4 (GLUT-4), insulin receptor substrate 1 (IRS1), and PI3K—were upregulated, while pro-inflammatory markers (nuclear factor-Kappa B (NF-κB), TNF-α) were suppressed. Histological findings demonstrated improved liver architecture with reduced steatosis in extract-treated rats (Hegazy et al. 2025).

In a diabetic rat model, alkali-extracted polysaccharides from *M. oleifera* seeds demonstrated significant glycemic improvements, including reduced fasting blood glucose, enhanced glucose tolerance, lowered insulin resistance, and improved insulin and lipopolysaccharides levels. The extract supplementation modulated the gut microbiome by increasing the Firmicutes/Bacteroidetes ratio, promoting beneficial strains like Lactobacillus and butyrate-producing Roseburia, and reinforcing intestinal barrier integrity. Fecal metabolomic profiling revealed the influence of the extract on multiple diabetes-associated pathways—including sphingolipid and amino acid metabolism, transient receptor potential channel modulation, and endogenous cannabinoid signaling (Yang et al. 2025).

Clinical trial

Taking *M. oleifera* leaf powder could reduce serum glucose in type 2 diabetes mellitus patients with obesity (Kumar and Mandapaka 2013). However, the findings of another clinical trial demonstrated that the administration of *M. oleifera* leaf capsules to diabetic patients had no significant effect on FBS and HbA_{1c} (Taweerutchana et al. 2017).

Some healthy subjects and Saharawi diabetics took *M. oleifera* leaf powder, and it was observed that while it had no significant effect on healthy people, it reduced postprandial glucose response peak time and mean glycemic meal response in diabetic patients (Leone et al. 2018). The administration of *M. oleifera* leaf powder to diabetic patients successfully decreased postprandial glycaemia, but it had no effect on healthy subjects (Sissoko et al. 2020). Taking *M. oleifera* leaf powder resulted in decreased FBS and HbA_{1c} in pre-diabetics (Gómez-Martínez et al. 2021) (Table 1).

Table 1. Effect of *M. oleifera* on diabetes

Type of extract/ Compound	Study design	Doses/Duration/Route of use	Results	References
		In vitro plus In vivo		
M. oleifera leaf aqueous extract	*In vitro*, Yeast cell	50–500 μg/ml, 60 min	↑Glucose tolerance and rate of glucose uptake ↓Activities of α-amylase and α-glucosidase	(Khan et al. 2017)
	In vivo, male Wistar rats and female C57BL/6 mice	100, 200 mg/kg, 3 weeks, p.o.	↓FBS	
		In vivo		
M. oleifera leaf aqueous extract	Male ICR mice	100, 200 and 300 mg/100 g body weight	-Anti-hyperglycemic activity in normal animals ↓Glucose tolerance impairment in mildly diabetic mice	(Luangpiom et al. 2013)
M. oleifera leaf aqueous extract	Albino rats	100, 200, and 300 mg/kg	↓Blood glucose levels	(Edoga et al. 2013)
M. oleifera leaf aqueous extract	Female Wistar Albino rats	250 mg/kg, 18 days, p.o.	↓Blood glucose levels, MDA amounts	(Abd El Latif et al. 2014)
M. oleifera leaf aqueous extract	Male Albino rats	200 mg/kg, 8 weeks, gastric intubation	↑GSH ↓FBS, MDA, damage of islets of Langerhans cells	(Yassa and Tohamy 2014)
M. oleifera leaf ethanolic extract	Male Albino rats	200, 400, and 800 mg/kg, a week, p.o.	↓Blood glucose levels	(Aja et al. 2015)
M. oleifera seed powder	Male Albino rats	50 and 100 mg/kg, 4 weeks, p.o.	↑Serum antioxidant enzyme ↓Lipid peroxide, IL-6, IgA, IgG, FBS, and HbA_{1c}	(Al-Malki and El Rabey 2015)
M. oleifera leaf methanolic extract	Male Wistar rats	300 and 600 mg/kg, 6 weeks, p.o.	↑Glucose tolerance, serum insulin level, glycogen amounts, glycogen synthase activities, and glucose uptake	(Olayaki et al. 2015)
M. oleifera leaf ethanolic extract and moringinine, quercetin, chlorogenic acid	Male Wistar rats	*M. oleifera* leaf ethanolic extract: 15 mg/kg, 21 days Moringinine: 3600 μmole/kg, 21 days Quercetin: 30 mg/kg, 21 days Chlorogenic acid: 10 mg/kg, 21 days	↓Blood glucose levels, MDA -Quercetin> chlorogenic acid> moringinine has antidiabetic activity	(Ali et al. 2015)
M. oleifera leaf aqueous extract	Albino mice	100 mg/kg, 14 days, p.o.	↑Total antioxidant capacity, INF-γ ↓HOMA-IR	(Tuorkey 2016)
M. oleifera seed, stem, flowers, leaves and roots ethanolic extracts	Wistar Albino Rats	100, 200, 400, and 800 mg/kg	-Seed and leaves extracts have more glucose lowering and enzyme inhibition activities ↓Blood glucose levels	(Adejoh et al. 2016)
M. oleifera leaves and callus tissues ethanolic extract	Albino mice	20%, 40% and 80%	Callus tissues ethanolic extract: ↓Blood glucose levels	(Oriabi 2016)
M. oleifera leaf aqueous extract	Wistar Albino rats	250 mg/kg, 18 days, p.o.	↑Glycogen synthase gene expression ↓Blood glucose levels	(Abd Eldaim et al. 2017)
M. oleifera different aqueous ethanol extracts	Male Sprague Dawley rats	*M. oleifera* aqueous ethanol extracts: 1,000 mg/kg, 14 days, p.o. Fractions: 500 mg/kg, p.o.	-No hypoglycemic effect on healthy rats ↓Blood glucose levels in diabetic rats	(Irfan et al. 2017)
M. oleifera leaf ethanolic extract	Male Db/ db mice	150 mg/kg, 5 weeks, p.o.	↑Insulin levels ↓FBS	(Tang et al. 2017)
M. oleifera leaf protein isolate	Conventional male mice	500 mg/kg, single dose, i.p.	-No effect on insulin secretion ↑CAT ↓Blood glucose levels, MDA	(C. Paula et al. 2017)
M. oleifera roots methanolic extract and roots powder	Wistar rats	*M. oleifera* roots powder: 15 mg/kg, 28 days, p.o. *M. oleifera* roots methanolic extract: 1 g/kg, 28 days, i.p.	↓Blood glucose levels, the amounts of serum SOD, CAT, GPx, damage of the islet of Langerhans.	(Umar et al. 2018)

Table 1. continued

M. oleifera leaf methanolic extract	Male Wister rats	250 and 500 mg/kg, 54 days, p.o.	↑Glucose tolerance, fasting plasma insulin ↓FBS	(Muzumbukilwa et al. 2019)
M. oleifera leaf methanolic extract	Male Albino Sprague Dawley rats	300 mg/kg, 60 days, p.o.	↑Plasma insulin, SOD, CAT, GPx, GRD, and GSH levels ↓Serum glucose, HbA_{1c}, TBARS, hydroperoxides	(Aju et al. 2019)
M. oleifera crude extract tablets	Rats	400 mg/kg, 5 days	↓Blood glucose level	(Okafo et al. 2019)
M. oleifera fruit ethanolic extract	Swiss albino mice	150 mg/kg, 12 weeks	↑Serum insulin level ↓Blood glucose level	(Kumari et al. 2021)
M. oleifera leaf ethanolic extract	Wistar Albino rats	250, 500 mg/kg, 21 days, p.o.	↑Glutathione, CAT, and SOD ↓Blood glucose levels, lipid peroxidation	(Chuks et al. 2022)
M. oleifera nanoparticles	*Rattus norvegicus*	75, 125, and 225 mg/kg, p.o.	↑Fasting insulin ↓TNF-α, IL-6, triglyceride, HOMA-IR, fasting blood glucose, insulin resistance indices	(Rusminingsih et al. 2023)
M. oleifera leaf powder	*Drosophila melanogaster*	0.5–5.5%, 4-5 days, p.o.	↑Glucose and carbohydrate metabolism ↓Lipid accumulation	(Lopez-Rodriguez et al. 2023)
M. oleifera leaf water extract	Female Sprague-Dawley rats	1 ml, 6 weeks, p.o.	↑Serum insulin ↓Glucose level	(El-Kady et al. 2023)
M. oleifera extract	Male Wistar rats	400 and 800 mg/kg, 4 weeks, p.o.	↓Fasting blood glucose	(Adawiyah et al. 2024)
M. oleifera leaf extract	Wistar rats	200, 400, and 800 mg/kg, 28 days, p.o.	↑Pancreatic β-cell integrity, glycogen synthesis ↓Blood glucose levels, diabetes-related hepatic and renal damage, serum urea and creatinine	(Amina et al. 2024)
M. oleifera leaf aqueous extract	Male albino Wistar rats	100 mg/kg, 30 days, p.o.	↑CAT, SOD, GPx, Nrf2 expression, GLUT-4, IRS1, PI3K, Akt ↓Insulin resistance, and hepatic damage, blood glucose, HOMA-IR, lipid peroxidation, NF-κB, TNF-α	(Hegazy et al. 2025)
M. oleifera nanoparticles	Male Sprague-Dawley rats	0.25 and 0.5 mg/kg, 28 days, p.o.	↑Insulin sensitivity, antioxidant defense ↓Hepatic inflammation	(Ahmad Tarmizi et al. 2025)
M. oleifera leaf extract	Male Wister adult albino rats	200 and 400 mg/kg	↓Fasting blood glucose, and glycated hemoglobin, adipocytokines	(Elsaadany et al. 2025)
		Clinical trial		
M. oleifera leaf powder	15 type 2 diabetes mellitus patients with obesity	50 g, 40 days, p.o.	↓Serum glucose	(Kumar and Mandapaka 2013)
M. oleifera leaf capsules	32 type 2 diabetes mellitus patients	8 g, 4 weeks, p.o.	-No significant effect on FBS and HbA_{1c}	(Taweerutchana et al. 2017)
M. oleifera leaf powder	10 healthy subjects and 17 Saharawi diabetics	20 g, p.o.	-No effect on healthy participants In diabetics: ↓Postprandial glucose response peak time, mean glycemic meal response	(Leone et al. 2018)
M. oleifera leaf powder	70 healthy non-diabetic and diabetic individuals	1, 2 g, p.o.	-No significant effect on blood glucose of healthy subjects ↓Blood glucose levels in diabetic patients	(Sissoko et al. 2020)
M. oleifera leaf powder	65 pre-diabetics	2400 mg/day, 12 weeks, p.o.	↓FBS and HbA_{1c}	(Gómez-Martínez et al. 2021)
M. oleifera leaf powder	45 Sahrawi women with type 2 diabetes	10 g, 3 months	↓HbA_{1c}, body fat	(Leone et al. 2025)

Akt: protein kinase B; CAT: catalase; FBS: fasting blood sugar; GLUT-4: glucose transporter-4; GPx: glutathione peroxidase; GRD: glutathione-reductase; GSH: reduced glutathione; HbA_{1c}: glycated hemoglobin; HOMA-IR: homeostatic model assessment for insulin resistance; Ig: immunoglobulin; IL-6: interleukin-6; INF-γ: interferon gamma; IRS1: insulin receptor substrate 1; MDA: malondialdehyde; NF-κB: nuclear factor-Kappa B; Nrf2: nuclear factor erythroid 2-related factor 2; PI3K: phosphoinositide 3-kinase; SOD: serum superoxide dismutase; TBARS: thiobarbituric acid reactive substances; TNF-α: tumor necrosis factor-alpha.

