

Moringa oleifera Supplementation and Metabolic Syndrome: A Critical Review of Human and Preclinical Evidence

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ABSTRACT

Introduction: Metabolic syndrome (MetS) is a multifactorial condition characterized by the coexistence of central adiposity, elevated blood pressure, dysglycemia, and atherogenic dyslipidemia, and is associated with increased risks of cardiovascular disease and type 2 diabetes mellitus (T2DM). *Moringa oleifera* (MO) has attracted considerable interest because of its diverse bioactive constituents and reported effects on glucose metabolism, lipid homeostasis, adiposity, vascular function, inflammation, and oxidative stress. However, the clinical relevance of these findings remains uncertain. This review critically evaluates the available evidence regarding the effects of MO supplementation on MetS and its individual components, with particular emphasis on differences between human and preclinical evidence, heterogeneity in botanical preparations and dosing, methodological limitations, and potential explanations for inconsistent findings. A structured literature search was conducted in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar from database inception to [updated search date]. Relevant human clinical studies and in vivo animal studies investigating oral MO preparations in relation to MetS or its major metabolic components were identified. Evidence was synthesized narratively according to study population, intervention characteristics, duration, outcomes, and methodological quality. Direct evidence in participants with clinically defined MetS was distinguished from indirect evidence derived from populations with diabetes, prediabetes, obesity, hypertension, or isolated metabolic abnormalities. Preclinical studies generally report beneficial effects of MO on glucose regulation, lipid metabolism, adiposity, blood pressure, oxidative stress, and inflammatory pathways; however, substantial heterogeneity exists in animal models, plant preparations, doses, and intervention durations. Human evidence is considerably more limited and heterogeneous. Earlier clinical studies conducted mainly in individuals with diabetes or isolated metabolic abnormalities have reported mixed effects on glycemic indices, lipid parameters, blood pressure, and anthropometric outcomes. Importantly, a recent double-blind randomized placebo-controlled trial in 73 participants with MetS directly evaluated MO supplementation for 8 weeks, providing the first evidence that should be interpreted specifically within a clinically defined MetS population. A 2025 meta-analysis of randomized controlled trials also found no consistent effects across most cardiometabolic outcomes and rated the overall certainty of evidence as very low because of heterogeneity and methodological limitations. MO demonstrates biologically plausible metabolic effects, particularly in preclinical models, but current clinical evidence does not yet establish a consistent therapeutic effect on MetS. Differences in botanical preparation, dose, treatment duration, baseline metabolic status, concomitant therapy, and study quality substantially limit cross-study comparisons. Future adequately powered and well-controlled clinical trials should use standardized MO preparations, clearly defined MetS populations, clinically relevant outcomes, longer follow-up, and systematic assessment of safety and potential herb drug interactions.

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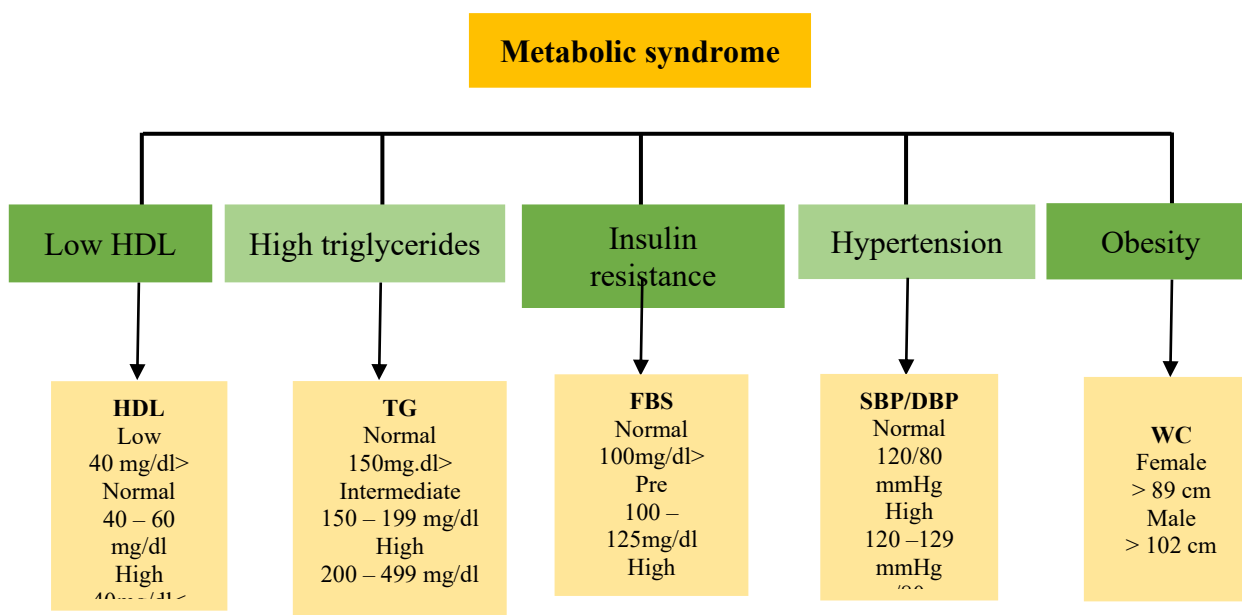
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Introduction

Metabolic syndrome (MetS) is a multifaceted cluster of interrelated metabolic abnormalities, defined clinically by the co-occurrence of central obesity, elevated blood pressure, atherogenic dyslipidemia (elevated triglycerides and reduced high-density lipoprotein cholesterol (HDL-C), and impaired fasting glucose or insulin resistance [1, 2](#)). Figure 1 outlines the harmonized diagnostic criteria and diagnostic thresholds defining this condition. The escalating global prevalence of MetS, surpassing 25% across developed and developing

economies [3-5](#)), is coupled with a significantly heightened risk of cardiovascular morbidity, all-cause mortality, and type 2 diabetes mellitus [6-8](#)). Although lifestyle interventions predominantly dietary restructuring such as adherence to mediterranean-style dietary patterns constitute the frontline approach [9-11](#)), there is growing biomedical interest in bioactive phytotherapeutic agents capable of modulating multi-target pathophysiological pathways implicated in MetS [12](#)).



Among these botanical candidates, *Moringa oleifera* (MO), commonly known as the “miracle tree” (family Moringaceae), has garnered substantial clinical and pharmacological attention [13-15](#)). Extensive compositional analyses demonstrate that MO leaves represent a dense matrix of bioactive constituents, including polyphenols, flavonoids (e.g., quercetin, kaempferol, rutin, chlorogenic acid), and isothiocyanate precursors such as glucomoringin [16-18](#)) ([Table 1](#)).

Although several narrative reviews have summarized the broad pharmacological attributes of MO frequently clustering its general antioxidant, anti-inflammatory, or anti-diabetic activities [19, 20](#)) critical scientific gaps remain unaddressed. First, existing literature frequently conflates isolated glycemic or lipid effects observed in overt diabetic populations with the therapeutic management of composite Metabolic Syndrome. Second, considerable discrepancies exist regarding the clinical efficacy of MO on human cardiometabolic risk factors, where promising preclinical data from rodent models frequently contrast with heterogeneous or neutral

outcomes in human clinical trials. Third, prior publications have rarely synthesized the pharmacological implications of preparation heterogeneity (e.g., whole dried leaf powder vs. standardized hydroethanolic extracts, variations in polyphenol standardization, and dosing protocols).

Therefore, unlike previous broad reviews, the present work was designed to provide a critical, methodologically transparent synthesis strictly centered on MO supplementation in the context of the defined components of MetS. Specifically, this review aims to: (i) systematically decouple preclinical mechanistic insights from clinical human trials; (ii) critically appraise clinical trials evaluating individual or composite MetS risk parameters; (iii) identify the underlying pharmacological, methodological, and botanical reasons for conflicting outcomes; and (iv) assess the clinical safety profile, potential herb-drug interactions, and translational limitations of MO therapeutic use.