Anorther study explored the potential of *M. oleifera* leaf powder as a complementary treatment for type 2 diabetes among Sahrawi women. Conducted over three months, participants receiving oral glucose-lowering drugs were randomized into two groups: one consumed Moringa daily, while the other received no supplementation. Researchers monitored changes in fasting glucose, glycated hemoglobin, and body composition. The Moringa-supplemented group showed significant improvements in HbA_{1c} levels and reductions in body fat, while the control group saw no notable changes (Leone et al. 2025).

A growing body of multidisciplinary research underscores *M. oleifera* as a promising botanical agent for managing type 2 diabetes and insulin resistance via diverse biochemical, physiological, and gut-mediated mechanisms. Across *in vitro*, *in vivo*, and clinical investigations, *M. oleifera* exhibits consistent hypoglycemic, insulin-sensitizing, antioxidant, and anti-inflammatory effects. Its bioactive constituents—including flavonoids, polyphenols, peptides, and fibers—act synergistically to modulate key insulin signaling pathways such as PI3K/Akt, IRS1, GLUT-4, AMPK, and ERK. These actions improve glucose uptake, reduce lipid accumulation, stimulate GLP-1 secretion, and inhibit digestive enzymes like α-amylase and α-glucosidase, thereby restoring hepatic and pancreatic function.

A particularly novel insight is the ability of moringa to reshape gut microbiota composition and fecal metabolomic profiles, pointing to a gut-centric axis that complements its systemic metabolic benefits. This introduces the potential of *M. oleifera* as a prebiotic nutraceutical—a dual-action agent that addresses both metabolic dysfunction and chronic inflammation. Dose-dependent findings from Drosophila models reinforce the importance of optimizing intake levels, as excessive supplementation may induce adverse effects such as increased lipid accumulation and reduced lifespan.

Moreover, the recent isolation of a novel procyanidin dimer from Moringa bark highlights the ongoing discovery of potent phytochemicals with insulin-sensitizing capacities. Such findings open doors to targeted compound development and phytopharmaceutical innovation.

Clinical trials, however, reveal a complex narrative. While several studies report reductions in fasting blood glucose, HbA_{1c}, and postprandial glycemia in diabetic and pre-diabetic populations, others show limited efficacy—likely attributable to inconsistencies in dosage, extract standardization, duration, and interindividual variability. Notably, Moringa demonstrates minimal impact in healthy individuals, supporting its role as a disease-specific intervention rather than a general health tonic.

A useful comparative lens is provided by study (Leone et al. 2018) and study (Leone et al. 2025), both conducted in Sahrawi diabetic populations. Study (Leone et al. 2018) illustrated acute action of Moringa in reducing postprandial glucose peaks, whereas study (Leone et al. 2025) demonstrated long-term benefits with significant improvements in HbA_{1c} and body fat reduction. Collectively, these findings suggest a complementary mechanism—where Moringa operates both immediately in glycemic regulation and cumulatively in improving metabolic health over time.

The strengths of current research include mechanistic depth, diverse models, and emerging clinical data that validate the multifaceted efficacy of Moringa. The integration of omics technologies—metabolomics, microbiomics, and transcriptomics—has provided a more comprehensive view of its systemic effects. Limitations, however, persist. These include heterogeneity in study designs, lack of extract standardization, varied dosing regimens, and reliance on animal or cell

models that may not fully translate to human physiology. Contradictory findings, particularly around insulin secretion and glycemic outcomes, may stem from differences in bioavailability, compound stability, and synergistic interactions between constituents.

Looking ahead, future research on *M. oleifera* should prioritize the development of standardized, pharmacologically profiled formulations that ensure consistency in bioactive content and clinical outcomes. Long-term, placebo-controlled trials involving diverse patient populations are essential to validate its efficacy, safety, and therapeutic scope across varying metabolic states. There is a compelling need to investigate the synergistic interactions among its compounds rather than isolating individual constituents—an approach that may unveil stronger physiological effects. Moreover, integrating multi-omics strategies, including metabolomics, transcriptomics, and microbiomics, could offer a systems-level understanding of the effect of Moringa on metabolic regulation and inflammatory networks. Lastly, its role as a gut-modulating nutraceutical warrants deeper exploration, especially in relation to microbiota diversity, barrier integrity, and host-microbe signaling pathways, thereby positioning *M. oleifera* as a promising candidate in the development of precision botanical therapies for diabetes management.

Obesity

Obesity arises when energy consumption consistently exceeds energy expenditure, leading to abnormal fat accumulation in the body. Its global incidence is increasing, largely as a result of modern lifestyle shifts (Ghasemzadeh Rahbardar et al. 2025a). Obesity was redefined not just as excess body weight but as a complex, chronic illness caused by excessive adiposity. It is classified it into two stages: preclinical obesity, where excess fat is present but organs still function normally, and clinical obesity, where bodily systems begin to deteriorate due to fat-related damage. Diagnosing clinical obesity requires either clear evidence of organ or tissue dysfunction or substantial limitations in daily life activities like walking, eating, or bathing. Body mass index (BMI) is no longer realized as a reliable individual tool for diagnosis; instead, it should be paired with direct fat measurements or other anthropometric indicators like waist circumference or waist-to-height ratio. For individuals with very high BMI (over 40 kg/m^2), excess adiposity is assumed (Rubino et al. 2025).

Clinical and epidemiological research has clearly demonstrated that central obesity is a critical factor in the development of metabolic syndrome. Central obesity, defined as excess fat buildup in the abdominal area, has been recognized as a major contributor to the development of metabolic syndrome. Over the last three decades, the global incidence of obesity has increased significantly, as has the prevalence of metabolic syndrome. Obesity is often regarded as the major cause of metabolic syndrome due to its close relationship with all of the metabolic risk factors involved. Visceral fat buildup in the abdomen not only adds to the typical abdominal obesity but also causes insulin resistance, dyslipidemia, hypertension, and poor glucose metabolism (Wang et al. 2020). Moreover, obesity and abnormal lipid metabolism are associated with fat accumulation in the liver. This condition is frequently linked to oxidative stress, moderate inflammation, and increased production of adipokines and pro-inflammatory factors such as IL-6, TNF-a, and IL-1β (Kilany et al. 2020).

Besides, considerable research efforts have been dedicated to investigating strategies targeting lipid metabolism, including the modulation of lipid digestion and absorption, appetite control, and promotion of energy expenditure through adaptive thermogenesis, with the aim of managing obesity and maintaining optimal body weight. Lipid metabolism is directed

by pivotal biochemical pathways such as lipogenesis and lipolysis, which play crucial roles in regulating the synthesis of lipoproteins and tissue inflammatory responses (Jalali and Ghasemzadeh Rahbardar 2022). The PPAR-α, which is prominently expressed in brown adipose tissue and the liver, is intimately associated with lipolysis. By restraining adipocyte development and differentiation while concurrently facilitating lipolysis, PPAR-α exerts regulatory control over lipid metabolism. Adiponectin, a hormone known to promote weight loss, improve glycemic control, and enhance insulin sensitivity, is regulated by various proteins, including PPAR-γ (Tian et al. 2013). PPAR-γ serves as a crucial modulator of adiponectin gene transcription and is also implicated in the insulin signaling pathway, insulin resistance, and inflammatory processes (Sahin et al. 2013; Tian et al. 2013).

On the other hand, the relationship between GLUT-4 and phosphorylated AMPK (p-AMPK) in obesity has been a subject of investigation. GLUT-4 is a key glucose transporter protein primarily found in adipose tissue and skeletal muscle cells. It plays a critical role in facilitating glucose uptake into these cells in response to insulin signaling. Impaired GLUT-4 function or expression has been associated with insulin resistance and obesity-related metabolic disorders (Maria et al. 2015). p-AMPK is an enzyme that acts as a cellular energy sensor, regulating energy homeostasis in various tissues. It is activated in response to low cellular energy levels, such as during exercise or calorie restriction. Activation of p-AMPK promotes glucose uptake and utilization, fatty acid oxidation, and inhibits anabolic processes, thus promoting energy expenditure and metabolic adaptation (Habegger et al. 2012; Jeon 2016; Turdi et al. 2011). In the context of obesity, there is evidence to suggest that dysregulation of GLUT-4 and p-AMPK signaling pathways may contribute to the development of insulin resistance and metabolic dysfunction (Mackenzie and Watt 2016; Pandey et al. 2019).

Pharmacological options for obesity management now include GLP-1 receptor agonists (e.g., semaglutide, liraglutide), naltrexone-bupropion, orlistat, and combinations like phentermine-topiramate (Atlas et al. 2023; Tchang et al. 2024). These agents promote weight loss by targeting appetite regulation and metabolic rate, yet long-term adherence, cost-related barriers, and recurrence of weight gain upon discontinuation remain significant concerns. Considering the multifactorial nature of obesity, there is growing interest in safe, natural alternatives such as *M. oleifera*, which may offer fat-metabolism-enhancing effects with a lower side-effect burden.

Numerous studies have specifically highlighted the anti-obesity properties of *M. oleifera* extracts and seeds.

In vitro

Treating 3T3-L1 adipocytes with *M. oleifera* leaf extract and iso-quercetin resulted in anti-proliferative activity. They downregulated the expression of genes associated with adipogenesis, leading to a reduction in triglyceride accumulation. Additionally, *M. oleifera* leaf extract induced apoptosis in adipocyte cells. Treatment with *M. oleifera* leaf extract upregulated Bcl-2-associated X protein (Bax) and downregulated B-cell lymphoma 2 (Bcl-2), resulting in increased caspase-3 activity, indicating apoptosis of adipocyte cells (Balusamy et al. 2019). In another *in vitro* study, the effects of *M. oleifera* leaf ethanolic extract and its fractions were examined on 3T3-L1 cells. The findings indicated that fractionated ethanol extracts of *M. oleifera* leaves had greater antioxidant and anti-obesity properties than water extracts. Moreover, while the expression of C/EBPα in 3T3-L1 cell differentiation did not exhibit a concentration-dependent inhibitory effect with *M. oleifera* leaf extracts, the expression of PPAR-γ, Fas cell surface

death receptor (FAS), and acetyl-coenzyme A carboxylase (ACC) was found to be inhibited in a concentration-dependent manner (Kim et al. 2020). Exposing 3T3-L1 cells to rutin (quercetin-3-O-rutinoside) derived from *M. oleifera* leaves caused an enhancement in the release of glycerol, glucose uptake, adiponectin, and p-AMPK amounts, as well as an increase in the expression of GLUT-4 and uncoupling protein-1 (UCP-1). It also attenuated the amounts of α-glucosidase, pancreatic lipase, lipid and leptin levels, adipogenesis, besides the expression of PPAR-γ (Ganjayi et al. 2023) (Table 2). Figure 1 depicts the proposed down-regulation mechanism of adipogenic transcription factors by *M. oleifera*.