Table1. Chemical compounds in MO leaves

Flavanoids and Flavanol Glycosides		
Rutin*	Quercetin*	Isoquercetin
Astragalin	Isorhamnetin	Kaempferol*
Apigenin*	Luteolin	Genistein
Daidzein	Myricetin*	Epicatechin
Vicenin-2	Quercetin-3-O-glucoside	Quercetin-3-O-(6"-malonyl) glucoside
Kaempferol-3-O-glucoside	Kaempferol-3-O-(6"-malonyl) glucoside	Kaempferol-3-rutinoside
Kaempferol-3-O- α -rhamnoside	Kaempferide 3-O-(2'',3''-diacetylglucoside)	Kaempferol-3-O-[[β -glucosyl-(1 $\rightarrow$ 2)]-[α rhamnosyl-(1 $\rightarrow$ 6)]- β -glucoside-7-O- α -rhamnoside
Kaempferide-3-O-(2''-O-galloylrhamnoside)	Kaempferide-3-O-(2''-O-galloylrutinoside)-7-O- α -rhamnoside	Kaempferol-3-O-[[α -rhamnosyl-(1 $\rightarrow$ 2)]-[α -rhamnosyl-(1 $\rightarrow$ 4)] β -glucoside-7-O- α -rhamnoside
Glucosinolate and Isothiocyanate		
4-(α -L-rhamnopyranosyloxy) benzyl glucosinolate (glucomoringin)*	4-[[2'-O-acetyl- α -L-rhamnosyloxy)benzyl] Glucosinolate	4-[[3'-O-acetyl- α -L-rhamnosyloxy)benzyl] Glucosinolate
4-[[4'-O-Acetyl- α -L-rhamnosyloxy)benzyl] Glucosinolate	4-[[α -L-rhamnosyloxy) benzyl] Isothiocyanate	4-[[2'-O-acetyl- α -L-rhamnosyloxy) benzyl] Isothiocyanate
Glucoconringiin	Sinalbin	4-[[β -D-glucopyranosyl-1- $\rightarrow$ 4- α -L-rhamnopyranosyloxy) benzyl] Isothiocyanate
Phenolic Acid 21-26		
Gallic acid*	Syringic acid	Ellagic acid*
Ferulic acid*	Caffeic acid*	o-Coumaric acid*
p-Coumaric acid	Sinapic acid	Chlorogenic acid*
Cryptochlorogenic acid	Gentisic acid	-
Alkaloid and Sterol 27-29		
4'-hydroxyphenylethanamide- α -L-rhamnopyranoside (marumoside A)*	3''-O- β -D-glucopyranosyl derivatives (marumoside B)*	N, α -L-Rhamnopyranosylvincosamide
Pyrrolemarumine-4''-O- α -L-rhamnopyranoside	Niazimicin	Niaziminin
β -sitosterol*	Lupeol	-
Others 30-32		
Tocopherols*	Benzoic acid 4-O- β -glucoside	Benzoic acid 4-O- α -rhamnosyl-(1 $\rightarrow$ 2)- β -glucoside
Benzaldehyde 4-O- β -glucoside	Niazirin*	Niaziridin*

Some research in mice reported that intake of MO had valuable impacts on the metabolic syndrome risk factors. Human studies, particularly clinical trials regarding the effects of MO on metabolic syndrome risk factors, are limited and controversial. However, the existing literature on the impact of MO on metabolic syndrome in humans and animals remains inconclusive and needs a review of evidence. Therefore, this review article aims to critically evaluate the current research on the effects of MO on metabolic syndrome or any of its factors in both humans and animals.

Methodology and Literature Search Strategy

1. Literature Search and Information Sources

As a comprehensive narrative review synthesizing both mechanistic and clinical evidence, a structured and transparent literature search was conducted following the recommendations of the SANRA (Scale for the Assessment of Narrative Review Articles) guidelines. Electronic bibliographic databases, including PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar, were comprehensively searched from their inception up to May 2024. In addition, the reference lists of retrieved original articles and pertinent narrative reviews were manually scrutinized to identify additional relevant publications.

2. Search Strategy and Terms

The search strategy utilized a combination of Medical Subject Headings (MeSH) and title/abstract keywords. The following boolean search string was developed and adapted across databases:

("MO" OR "Moringa" OR "drumstick tree") AND ("metabolic syndrome" OR "insulin resistance" OR "dyslipidemia" OR "hyperglycemia" OR "hypertension" OR "obesity" OR "lipid profile" OR "blood pressure" OR "fasting blood glucose"). Language was restricted to peer-reviewed articles published in English.