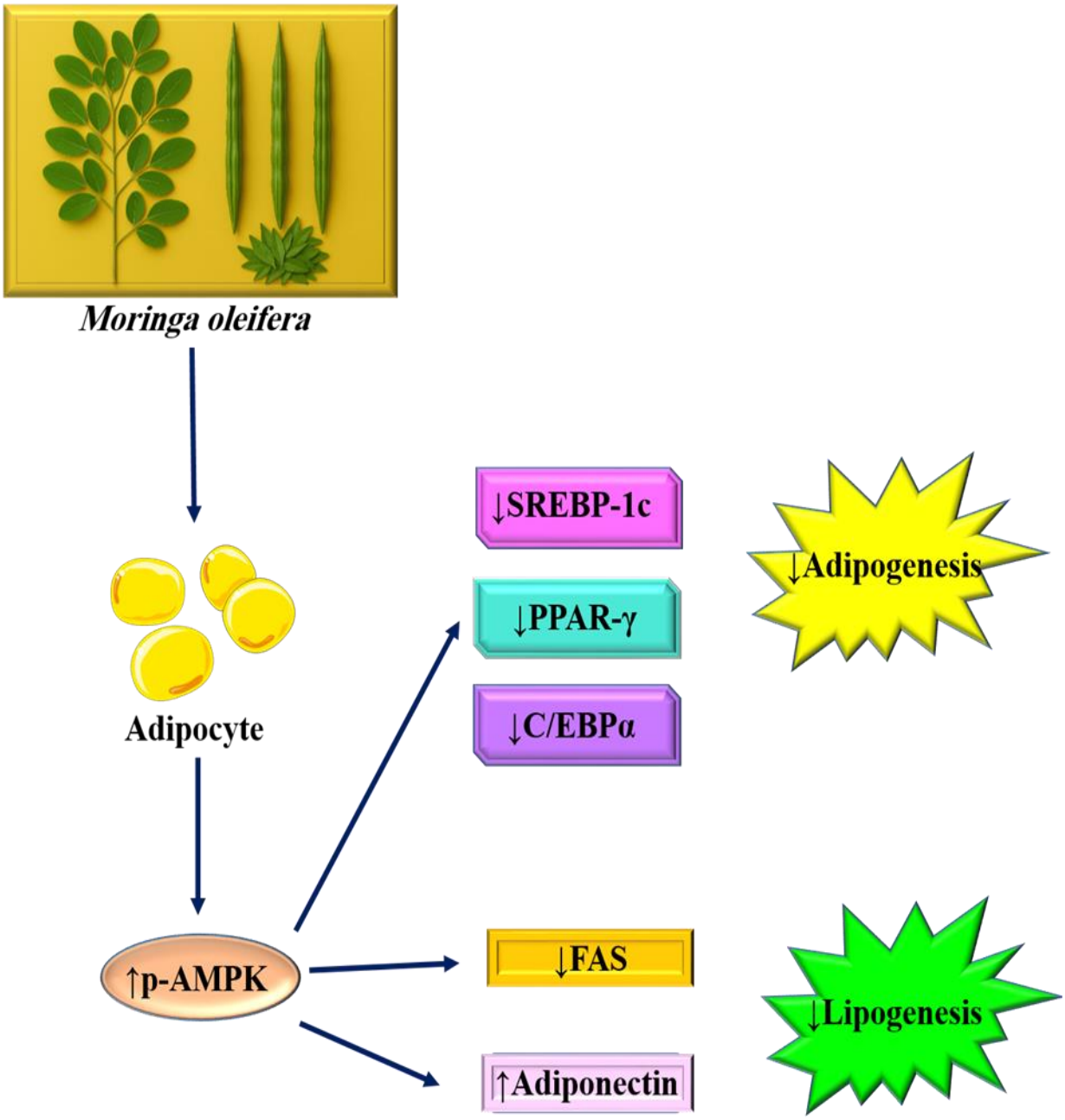

Figure 1. The suggested down-regulation mechanism of adipogenic transcription factors by *M. oleifera* and its main constituents**.** Abbreviations: C/EBPα: CCAAT/enhancer-binding protein alpha; FAS: fatty acid synthase; p-AMPK: phosphorylated AMP-activated protein kinase; PPAR-γ: peroxisome proliferator-activated receptor gamma; SREBP-1c: sterol regulatory element-binding protein-1c.

Table 2. Effect of *M. oleifera* on obesity

Type of extract/ Compound	Study design	Doses/Duration/Route of use	Results	References
		In vitro		
M. oleifera leaf extract and iso-quercetin	3T3-L1 adipocytes	*M. oleifera* leaf extract: 0–2000 µg/ml, 72 hr	*M. oleifera* leaf extract: ↑Anti-proliferative activity, apoptosis, Bax and caspase-3 levels ↓Adipogenesis related genes expression, triglyceride accumulation, and Bcl-2 levels	(Balusamy et al. 2019)
		Iso-quercetin: 0–20 µg/ml, 72 hr	Iso-quercetin: ↑Anti-proliferative activity ↓Adipogenesis related genes expression, triglyceride accumulation	
M. oleifera leaf ethanolic extract and its fractions	3T3-L1		↓PPAR-γ, FAS, and ACC expression	(Kim et al. 2020)
Rutin (quercetin-3-O-rutinoside) derived from *M. oleifera* leaf	3T3-L1	10–160 µg/ml, 24 or 48 hr	↑Release of glycerol, glucose uptake, adiponectin and p-AMPK amounts, expression of GLUT-4 and UCP-1 ↓α-glucosidase and pancreatic lipase, lipid and leptin levels, adipogenesis, expression of PPAR-γ	(Ganjayi et al. 2023)
		In vivo		
M. oleifera leaf ethanolic extract	Wistar rats	300 and 600 mg/kg, 14 days by oropharyngeal cannula	↓Weight gain in female rats	(Atsukwei et al. 2014)
M. oleifera leaf ethanolic extract	Male Sprague Dawley rats	200 mg/kg, 8 weeks	↑Adiponectin ↓Body weight, TNF-α, and leptin	(Daba et al. 2015)
Isothiocyanate-rich *M. oleifera* extract	Male C57BL/6J mice	5% *M. oleifera* (delivering 66 mg/kg of isothiocyanate), 3 months, p.o.	↓Weight gain, plasma levels of leptin, resistin, cholesterol, IL-1β, and TNF-α	(Waterman et al. 2015)
Table 2 continued				
M. oleifera leaf powder	Male long Evans rats	50 mg/day, 35 days, p.o.	↓Food intake and BMI	(Nahar et al. 2016)
Afya tea® (Aqueous extract of *M. oleifera*)	Wistar albino rats	84.6 g/kg, 14 days, p.o.	↓Body weight, food and water intake	(Zofou et al. 2017)
M. oleifera leaf powder	Male Wistar rats	700 mg/kg, 3 weeks, p.o.	↓Abdominal circumference	(López et al. 2018)
M. oleifera leaf aqueous extract	Swiss Albino mice	200 mg/kg, 3 months, p.o.	↓Body weight, serum level of IL-6	(Elabd et al. 2018)
M. oleifera seed oil extract	Male Sprague Dawley rats	800 mg/kg, 8 weeks, p.o.	↑Activities of total antioxidant capacity, SOD and CAT ↓Final body weights and weight gain, food consumption, leptin, resistin, and HFABP, serum levels of IL-6, IL-1β and TNF-α, MDA level	(Kilany et al. 2020)
M. oleifera leaf ethanolic (70%) extract	Male Albino rats	200 and 400 mg/kg, a month, p.o.	↑Expression of adiponectin, omentin and GLUT-4, mRNA expression of MC4R and PPAR-α ↓Final weights, leptin, vaspin, FAS, and HMG-CoA reductase	(Ezzat et al. 2020)
M. oleifera leaf extracts	Male Sprague Dawley rats	30 days, 1000 mg/kg, p.o.	↓BMI, abdominal fat, and MCP-1	(Irfan et al. 2022)
M. oleifera fresh leaf aqueous ultrasound assisted extract and *M. oleifera* dried leaf ethanolic extract	Swiss Albino mice	500 mg/kg, 7 days	↑Body weight	(Sadat et al. 2022)
M. oleifera leaf/seed methanolic extract	Male Swiss albino mice	500 mg/kg, 1 and 3 months, p.o.	↓Diabetic weight loss	(Aljazzaf et al. 2023)
M. oleifera aqueous extract	Male albino rats	5%, 28 days, p.o.	↑HDL ↓Body weight, total cholesterol, triglycerides, and LDL	(Bakr et al. 2024)
M. oleifera methanolic extract	Rats	250 and 500 mg/kg, p.o.	↑ IL-10, IL-6, COX-2 ↓Body weight, liver weight, plasma glucose levels, IL-1β, TNF	(Ibrahim et al. 2024)

Table 2. Continued

M. oleifera root extract	Male Wistar rats	200 and 400 mg/kg, 5 weeks	↓Body weight, organ weights, and Lee index, liver histopathology ↓Serum triglycerides, total cholesterol	(Hardjo et al. 2025)
		Clinical trial		
M. oleifera leaf ethanolic (70%) extract ethanolic (70%) extract	15 female overweight or obese participants	400 mg, 8 weeks, p.o.	↓BMI, total cholesterol, and LDL	(Ezzat et al. 2020)
M. oleifera leaf extract	30 obese individuals	2 sachets, 30 days, p.o.	↓Body weight	(Patel et al. 2023)
M. oleifera powder	60 hyperlipidemic patients	1g, twice a day, 45 days, p.o.	- Improved BMI and lipid profile	(Sarfraz et al. 2023)
M. oleifera powder	30 obese women with polycystic ovarian syndrome	5 grams, 3 months, p.o.	↓Body weight	(Muthukumar and Sultana 2024)
M. oleifera leaf powder	40 overweight, hyperlipidemic adults aged 30-60	0.5 g, 12 weeks, p.o.	↑HDL-C ↓Body weight, BMI, waist circumference, blood pressure, triglycerides, LDL cholesterol levels, consumption of carbohydrates, energy, cholesterol	(Munir et al. 2025)

ACC: acetyl- coenzyme A carboxylase; Bax: Bcl-2-associated X protein; Bcl-2: B-cell lymphoma 2; BMI: body mass index; CAT: catalase; COX-2: cyclooxygenase-2; FAS: Fas cell surface death receptor; GLUT-4: glucose transporter-4; HDL-C: high-density lipoprotein cholesterol; HFABP: heart fatty acid binding protein; HMG-CoA: 3-Hydroxy 3-methylglutaryl- coenzyme A; IL: interleukin; LDL: low-density lipoprotein; MC4R: melanocortin-4 receptor; MCP-1: monocyte chemoattractant protein-1; mRNA: messenger ribonucleic acid; p-AMPK: phosphorylated adenosine monophosphate-activated protein kinase; PPAR: peroxisome proliferator–activated receptor; SOD: superoxide dismutase; TNF-α: tumor necrosis factor-alpha; UCP-1: uncoupling protein-1.