Human studies

Based on current knowledge, no direct study has examined the effect of MO on metabolic syndrome. Nevertheless, by delving into the individual components of the syndrome, we can gain insights into the potential impact of this plant. For instance, a study conducted in 2023 by Maryam Jabbar et al. aimed to investigate the effect of MO on blood pressure and intraocular pressure. The study involved 60 individuals aged 20-35 with an IOP range of 11-22 mmHg and BP of 120/80 mmHg. The results indicated that MO can have a significant effect in reducing blood pressure and intraocular pressure ($P < 0.05$). Another study conducted in 2022 revealed that MO leaf extract plays a role in reducing the subjects' blood pressure ($P < 0.05$).

However, it should be noted that a study conducted by Sarra HENOUDA et al. in 2023 on diabetic patients showed that MO leaf powder did not significantly affect blood pressure, FBS, BMI, or weight of diabetic patients ($P > 0.05$). Nevertheless, they reported reduced LDL-C and HbA1c levels and elevated HDL-C ($P < 0.05$). In a 2023 clinical study by Ifeoma C. Afiaenyi et al. on 40 adult male and female diabetic subjects, receiving 40 mg of MO daily for 14 days significantly decreased mean systolic pressure and triglyceride values. However, there were no changes in fasting blood glucose levels.

Given the heterogeneous results from various studies, further research is needed to determine the optimal dosage of MO powder and investigate its long-term effects. A summary of clinical trials investigating the effects of *Moringa oleifera* on metabolic syndrome components is presented in (Table 2).

Table 2. Summary of human studies related to the effect of *Moringa oleifera* on metabolic syndrome risk factors

Author & Year (Ref)	Study Design & Cohort Characteristics	Intervention, Botanical Form & Daily Dose	Comparator / Control	Follow Up duration	Result
Afiaenyi et al., 2023 33)	Single-blind RCT; n = 40 adult T2DM patients (both sexes)	MO leaf powder: graded doses (20 g, 40 g, and 60 g/day) in meals	Placebo control (standard diet)	14 days	↓ blood pressure ↑ TG ↔ FBG
Henouda et al., 2023 34)	Open-label RCT; n = 44 patients with established T2DM	Standardized MO dried leaf powder: 3,600 mg/day (in divided doses)	Routine antidiabetic standard-of-care alone	3 months	↓ LDL ↓ HbA1c ↑ HDL

Kumari, 2010 35)	Controlled clinical trial; <i>n</i> = 60 NIDDM patients aged 30–60 years	Whole MO leaf powder: 8 g/day incorporated into daily food	Matched diabetic control group receiving baseline dietary advice	40 days	↓ SBP&DBP ↓ LDL ↓ TG ↑ HDL
Taweerutchana et al., 2017 36)	Double-blind, randomized, placebo-controlled trial; <i>n</i> =32 T2DM patients	MO dried leaf capsules: 8 g/day (4 g twice daily before meals)	Matched inert placebo capsules	4 weeks	↔ blood glucose ↓ SBP
Ghiridhari et al., 2011 37)	Prospective clinical trial; <i>n</i> = 70 participants (35 diet-controlled T2DM; 35 healthy controls)	MO leaf powder tablets: 1 g/day titrated to 2 g/day	Baseline self-control & healthy normoglycemic group	45 days	↓ FBG
Nambiar et al., 2010 38)	Pilot intervention trial; <i>n</i> = 15 obese subjects with impaired glucose tolerance / NIDDM	Dehydrated MO leaf powder: 50 g/day administered in traditional recipes	Matched baseline period (pre-post crossover)	20 days	↓ blood glucose ↓ TC ↓ TG ↓ VLDL ↓ LDL
Dominguez-Rodriguez 39)	Double-blind, randomized crossover trial; <i>n</i> = 24 recruited obese T2DM patients on metformin (17 women, 7 men; 20–60 y)	MO leaves alongside a moderate-carbohydrate diet	Placebo-matched control cross-over phase	22 weeks total (10 weeks intervention, 2 weeks washout, 10 weeks crossover)	↓ LDL ↓ HbA1c ↓ Fasting glucose ↓ BP

Abbreviations: SBP: systolic blood pressure; DBP: diastolic blood pressure; TG: triglycerides; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; VLDL-C: very-low-density lipoprotein cholesterol; HbA1c: glycated hemoglobin A1c; NIDDM: non-insulin-dependent diabetes mellitus; T2DM: type 2 diabetes mellitus; RCT: randomized controlled trial

Animal studies

A 2018 study by Marisa López et al. aimed to investigate the effects of MO on metabolic syndrome by feeding male rats a high-fat diet and water containing 10% fructose for six weeks to induce metabolic syndrome. MO leaf powder was administered at a dose of 700 mg/kg bw daily for three weeks. The results showed that the oral administration of MO in mice with induced MS caused a decrease in the fasting glucose (FG) level in the treatment group compared to the control group, but did not cause a significant difference in the insulin tolerance test (ITT). It also caused a significant decrease in the area under the curve for triglyceride (TG), oral glucose tolerance test (OGTT), and abdominal circumference (AC) compared to the control group ($P < 0.05$).

The reviews of another study conducted in 2019 by Hafez Mohammad Irfan et al. determined that a high-fat diet, Plasma insulin level, and HOMA-IR significantly increase body weight, abdominal fat, BMI, and liver weight ($P > 0.05$). In addition, the final findings of a study conducted in 2015 by Mohamed-Hesham Daba indicated that MO, metformin, and especially the combined treatment of both, reduce fasting blood

glucose, insulin level, and HOMA index. They also effectively improve the lipid profile, liver enzymes, and insulin resistance. Therefore, MO effectively improves the factors of metabolic syndrome.

A study conducted in 2020 by Ezzat et al. [40](#)) aimed to investigate the effect of ethanolic extract of MO leaves. MO with doses of 200 and 400 mg/kg body weight was evaluated for its anti-obesity activity for one month. 400 mg/capsule MO significantly affected weight gain percentage, final weight loss, and obesity index of mice ($P < 0.05$). Another study conducted in 2023 showed that rutin, one of the compounds isolated from MO leaves, is considered a therapeutic agent against glucose intolerance and obesity ($P < 0.05$).

It is worth mentioning that three studies conducted in 2018, 2020, and 2021 were aimed at investigating the anti-obesity potential of the methanolic extract of MO leaves on obesity in mice. The results determined that MO optimizes histopathological changes and obesity caused by a high-fat diet. In addition, it significantly affects the pathophysiological regulation of weight gain.

Another study conducted in 2019 concluded that the evaluations showed that MO leaves reduce fat cell formation. Therefore, they can control obesity ($P<0.05$). A study conducted in 2017 aimed to elucidate the molecular mechanisms of the anti-obesity effect of MO and its relationship with metabolic and vascular risks [41](#)). The results of the investigation revealed that the ethanolic extract of MO at the rate of 600 mg/kg bw in obese female rats causes a decrease in body weight ($P<0.05$). A study was conducted in 2019 and 2020 to investigate the effect of MO leaf aqueous extract on ω -nitro-L-arginine-methyl ester (L-NAME)-induced hypertension and reduction of mean arterial pressure (MAP), respectively. The results indicated that MO can significantly reduce blood pressure. A study conducted in 2019 by Aktar et al. aimed to investigate the antihypertensive effect of MO leaf methanolic extract of MO. The results showed that MO can act as a natural blood pressure inhibitor. Another study conducted in 2021 confirmed that bioactive peptides from MO leaves have the potential to be used in the treatment of high blood pressure in humans. Another study related to blood pressure in 2023 by Zaffar and colleagues aimed to investigate the effect of MO leaf extract on rat aortic tissue contraction [42](#)). Data analysis showed that MO leaf extract could relax the smooth muscles of blood vessels. The study conducted in 2020 aimed to investigate the effect of MO leaf diet on serum lipid

profile and genes related to lipid metabolism in unisex Nile tilapia. The results indicated that MO leaf powder could be used in the diet of Nile tilapia as a food ingredient to reduce blood fat and improve growth performance ($P<0.05$). Another study conducted in 2017 by Mardewi et al [43](#)). showed that adding 10% MO leaf meal reduced TG levels without affecting HDL levels. Also, adding 12% MO leaf meal does not harm the weight of internal organs. A study conducted in 2015 aimed to investigate the effect of the methanolic extract of MO on glycogen synthesis, glucose tolerance, and lipid metabolism in rats with alloxan-induced diabetes [44](#)). Preclinical and animal experimental studies evaluating *Moringa oleifera* in the context of metabolic abnormalities are summarized in [Table 3](#). The results indicated that the hypoglycemic effects of MO may lead to increased glycogen synthesis and glucose uptake by stimulating insulin release. A study was conducted in 2022, which aimed to investigate the effect of ice creams enriched with MO leaves on the lipid profile of rats [45](#)). Studies have shown that MO leaf compounds reduce lipid profile (TG, TC, and LDL) ($P<0.05$). Nevertheless, the results of a study conducted in 2015 by Seriki et al, showed that MO does not affect the lipid profile [46](#)).