In vivo

In hypercholesterolemic rats, the supplementation of *M. oleifera* leaf ethanolic extract could result in weight loss or inhibition of weight gain in female animals, but this effect was not significant in male rats (Atsukwei et al. 2014). The administration of *M. oleifera* leaf ethanolic extract to rats with a high-fat diet attenuated their body weight and the amounts of TNF-α, and leptin. The extract also increased adiponectin levels (Daba et al. 2015). Mixing isothiocyanate-rich *M. ole*ifera extract with the diet of mice on a very high-fat diet reduced weight gain and plasma levels of leptin, resistin, cholesterol, IL-1β, and TNF-α (Waterman et al. 2015). It has been shown that supplementing obese rats with *M. oleifera* leaf powder attenuated their food intake and BMI (Nahar et al. 2016). Receiving Afya tea® (aqueous extract of *M. oleifera*) in diabetic rats pointedly decreased body weight, food and water intake (Zofou et al. 2017). The administration of *M. oleifera* leaf powder to rats with metabolic syndrome pointedly decreased their abdominal circumference (López et al. 2018). Likewise, the supplementation of *M. oleifera* leaf aqueous extract with mice on a high-fat diet lowered their body weight and serum levels of IL-6 (Elabd et al. 2018). It has been observed that the oral administration of *M. oleifera* seed oil extract to obese rats with a high-fat diet could successfully increase the activities of total antioxidant capacity, SOD, and CAT. It also decreased final body weights and weight gain, food consumption, leptin, resistin, and heart fatty acid binding protein (HFABP), serum levels of IL-6, IL-1β, TNF-α, and malondialdehyde (MDA) amounts (Kilany et al. 2020). Supplementing *M. oleifera* leaf ethanolic extract in obese rats resulted in enhanced expression of adiponectin, omentin, and GLUT-4, messenger ribonucleic acid (mRNA) expression of melanocortin-4 receptor (MC4R), and PPAR-α. Meanwhile, it reduced final weights, and the amounts of leptin, visceral adipose tissue-derived serine protease inhibitor (vaspin), FAS, and 3-Hydroxy 3-methylglutaryl-coenzyme A (HMG-CoA) reductase (Ezzat et al. 2020). Similarly, it

has been shown that *M. oleifera* leaf extracts administration to rats with high-fat diet caused a decrease in BMI, abdominal fat, and monocyte chemoattractant protein-1 (MCP-1) (Irfan et al. 2022). It has been demonstrated that *M. oleifera* fresh leaf aqueous ultrasound-assisted extract and *M. oleifera* dried leaf ethanolic extract could increase the weight of diabetic mice (Sadat et al. 2022). The administration of *M. oleifera* leaf and/or seed methanolic extract to diabetic mice was shown to prevent the weight loss induced by alloxan (Aljazzaf et al. 2023).

An experimental study evaluated the antioxidant and anti-obesity properties of *M. oleifera* root extract in obese rats. Obesity was induced using a high-fat diet, followed by treatment with *M. oleifera* root extract. It significantly reduced body weight, organ weights, and Lee index, and improved liver histopathology (Hardjo et al. 2025).

Clinical trial

Taking *M. oleifera* leaf ethanolic extract for 8 weeks reduced BMI in overweight and obese women (Ezzat et al. 2020). Drinking *M. oleifera* leaf extract as an herbal tea twice a day could reduce body weight in obese individuals (Patel et al. 2023).

In a randomized controlled trial investigating lipid-lowering strategies in hyperlipidemic patients, *M. oleifera* was evaluated individually and in combination alongside atorvastatin therapy. Participants were randomly assigned to different groups: one receiving atorvastatin and placebo, and anther supplemented with *M. oleifera* in addition to atorvastatin. The group supplemented with *M. oleifera* showed statistically significant improvements in BMI and lipid profile (Sarfraz et al. 2023).

A randomized controlled clinical trial explored the effects of moringa leaf powder on overweight, hyperlipidemic adults aged 30 to 60. Participants received either moringa capsules or placebo (corn starch), coupled with moderate physical activity over 12 weeks. Results showed that moringa supplementation led to significant reductions in body weight, BMI, and waist circumference. Dietary intake analysis also revealed decreased consumption of carbohydrates, energy, and cholesterol in the moringa group (Munir et al. 2025).

The collective evidence from *in vitro*, *in vivo*, and clinical studies clearly demonstrates that *M. oleifera* exerts a multifaceted protective role in obesity management (Figure 2). At the molecular level, *M. oleifera* leaf extract and its bioactive compounds inhibit adipogenesis by downregulating transcription factors such as PPAR-γ, ACC, and FAS. These factors are instrumental in fat cell differentiation and lipid accumulation, and their suppression leads to reduced triglyceride storage and enhanced adipocyte apoptosis—particularly through Bax/Bcl-2 modulation and increased caspase-3 activity. *M. oleifera* also boosts metabolic efficiency by upregulating GLUT-4 and UCP-1 expression, promoting glucose uptake and thermogenesis. Rutin and iso-quercetin further enhance these effects by activating AMPK and increasing adiponectin secretion, supporting insulin sensitivity and fat metabolism.

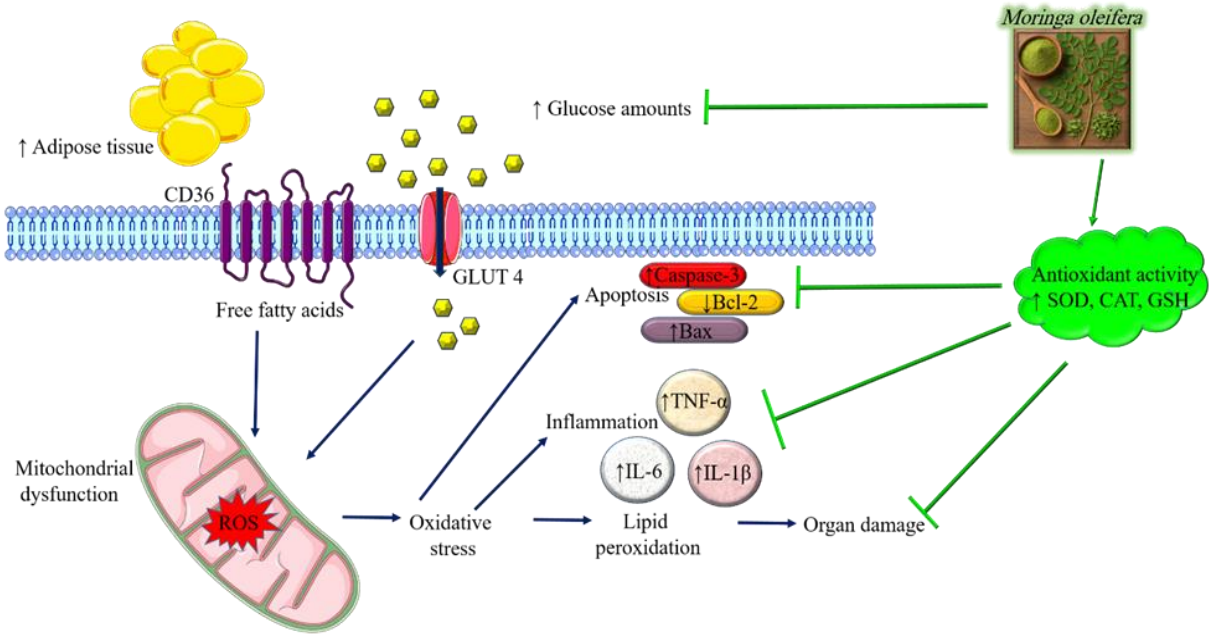

Figure 2. The proposed mechanism of anti-obesity action of *M. oleifera* and its main constituents. Abbreviations: Bax: Bcl-2-associated X protein; Bcl-2: B-cell lymphoma 2; CAT: catalase; CD36: cluster of differentiation 36; GSH: glutathione; GLUT4: glucose transporter4; IL-1β: interleukin 1 beta; IL-6: interleukin 6; ROS: reactive oxygen species; SOD: superoxide dismutase; TNF-α: tumor necrosis factor-alpha.

In animal models, *M. oleifera* consistently demonstrates anti-obesity effects regardless of the extract type—leaf, seed, or root—and preparation method (aqueous, ethanolic, methanolic, or ultrasound-assisted). It reduces body weight, BMI, visceral fat, and inflammatory markers like IL-1β, IL-6, TNF-α, and leptin, while improving antioxidant enzyme activity and adipokine profiles. *M. oleifera* supplementation also improves behavioral and neuroprotective parameters such as physical activity levels and anxiety-like behaviors, suggesting that its influence may extend to central nervous system regulation. Clinically, *M. oleifera* shows promising potential in reducing body weight and waist circumference in overweight individuals and those with polycystic ovary syndrome, while enhancing hemoglobin levels and reducing carbohydrate and cholesterol intake—highlighting its suitability as both a therapeutic agent and a nutrient-dense dietary alternative.

One novel proposition is that *M. oleifera* functions as a botanical metabolic modulator, capable of reprogramming adipose tissue behavior and energy homeostasis across multiple biological systems. Its ability to balance inflammation, oxidative stress, glucose metabolism, and lipid regulation concurrently makes it a strong candidate for incorporation into integrative obesity therapies. However, despite the strengths of diverse experimental designs, consistent outcomes across animal and human trials, and the inclusion of molecular insights, limitations persist. Many human studies are short-term with small sample sizes, and sex-dependent variations observed in some rodent studies—such as reduced efficacy in males—suggest a need for further exploration into hormonal interactions.

Contradictions in findings, such as occasional weight gain in diabetic mice treated with certain *M. oleifera* extracts, may stem from differences in extract concentrations, preparation methods, or compensatory feeding behavior due to improved metabolism. These inconsistencies highlight the importance of standardizing extract formulations and treatment protocols in future investigations. Additionally, while anti-obesity effects of

M. oleifera are well supported, its long-term safety profile, interaction with conventional drugs, and influence on gut microbiota remain underexplored.

Building on this comprehensive discussion, future research should prioritize exploring the additive and synergistic effects of *M. oleifera* when combined with conventional pharmacological agents or other herbal interventions. Notably, its co-administration with statins or bioactives such as *Allium sativum* has already shown enhanced lipid-lowering and metabolic outcomes in clinical settings (Sarfraz et al. 2023). Investigating such combinations may reveal whether *M. oleifera* can potentiate therapeutic efficacy, reduce required drug dosages, or improve safety profiles in obesity management. Additionally, longitudinal studies should aim to assess sustained outcomes, clarify sex-specific metabolic responses, and establish standardized dosing across varied extract types. Integrating Moringa into polyherbal formulations or personalized nutrition strategies may unlock broader applications for mitigating obesity and its systemic complications.

Dyslipidemia

Dyslipidemia refers to abnormal blood lipid profiles, characterized by increased concentrations of total cholesterol (≥200 mg/dl), low-density lipoprotein cholesterol (LDL-C ≥130 mg/dl), very low-density lipoprotein cholesterol (VLDL-C ≥30 mg/dl), and triglycerides (≥150 mg/dl), along with reduced levels of HDL-C (<40 mg/dl in men and <50 mg/dl in women). This lipid imbalance is a key contributor to the onset and progression of metabolic syndrome (Ghasemzadeh Rahbardar et al. 2025c). This disorder can be caused by metabolic abnormalities as well as an improper lifestyle and nutrition. Atherosclerotic heart disease, atherosclerosis, and coronary atherosclerosis are all risk factors for hypercholesterolemia and hyperlipidemia. Elevated blood cholesterol and LDL levels are especially linked to these illnesses and can lead to plaque buildup in the arteries, decreasing blood flow to the heart and raising the risk of heart attack and stroke (Jalali and Ghasemzadeh Rahbardar 2022). It has been observed that dyslipidemia is associated with inflammation and altered gene expression in various cells and tissues. One aspect of this association involves the release of certain pro-inflammatory cytokines, such TNF-α and IL-1β, by immune cells and adipose tissue. These cytokines can contribute to the development of dyslipidemia by promoting inflammation and insulin resistance, which can lead to impaired lipid metabolism and dysregulation of lipid homeostasis (Nicolaiciuc et al. 2017; Wang et al. 2017). Moreover, in the presence of dyslipidemia and inflammation, the expression of PPAR-γ may be altered. PPAR-γ downregulation has been linked to some dyslipidemia-related conditions such as obesity and insulin resistance (Xie et al. 2020; Youssef et al. 2019).

Besides, SREBP-1c is a transcription factor that regulates the expression of genes involved in fatty acid and triglyceride synthesis (Pathak and Chiang 2019; Zhang et al. 2022). Dyslipidemia and inflammation can influence the expression of SREBP-1c, leading to increased lipogenesis and triglyceride accumulation (Li et al. 2022). Alpha subunit-like effector A (Cidea) and alpha subunit-like effector c (Cidec) are genes that are implicated in lipid metabolism and adipocyte differentiation (Zhao et al. 2020). Dysregulation of Cidea and Cidec expression has been observed in conditions such as obesity and dyslipidemia (Li et al. 2022).

Standard lipid-lowering therapies include statins, which remain the primary agents for managing dyslipidemia, along with ezetimibe, fibrates, omega-3 fatty acids, and newer options like proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors (Chhetry and Jialal 2019; de Castro 2020; Taher et al. 2024). Despite

their widespread use, challenges such as statin intolerance, limited triglyceride-lowering efficacy, and persistent cardiovascular risk in treated individuals underscore the need for alternative therapeutic approaches. Consequently, bioactive-rich plants such as *M. oleifera* are being investigated for their potential lipid-modulating properties as adjuncts or substitutes to conventional pharmacotherapy.

The investigations on the effects of *M. oleifera* on dyslipidemia are discussed further below.

In vitro

Treating HepG2 cells with a phenolic-enriched extract of *M. oleifera* leaf reduced total intracellular cholesterol and HMG-CoA reductase activity. It also enhanced LDL receptor binding activity as well as the expressions of HMG-CoA reductase and LDL receptor (Tabboon et al. 2016). It has also been shown that treating 3T3-L1 cells with *M. oleifera* leaf ethanolic extract reduced α-glucosidase and pancreatic lipase, as well as TC, TG, and LDL-C amounts. Besides, it increased HDL-C levels (Chen et al. 2020).

Studies containing both *in vitro* and *in vivo* parts

Research was conducted to evaluate the optimal combination of the cholesterol-lowering capabilities of a blend of *M. oleifera* leaf and fruit methanol extracts. The obtained data revealed that the 1:1 mixture of *M. oleifera* leaf and fruit extracts had the best results for suppressing lipase *in vitro*. Therefore, this mixture demonstrated a notable decrease in the total levels of serum total cholesterol in the *in vivo* part of the study. Furthermore, in a model replicating a high-cholesterol diet, the extract mixture reduced blood triglyceride levels significantly (Gururaja G M et al. 2016) (Table 3).

Table 3. Effect of *M. oleifera* on dyslipidemia

Type of extract/ Compound	Study design	Doses/Duration/Route of use	Results	References
		In vitro		
Phenolics-enriched extract of *M. oleifera* leaf	HepG2 cells	0, 25, 50, 100, 200, 400 µg/ml	↑LDL receptor binding activity, expressions of HMG-CoA reductase and LDL receptor ↓Total intracellular cholesterol, HMG-CoA reductase activity	(Tabboon et al. 2016)
M. oleifera leaf ethanolic (90%) extract	3T3-L1 cells	7.5, 11.0, and 14.5 g/ml, 48 hr	↑ HDL-C ↓α-glucosidase and pancreatic lipase, total cholesterol, triglycerides, and LDL-C	(Chen et al. 2020)
		In vitro* plus *in vivo		
M. oleifera leaf and fruit methanolic extracts	*In vitro*	5, 10, 25, and 50 µg/ml	- The 1:1 proportion was the best in inhibiting lipase	(Gururaja G M et al. 2016)
	In vivo, male Albino Wistar rats	22.5, 45, and 90 mg/kg, 42 days, p.o.	↓Serum total cholesterol and triglycerides	
		In vivo		
M. oleifera leaf aqueous extract	Male Wistar Albino rats	400 mg/kg, 28 days, p.o.	↓Plasma cholesterol, triglycerides, and LDL level	(Oyedepo et al. 2013)
M. oleifera leaf methanolic extract	Male and female Wistar Albino rats	200 and 400 mg/kg, 3 weeks, p.o.	↑HDL level ↓Total cholesterol, triglycerides, LDL, and VLDL levels	(Bais et al. 2014)
M. oleifera leaf ethanolic extract	Male Wistar Albino rats	100, 200, and 300 mg/kg, 10 days, p.o.	↓Serum cholesterol	(Sule and Arhoghro 2016)
M. oleifera leaf powder or its ethanolic extract	Male Albino rats	Leaf powder: 0.737% or 1.475% of the diet Leaf extract: 200 or 400 mg/kg, 60 days, p.o.	↓Cholesterol, triglycerides, and LDL-C	(Helmy et al. 2017)
M. oleifera leaf methanolic extract	Male Wistar rats	250 mg/kg, 42 days, p.o.	↓Cholesterol, LDL	(Omodanisi et al. 2017)

Table 3. Continued

M. oleifera leaf powder	Male Wistar Albino rats	5, 10, and 20% of diets, 28 days	↑HDL-C ↓Total cholesterol	(Asogwa 2017)
M. oleifera seed powder	Male rats	50, 100 mg/kg	- Amended the lipid profile	(Rabey et al. 2017)
M. oleifera leaf	Male Albino rats	5%, 10%, and 15% of diet, 6 weeks, p.o.	↑ Serum HDL-C ↓Serum cholesterol, triglycerides, LDL-C, VLDL-C	(Bushuty and Shanshan 2020)
M. oleifera leaf methanolic extract	Female Sprague Dawley rats	400 mg/kg, 10 weeks, p.o.	-No effect on hypertriglyceridemia ↓ Hepatic lipid stores	(Muhammad et al. 2020)
M. oleifera polysaccharides	Male C57BL/6J mice	100, 200, and 400 mg/kg, 12 weeks, p.o.	↑HDL amounts, expression of PPAR-α, Fiaf, Cyp7a1, and Cyp7b1 ↓Lipid accumulation, total cholesterol, triglycerides, and LDL, serum TNF-α and IL-1β levels, expression of PPAR-γ, SREBP-1c, Cidea, and Cidec genes	(Li et al. 2022)
M. oleifera leaf	Male Albino Sprague-Dawley rats	10% of diet, 6 weeks, p.o.	↑HDL amounts ↓Total cholesterol, total triglyceride, and LDL levels	(Alqurashi and Alholaibi 2023)
M. oleifera leaf alcoholic extract	Male Wistar rats	300 mg/kg, 6 weeks, gavage	↑HDL ↓Body weight, serum levels of total cholesterol, triglycerides, LDL	(abaza 2024)
M. oleifera leaf ethanolic extract	Male rats	250, 500, and 750 mg/kg, 30 days	↑HDL ↓Total cholesterol, triglycerides, LDL-C	(Himi et al. 2024)
M. oleifera leaf powder	Golden Misri Chickens	1.5%, 4 weeks, p.o.	↑HDL-, triglycerides, hemoglobin, red blood cell counts ↓LDL-C, total cholesterol, white blood cells	(Saleem et al. 2024)
M. oleifera leaf powder	Male Wistar rats	500 mg/kg, 4 weeks	↑HDL-C ↓Total cholesterol	(Ariestiningsih et al. 2024)
M. oleifera leaf extract	Rats	0.2 mg/kg, orally	↑HDL, antioxidant defense ↓Fasting blood glucose, HbA_{1c}, total cholesterol, triglycerides, LDL-C	(Al Khuzaee et al. 2025)
		Clinical trial		
M. oleifera leaf capsule	79 participants with LDL> 2.6 mmol/l	2100 mg, 30 days, p.o.	-No effect on LDL amounts	(Sandoval and Jimeno 2013)
M. oleifera leaf powder	20 free-living diabetic males at the early stages of type 2 diabetes	20 g, 3 months, p.o.	↑HDL levels ↓Serum triglyceride, total cholesterol, and LDL levels	(Tollo et al. 2016)
M. oleifera leaf powder	3 young men with hypercholesterolemia	10 g, 4 weeks, p.o.	-No effect on lipid profile	(JUAN 2021)
M. oleifera leaf and/or seeds	Hyperlipidemic patients		Leaves and leaves-seeds: ↓Total cholesterol, LDL-C, triglycerides All groups: ↑ HDL	(Ilyas et al. 2023)
M. oleifera tea	25 patients with coexisting type 2 diabetes and hypertension	14 days, p.o.	↑Serum HDL, ↓LDL, triglycerides, and total cholesterol, hepatic enzyme activities, albumin, total protein, bilirubin fractions	(Nurudeen et al. 2023)
M. oleifera leaf powder	44 Algerian diabetic patients	3.6 g, twice daily, 90 days	↑ HDL-C ↓HbA1c, LDL-C	(Henouda et al. 2023)

Cidea: cell death inducing DNA fragmentation factor, alpha subunit-like effector A; Cidec: cell death-inducing DNA fragmentation factor, alpha subunit-like effector c; Cyp7a1: cytochrome P450 family 7 subfamily A member 1; Cyp7b1: cytochrome P450 family 7 subfamily B member 1; HDL: high-density lipoprotein; HMG-CoA: 3-Hydroxy 3-methylglutaryl- coenzyme A; IL-1β: interleukin-1β; LDL: low-density lipoprotein; PPAR: peroxisome proliferator–activated receptor; SREBP-1c: sterol regulatory element-binding protein-1c gene; TNF-α: tumor necrosis factor-alpha; VLDL: very low-density lipoprotein.

In vivo

The oral administration of *M. oleifera* leaf aqueous extract to diabetic rats could significantly decrease the plasma levels of cholesterol, triglycerides, and LDL (Oyedepo et al. 2013). Receiving *M. oleifera* leaf methanolic extract pointedly ameliorates the lipid profile of rats with a high-fat diet by attenuating total cholesterol, triglycerides, LDL, and VLDL levels, besides increasing HLD amounts (Bais et al. 2014). The administration of *M. oleifera* leaf ethanolic extract to high-fat diet rats resulted in decreased amounts of serum cholesterol (Sule and Arhoghro 2016). The supplementation of *M. oleifera* leaf powder or its ethanolic extract in hypercholesterolemic rats caused a reduction in serum cholesterol, triglycerides, and LDL-C amounts (Helmy et al. 2017). Likewise, the oral administration of *M. oleifera* leaf methanolic extract to diabetic rats resulted in decreased levels of LDL and cholesterol (Omodanisi et al. 2017). *M. oleifera* leaf powder supplementation in rats with a high-fat diet increased HDL-C amounts and attenuated total cholesterol (Asogwa 2017). Adding *M. oleifera* leaves to the diet of hypercholesterolemic rats increased serum HDL-C and reduced serum cholesterol, triglycerides, LDL-C, and VLDL-C (Bushuty and Shanshan 2020). It has been observed that oral administration of *M. oleifera* leaf methanolic extract to female rats with a high fructose diet could remarkably reduce their hepatic lipid stores, but it had no significant effect on hypertriglyceridemia (Muhammad et al. 2020). Supplementing *M. oleifera* polysaccharides to mice with a high-fat diet resulted in increased serum HDL amounts, as well as augmented expression of PPAR-α, Fiaf, cytochrome P450 family 7 subfamily A member 1 (Cyp7a1), and cytochrome P450 family 7 subfamily B member 1 (Cyp7b1). The findings also indicated that *M. oleifera* polysaccharides declined lipid accumulation, total cholesterol, triglycerides, and LDL, serum TNF-α and IL-1β levels, expression of PPAR-γ, SREBP-1c, cell death-inducing DNA fragmentation factor, alpha subunit-like effector A (Cidea), and cell death-inducing DNA fragmentation factor, alpha subunit-like effector c (Cidec) genes (Li et al. 2022). Mixing *M. oleifera* leaf in the high-fat diet of rats augmented serum HDL amounts and also lowered total cholesterol, total triglyceride, and LDL levels (Alqurashi and Alholaibi 2023).

The synergistic effect of *M. oleifera* extract and simvastatin on obesity and dyslipidemia in rats fed a high-fat diet. Over ten weeks, rats treated with both M. oleifera and simvastatin showed the most substantial improvements in body weight reduction and lipid profiles compared to control groups. The combined treatment significantly lowered serum levels of total cholesterol, triglycerides, and LDL, while raising HDL levels (abaza 2024).

Another study evaluated the impact of dietary supplementation with *M. oleifera* leaf powder in chicks following a high-fat diet. Specifically, a 1.5% inclusion of moringa powder led to significant increases in body weight, HDL-C, triglycerides, hemoglobin, and red blood cell counts. Simultaneously, there were notable decreases in LDL-C, total cholesterol, white blood cells, and several hematological parameters (Saleem et al. 2024).

Clinical trial

Taking *M. oleifera* leaf capsules in individuals with LDL> 2.6 mmol/L reduced LDL levels slightly but not significantly compared to the placebo group (Sandoval and Jimeno 2013). Adding *M. oleifera* leaf powder to the diet of individuals in the early stages of type 2 diabetes successfully attenuated serum triglyceride, total cholesterol, and LDL levels while enhancing HDL amounts (Tollo et al. 2016). The administration of *M. oleifera* leaf powder to young men with hypercholesterolemia revealed no significant effect on the serum lipid profile

(JUAN 2021). A clinical trial aimed to examine the ameliorative effect of *M. oleifera* seeds and/or leaf-supplemented cookies on the lipid profile of hyperlipidemic patients. The findings indicated that leaves and leaf-seeds cookies could reduce total cholesterol, LDL-C, and triglycerides in patients. Besides, all kinds of *M. oleifera* cookies enhanced HDL levels (Ilyas et al. 2023).

A pilot clinical study evaluated the long-term effects of *M. oleifera* leaf powder on lipid and carbohydrate profiles in Algerian diabetic patients. The intervention led to statistically significant improvements in glycemic control and lipid parameters, specifically reductions in LDL-C, and an increase in HDL-C levels (Henouda et al. 2023).

M. oleifera has emerged as a promising botanical agent in the modulation of dyslipidemia through a diverse array of protective mechanisms. Broadly, its lipid-lowering effects appear to stem from its ability to inhibit key digestive enzymes such as pancreatic lipase and α-glucosidase, which reduces the absorption of dietary fats. At the cellular level, moringa has demonstrated the capacity to regulate cholesterol biosynthesis by suppressing HMG-CoA reductase activity, while simultaneously enhancing LDL receptor expression and binding affinity. This dual modulation not only decreases the synthesis of cholesterol but also accelerates its clearance from circulation.

Further, the effect of moringa extends to the genetic regulation of lipid metabolism. The upregulation of genes like PPAR-α, Cyp7a1, and Cyp7b1 coupled with the downregulation of pro-lipogenic markers such as SREBP-1c and PPAR-γ indicates a shift toward enhanced lipid catabolism and reduced lipid accumulation. These effects are complemented by the attenuation of inflammatory cytokines like TNF-α and IL-1β, suggesting an anti-inflammatory role that may indirectly benefit lipid metabolism and cardiovascular health.

Across *in vitro*, *in vivo*, and clinical models, moringa consistently lowered total cholesterol, triglycerides, LDL-C, and VLDL-C while elevating HDL-C levels. Notably, a synergistic effect was observed when combined with simvastatin, hinting at its potential utility as an adjunct to pharmaceutical therapy. These effects underscore its suitability as part of an integrative approach for managing dyslipidemia, especially in resource-limited situations where access to medications may be restricted.

However, some contradictions arose in the clinical findings. While most animal and combined model studies yielded strong results, human trials showed variable efficacy. For instance, supplementation in healthy young males and individuals with mildly elevated LDL levels resulted in no significant improvement in lipid profiles. These inconsistencies may be explained by differences in participant health status, age, duration of treatment, and dietary backgrounds, which can all influence the bioavailability and effectiveness of moringa's active compounds.

The strengths of the collective studies lie in their multimodal approach, covering biochemical, genetic, and physiological aspects. The diversity in extract types and delivery methods—ranging from powders to cookies and capsules—also supports the feasibility of real-world applications. Yet, the variability in extract composition and dosing regimens remains a limitation, hindering standardized clinical translation. To bridge these gaps, future research should focus on defining standardized extract formulations and conducting targeted clinical trials with stratified populations based on metabolic risk. Moreover, exploring moringa's role in nutrigenomics could unveil its potential to modulate gene expression in humans, offering a novel pathway for dietary intervention in dyslipidemia.

Hypertension

Hypertension, commonly referred to as high blood pressure, is a long-term health condition marked by consistently elevated arterial pressure beyond normal limits (Rashki et al. 2025). Hypertension in humans is defined as a sustained elevation in blood pressure at or above 130/80 mmHg. Accurate measurements are vital since common errors often lead to overdiagnosis and overtreatment. Stage 1 hypertension (130–139/80–89 mmHg) does not always require medication unless the individual has high-risk traits like age above 65, cardiovascular disease, chronic kidney disease, or diabetes. In these cases, treatment begins at ≥130/80 mmHg, while for others it starts at ≥140/90 mmHg. Regardless of the starting point, the target blood pressure is usually below 130/80 mmHg, with older adults aiming for <130 mmHg systolic. When initial values exceed the goal by more than 20/10 mmHg, dual-drug therapy with complementary mechanisms is recommended (Flack and Adekola 2020).

It represents one of the most widespread risk factors for cardiovascular disease worldwide. This condition plays a critical role in the onset and advancement of various cardiovascular disorders, including stroke, heart failure, and coronary artery disease (Naraki et al. 2023). Hypertension development has a high correlation with metabolic syndrome. The underlying mechanisms linking metabolic syndrome and hypertension are multifactorial and complex. Insulin resistance plays a crucial role in inducing hypertension. When cells become resistant to the effects of insulin, glucose uptake is impaired, resulting in elevated blood sugar levels and triggering a cascade of events, including increased production of inflammatory molecules and oxidative stress, both of which contribute to endothelial dysfunction, which impairs blood vessel dilation and regulation (Tsimihodimos et al. 2018). Furthermore, dyslipidemia is frequently associated with metabolic syndrome. These lipids can build up within blood vessel walls, causing atherosclerosis and constriction (Lechner et al. 2020). Furthermore, excess weight and central adiposity, which are frequent in metabolic syndrome, lead to an augmented activity of the sympathetic nervous system as well as the overproduction of vasoactive chemicals, which raise blood pressure even further (Vaněčková et al. 2014).

First-line antihypertensive therapies typically include angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), calcium channel blockers, thiazide diuretics, and beta-blockers (Khalil and Roman 2023). Although these agents effectively lower blood pressure in the majority of patients, some cases remain resistant to treatment, and polypharmacy often introduces complications and compliance challenges. The need for adjunctive therapies that exhibit vasodilatory and renoprotective effects has driven research into botanicals like *M. oleifera*, which has demonstrated antihypertensive activity in preclinical and emerging clinical studies.

Several studies have demonstrated that extracts and seeds of *M. oleifera* possess hypotensive and cardiovascular protective properties.

In vitro

M. oleifera leaf aqueous extract was discovered to relax the mesenteric arterial beds of L-NG-nitroarginine methyl ester (L-NAME) hypertensive rats via two separate mechanisms: endothelium-dependent and endothelium-independent pathways. Endothelium-dependent action is achieved by hyperpolarizing cells, which is mediated by endothelium-derived hyperpolarizing factors. The endothelium-independent effect, on the other hand, includes limiting extracellular calcium entry through voltage- and receptor-operated calcium channels, as well as preventing calcium release from inositol triphosphate receptor channels in the sarcolemma (Aekthammarat et al. 2020a) (Figure 3). Treating mice aortic tissue

exposed to adrenaline and KCl^- with *M. oleifera* leaf extract could significantly reduce contractions in the aorta and increase vasodilation (Zaffar et al. 2023).

Studies containing both *in vitro* and *in vivo* parts

It has been reported that treating primary human pulmonary artery endothelial cells with *M. oleifera* leaf aqueous extract increased NO production. In the *ex vivo* part of the research, it was observed that the aqueous extract derived from *M. oleifera* leaves caused relaxation in mesenteric arterial beds that were pre-contracted with methoxamine. However, this effect was eliminated when the endothelium was removed. The findings of the *in vivo* part of the study indicated that intravenous injection of the extract reduced mean arterial blood pressure (MAP) in rats (Aekthammarat et al. 2020b).

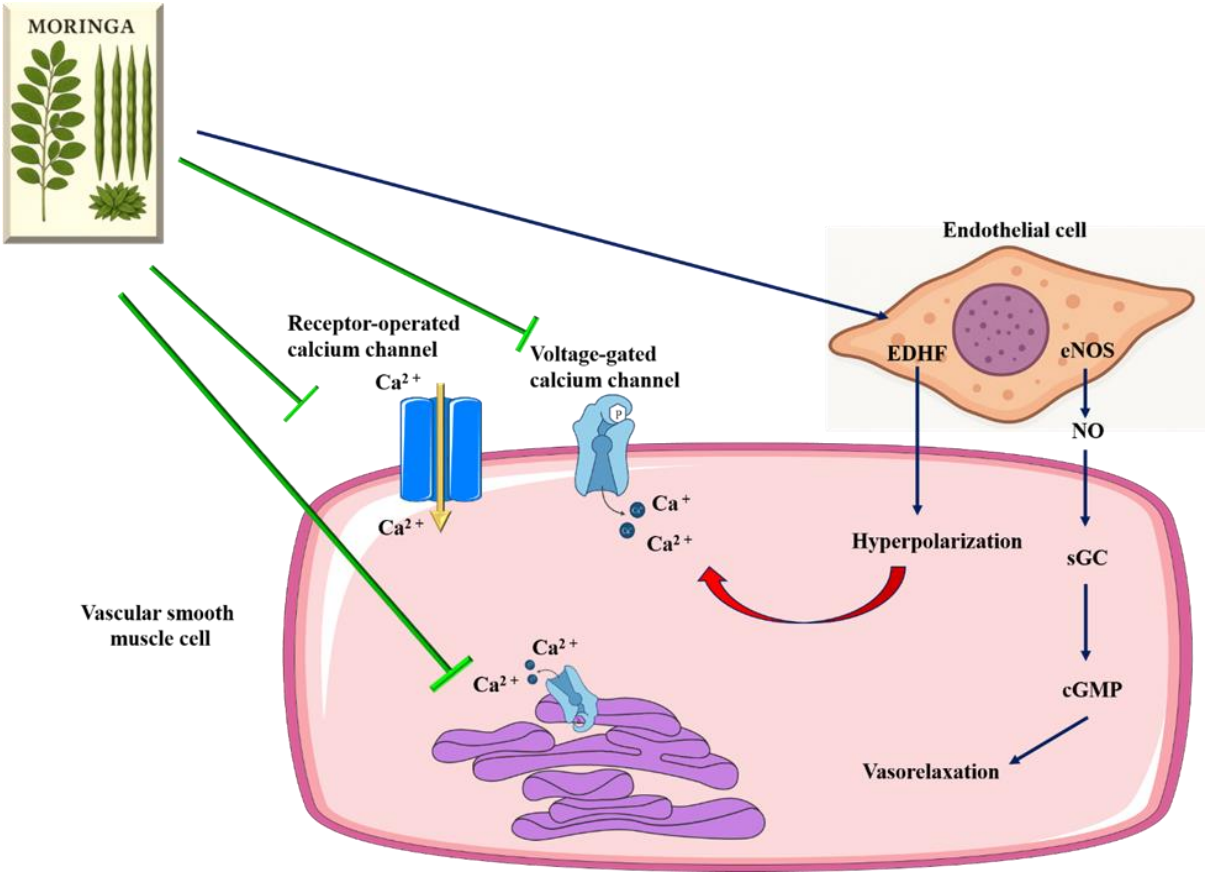

Figure 3. The anti-hypertensive effect of *M. oleifera* and its main constituents. Abbreviations: cGMP: cyclic guanosine monophosphate; EDHF: endothelium-derived hyperpolarizing factor; eNOS: endothelial nitric oxide synthase; NO: nitric oxide; sGC: soluble guanylate cyclase.

In vivo

It has been illustrated that an ethanolic extract of *M. oleifera* seeds reduced MAP in a normotensive cat (Chidozie et al. 2015). The oral administration of *M. oleifera* seed powder to spontaneous hypertensive rats had no significant effect on their blood pressure (Randriamboavonjy et al. 2016). Another study provided clear evidence that *M. oleifera* leaf aqueous extract possesses antihypertensive effects by inhibiting the secretion of IL-2 and modulating T-cell calcium signaling specifically in hypertensive rats. However, these effects were not observed in normotensive Wistar-Kyoto rats (Attakpa et al. 2017). In an *in vivo* study, the *M. oleifera* leaf methanol fraction was administered to hypertensive rabbits, and the obtained data showed that the blood

pressure, heart/body weight ratio, and cardiac production of H_2O_2 amounts were attenuated, while the cardiac NO amounts were increased (Adedapo et al. 2018). In addition, it has been indicated that the antihypertensive effect of *M. oleifera* leaf aqueous extract in L-NAME hypertensive rats is likely achieved through several mechanisms. These include alleviating vascular dysfunction and oxidative stress (decreasing vascular $O_2^{\bullet-}$ production and MDA levels in the plasma and thoracic aorta while increasing SOD and CAT activities), as well as promoting endothelium-dependent vasorelaxation (Aekthammarat et al. 2019). It has been reported that the administration of *M. oleifera* leaf hydro-ethanol extract to rats with hypertension could significantly decrease the blood pressure (Gbankoto et al. 2019). Likewise, the supplementation of *M. oleifera* leaf methanolic and ethyl acetate extracts in mice with hypertension resulted in a decreased in blood pressure (Acuram and Chichioco Hernandez 2019). The oral administration of *M. oleifera* hydrolysates and peptide fractions resulted in significant decreases in systolic, diastolic pressure, and MAP in spontaneously hypertensive rats. Among these fractions, the 1-3 kDa peptide fraction reduced systolic blood pressure the most significantly, as well as the fastest in both systolic blood pressure and MAP. The hydrolysate, on the other hand, displayed the most consistent drop in systolic blood pressure. Surprisingly, the longer peptides (>10 kDa) were the most successful in lowering the heart rate. (Aderinola et al. 2019). In another investigation, the protein was extracted from *M. oleifera* leaves and subsequently hydrolyzed using alcalase enzyme to generate *M. oleifera* leaf protein hydrolysate. The *M. oleifera* leaf protein hydrolysate was ultrafiltered to obtain peptide fractions with varying molecular weights. Both the *M. oleifera* leaf protein hydrolysate and ultrafiltration fractions were tested for antihypertensive activity. Notably, peptides having a molecular weight of less than 1 kDa inhibited ACE and renin much better than other fractions. The 1 kDa component was administered orally to spontaneously hypertensive rats and resulted in a favorable decrease in blood pressure. Following that, the 1 kDa component was separated, purified, and the final active component was identified using mass spectrometry and amino acid sequence analysis. Leu-Gly-Phe-Phe (LGF) and Gly-Leu-Phe-Phe (GLFF), two highly active peptides with dual inhibitory action against ACE and renin, were isolated. In spontaneously hypertensive rats, LGF and GLFF were shown to dramatically lower both systolic and diastolic blood pressure (Ma et al. 2021). Moreover, the crude *M. oleifera* methanolic leaf extract and other fractions revealed a dose-dependent negative inotropic and chronotropic effect on African giant rats. Besides, *M. oleifera* methanolic leaf extract and methanol fraction disclosed considerable hypotensive properties (Odii et al. 2022).

Another experimental study explored the antihypertensive and cardioprotective effects of dietary supplementation with *M. oleifera* seeds in rats subjected to L-NAME-induced hypertension. At inclusion levels of 5% and 10%, the seeds significantly lowered blood pressure and inhibited cardiac acetylcholinesterase (AChE) and monoamine oxidase (MAO) activity, indicating potential neuromodulatory effects. Additionally, treated rats exhibited enhanced antioxidant defense, evidenced by elevated non-protein thiol (NP-SH) levels and reduced lipid peroxidation (Oyeleye et al. 2023).

This study investigated the antihypertensive potential of *M. oleifera* seeds in rats exposed to L-NAME. Over a five-week period, *M. oleifera* supplementation at 10% and 20% dietary inclusion significantly countered the hypertensive effects of L-NAME, as evidenced by lowered systolic and diastolic blood pressure. The cardioprotective effects were further supported by improvements in oxidative stress markers, including elevated

nitric oxide levels and reduced MDA concentrations in heart and kidney tissues. Additionally, *M. oleifera* co-treatment with Lisinopril mitigated apoptosis, as shown by decreased caspase-3 expression in cardiac and renal cells (Ake et al. 2024). (Silva et al. 2024).

Clinical trial

Receiving *M. oleifera* powder was found to have no significant effect on the blood pressure of normal individuals (Seriki et al. 2015). Taking *M. oleifera* leaf capsules for four weeks in patients with type 2 diabetes resulted in a decreased systolic blood pressure (Taweerutchana et al. 2017). According to a clinical trial, eating cooked *M. oleifera* leaves during the two-hour postprandial interval was observed to reduce blood pressure. Even in people who consumed a lot of salt before the experiment, this study showed a possible drop in both systolic blood pressure and diastolic blood pressure (Chan Sun et al. 2020). Another clinical trial reported that the prescription of *M. oleifera* dry leaf powder to prediabetic patients had no considerable effect on their blood pressure (Díaz-Prieto et al. 2022). The prescription of *M. oleifera* leaves to diabetic patients could successfully decrease their systolic blood pressure (C. Afiaenyi et al. 2023). The supplementation of *M. oleifera* leaf capsules to type 2 diabetic patients could significantly lower their blood pressure (Hameed et al. 2023) (Table 4).

Table 4. Effect of *M. oleifera* on blood pressure

Type of extract/ Compound	Study design	Doses/Duration/Route of use	Results	References
		In vitro		
M. oleifera leaf aqueous extract	Mesenteric arterial bed of male Wistar rats	0.001–3 mg in 0.1 ml	↑Vasodilation	(Aekthammarat et al. 2020a)
M. oleifera leaf extract	Mice aortic tissue		↑Vasodilation ↓Contractions in aorta	(Zaffar et al. 2023) 2023
		In vitro* plus *in vivo		
M. oleifera leaf aqueous extract	*In vitro*, primary human pulmonary artery endothelial cells	3–30 µg/ml, 30 or 60 min	↑NO production	(Aekthammarat et al. 2020b)
	Ex vivo, mesenteric arterial beds	0.001–3 mg	↑Relaxation in mesenteric arterial beds	
	In vivo, male Wistar rats	1–30 mg/kg, i.v.	↓MAP	
		In vivo		
M. oleifera seeds ethanolic extract	A female cat	10, 20 and 40 mg/ml, arterial injection	↓MAP	(Chidozie et al. 2015)
M. oleifera seed powder	Male spontaneous hypertensive rats	750 mg/day, 8 weeks, p.o.	-No effect on blood pressure	(Randriamboavonjy et al. 2016)
M. oleifera leaf aqueous extract	Wistar-Kyoto Rats and male spontaneously hypertensive rats	200, 400, 600 mg/kg, 8 weeks, p.o.	↓Blood pressure, secretion of IL-2 ↑Intracellular free calcium	(Attakpa et al. 2017)
M. oleifera leaf methanol fraction	Male rabbits	100, 200, and 400 mg/kg, 3 weeks, gastric intubation	↑Cardiac NO levels ↓Blood pressure, heart/body weight ratio, cardiac H_2O_2 production	(Adedapo et al. 2018)
M. oleifera leaf aqueous extract	Male Wistar rats	30 and 60 mg/kg, 3 weeks, p.o.	↑SOD and CAT activities ↓Blood pressure and tachycardia, vascular $O_2^{\bullet-}$ production, MDA levels of plasma and thoracic aorta	(Aekthammarat et al. 2019)
M. oleifera leaf hydro-ethanol extract	Male Wistar rats and shrimp larvae	500 mg/kg, 2 weeks, p.o.	↓Blood pressure	(Gbankoto et al. 2019)

Table 4. Continued

M. oleifera leaf methanolic and ethyl acetate extracts	Female ICR mice	0.01, 0.3 g/kg, 25 days, gavage	↓Blood pressure	(Acuram and Chichioco Hernandez 2019)
M. oleifera seed enzymatic protein hydrolysate	Rats	200 mg/kg, p.o.	↓Systolic and diastolic blood pressure, MAP	(Aderinola et al. 2019)
M. oleifera leaf protein hydrolysate and the peptides fraction	Male spontaneously hypertensive rats	*M. oleifera* leaf protein hydrolysate and 1 kDa polypeptide: 100 mg/kg, i.g. LGF and GLFF: 30 mg/kg, i.g.	-Inhibited ACE and renin ↓Blood pressure	(Ma et al. 2021)
M. oleifera leaf methanolic extract and its fractions	African giant rats	5 mg/ml, i.v.	↓Blood pressure	(Odii et al. 2022)
M. oleifera seed powder	Male albino Wistar rats	5% and 10%, 14 days, p.o.	↑Antioxidant defense ↓Blood pressure, cardiac AChE, MAO activity, lipid peroxidation	(Oyeleye et al. 2023)
M. oleifera seeds	Male Wistar rats	10% and 20%, 5 weeks, p.o.	↑NO ↓Systolic and diastolic blood pressure, MDA in heart and kidney tissues, apoptosis, caspase-3 expression in cardiac and renal cells	(Ake et al. 2024)
M. oleifera seed compounds	Female Wistar rats	-	↓Oxidative stress across the maternal-placental-fetal axis, lipid peroxidation, superoxide anion production, NADPH oxidase activity in both placental and fetal kidney tissues, systolic blood pressure, oxidative injury markers in the adult offspring	(Silva et al. 2024)
		Clinical trial		
M. oleifera powder	16 normal individuals	0.03, 0.07 g/kg, 15 days, p.o.	-No significant effect on blood pressure	(Seriki et al. 2015)
M. oleifera leaf capsule	32 participants with type 2 diabetes	8 g, 4 weeks, p.o.	↓Systolic pressure	(Taweerutchana et al. 2017)
Cooked *M. oleifera* leaf	41 healthy individuals	120 g, p.o.	↓Systolic and diastolic pressures	(Chan Sun et al. 2020)
M. oleifera dry leaf powder	73 prediabetic patients	6 × 400 mg/day, 12 weeks, p.o.	-No significant effect on blood pressure	(Díaz-Prieto et al. 2022)
M. oleifera leaves	40 diabetic subjects	20, 40, and 60 g, 14 days, p.o.	↓Systolic blood pressure	(C. Afiaenyi et al. 2023)
M. oleifera leaf capsules	24 individuals with type 2 diabetes	3 and 6g, twice a day, 3 months, p.o.	↓Blood pressure	(Hameed et al. 2023)
M. oleifera leaf powder	60 subjects aged 20–35 years with normal ocular and systemic parameters	53 and 75.5 mg, p.o.	↓Intraocular pressure at 90 min and blood pressure at 60 min	(Jabbar et al. 2023)
M. oleifera leaf juice	48 pregnant women with anemia and hypertension	1x400 ml, 7 days, p.o.	↑Hemoglobin levels ↓Systolic and diastolic blood pressure	(Usman et al. 2025)

AChE: acetylcholinesterase; ACE: angiotensin-converting enzyme; CAT: catalase; H_2O_2: hydrogen peroxide; IL-2: interleukin-2; MAO: monoamine oxidase; MAP: mean arterial pressure; MDA: Malondialdehyde; NO: nitric oxide; SOD: superoxide dismutase.

Another randomized controlled trial evaluated the effects of graded doses of *M. oleifera* leaves on metabolic parameters in type 2 diabetic individuals from a rural Nigerian community. Over a 14-day intervention period, participants consumed 20, 40, or 60 g of moringa leaves daily alongside their regular diets. While fasting blood glucose levels did not show significant differences across groups, the highest dose group (60 g/day) exhibited a significant reduction in systolic blood pressure and a notable increase in triglyceride levels. However, after adjusting for baseline values, no statistically significant differences were observed in any of the measured parameters (C. Afiaenyi et al. 2025).

A quasi-experimental study evaluated the short-term effects of various doses of *M. oleifera* on intraocular pressure and blood pressure in healthy young adults. The study involved subjects aged 20–35 years with normal ocular and systemic parameters. Participants were divided into two groups and monitored at multiple time intervals post-ingestion. Results showed that moringa intake significantly reduced intraocular pressure at 90 min and blood pressure at 60 min, with the reductions in both parameters being dose-dependent. However, values gradually returned to baseline by 120 min, indicating a transient but notable hypotensive and ocular pressure-lowering effect (Jabbar et al. 2023).

This observational study assessed the impact of moringa leaf tea on blood pressure among hypertensive patients participating in the Prolanis program at Sidabowa Primary Clinic, Indonesia. Involving 41 participants, the case-control design revealed no statistically significant changes in either systolic or diastolic blood pressure following 14 days of moringa tea consumption. Despite this, the case group exhibited a greater reduction in systolic pressure compared to controls (Pratomo et al. 2025).

In addition, the potential of *M. oleifera* leaf juice was assessed as a nutritional intervention for pregnant women dealing with both anemia and hypertension. The pretest-posttest analysis involved 48 participants and revealed significant improvements following moringa juice consumption. Specifically, hemoglobin levels rose from 9.77 g/dl to 10.25 g/dl, indicating enhanced iron status, while both systolic and diastolic blood pressure showed notable reductions (Usman et al. 2025).

M. oleifera has demonstrated promising antihypertensive effects across various experimental models, suggesting that it modulates blood pressure through a complicated network of protective mechanisms. These mechanisms include enhanced endothelium-dependent and -independent vasorelaxation, inhibition of calcium influx through vascular channels, and promotion of nitric oxide bioavailability. Additionally, moringa exhibits strong antioxidant properties, suppressing oxidative stress markers such as MDA and superoxide anions while boosting endogenous antioxidants like superoxide dismutase and catalase. Furthermore, its bioactive peptides have shown direct inhibition of ACE and renin, indicating a capacity to block pathways central to blood pressure regulation.

An especially novel insight from the gathered data is the dual effect of moringa on both vascular tone and immune modulation. Some studies highlighted its ability to suppress IL-2 secretion and T-cell calcium signaling in hypertensive but not normotensive subjects, which suggests a context-specific immune–vascular interface. This, coupled with the effect f moringa on neurotransmission-related enzymes (e.g., AChE and MAO), underscores its potential as a neuromodulatory agent in hypertension management.

The collective strength of the studies lies in the wide spectrum of evidence spanning *in vitro* assays, animal models,

and human trials. They also explore different parts of the plant (leaves, seeds, peptides) and various formulations (extracts, teas, capsules, juices). However, the limitations include variability in results among human clinical trials—particularly in normotensive or prediabetic individuals—potentially due to differences in baseline blood pressure, metabolic status, dosage, or duration of intervention. These discrepancies highlight the importance of personalized approaches and longer-term studies with standardized moringa preparations.

Moving forward, moringa may be effectively integrated into therapeutic regimens as a plant-based adjunct for hypertension management, particularly in low-resource settings or among individuals with mild to moderate blood pressure elevations. Considering its biochemical diversity and multi-targeted mechanisms, moringa could act not only as a natural remedy but also as a catalyst for vascular and metabolic reprogramming. Future applications may include personalized therapies, functional foods, and broader adoption in community health initiatives. Realizing these benefits will require standardized formulations, advanced molecular investigations, and long-term clinical trials.

Discussion

In conclusion, the consolidated evidence from this review supports *M. oleifera* as a promising natural approach for managing various components of metabolic syndrome. *M. oleifera* exhibits potential in improving glucose homeostasis by enhancing glucose tolerance, lowering fasting blood sugar and HbA_{1c} levels, and modulating key enzymes and pathways (e.g., α-glucosidase, glucose transporters) involved in carbohydrate metabolism. These actions are primarily attributed to its antioxidant capacity, which aids in restoring hepatic enzyme balance, repairing oxidative tissue damage, and protecting pancreatic islet cells.

Additionally, *M. oleifera* demonstrates anti-obesity effects by suppressing adipogenesis, reducing triglyceride storage, promoting apoptosis in adipocytes, lowering pro-inflammatory cytokines, and boosting adiponectin expression. Its impact on lipid metabolism includes elevating HDL, reducing LDL and total cholesterol, increasing LDL receptor binding activity, and influencing the expression of lipid-regulating genes. In terms of blood pressure regulation, moringa extracts promote vasodilation via both endothelium-dependent and -independent mechanisms, enhancing NO bioavailability and limiting calcium influx.

Despite these promising outcomes, significant gaps remain in the field. Future investigations should prioritize large-scale, well-designed randomized clinical trials to validate efficacy and safety. Identifying the specific bioactive compounds responsible for therapeutic effects, establishing dose-response relationships, and evaluating synergistic interactions with conventional medications are also essential steps. These efforts will clarify the role of moringa as a therapeutic tool and accelerate its translation into evidence-based interventions for metabolic syndrome.

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