Table 3. Animal experimental studies investigating *Moringa oleifera* in metabolic syndrome and related metabolic abnormalities

Authors (year)	Animal model	Metabolic model/condition	Moringa preparation	Duration	Dose	Result
López et al., 2018 47	Male Wistar rats	High-fat diet + 10% fructose-induced metabolic syndrome	Leaf powder	3 weeks of treatment after MetS induction	700 mg/kg/day	↓ FG ↓OGTT ↓TG ↓AC
Irfan et al., 2022 48	Sprague-Dawley rats	High-fat diet + 20% fructose-induced metabolic syndrome	Leaf extract	30 days of treatment after 60-day induction	1000 mg/kg	↓FAS, LDL-C & TG
Daba et al., 2015 49	Rats	High-carbohydrate/high-fat diet-induced metabolic syndrome	<i>M. oleifera</i> leaf extract ± metformin	8 weeks	200 mg/kg per day	↓ FG, insulin, ↑HDL-C ↓LDL-C ↓TG ↓T-CHO
Ezzat et al., 2020v 40	Obese rats	High-fat diet-induced obesity	70% ethanolic leaf extract	1 month	200 , 400 mg/capsule	↓Weight
Li et al., 2022 50	C57BL/6J mice	High-fat diet-induced obesity	Leaf polysaccharide	12 weeks	100, 200 and 400 mg/kg	↓weight ↑HDL-C ↓LDL-C ↓TG ↓T-CHO

El-Shehawi et al., 2021 (51)	Male Wistar rats	High-fat diet-induced obesity	Ethanollic leaf extract	14 weeks	300 mg/kg	↑TG ↑LDL ↑TC ↑insulin ↑glucose ↑leptin ↓Adiponectin ↓HDL
Mabrouki et al., 2020 (52)	Rats	High-fat diet-induced obesity	Leaf methanolic extract	12 weeks	200 , 400 mg/kg	↑weight ↑TC ↑TG ↓HDL-C
Metwally et al., 2017 (41)	Obese female rats	High-fat diet-induced obesity	Leaf ethanollic extract	12 weeks	600 mg/kg	↓Weight
Elabd et al., 2018 (53)	Swiss mice	High-fat diet-induced obesity	Leaf aqueous extract/juice preparation	12 weeks	200 mg/kg	↓Weight ↑HDL-C ↑Bifidobacteria ↓TG ↓LDL ↓TC
Aekthammarat et al., 2019 (54)	Male Wistar rats	L-NAME-induced hypertension	Leaf extract	3 weeks	30 and 60 mg/kg	↓high blood pressure
Aekthammarat et al., 2020 (55)	Male Wistar rats	Experimental hypertension/vascular dysfunction	Leaf aqueous extract	6 weeks	1-30 mg/kg	↓high blood pressure
Experimental diabetes						
Olayaki et al., 2015 (56)	Alloxan- induced diabetic rats		Leaf methanolic extract	6 weeks	300 or 600 mg/kg	↑ HDL ↓ LDL ↓TG ↓TC
Experimental type 2 diabetes						
Tang et al., 2017 (57)	Type 2 diabetic mice		Leaf extract	5 weeks	150 mg/kg	↓ fasting plasma glucose ↓ TG ↓LDL ↑insulin levels
Oganezi., 2023 (58)	Wistar rats	Streptozotocin (STZ) induced type 2 diabetic W	MO leaves	28 days	50 g	↑ blood glucose
Aljazzaf et al., 2023 (59)	Male/female mice	Alloxan-induced diabetes	Combined leaf and seed extracts	3 months	500 mg/kg body weight of MO leaves	↓ blood glucose ↓TC ↓TG
Omabe et al., 2014 (60)	Albino rats	Alloxan-induced diabetes	Ethanollic leaf extract	5-6 days	200 mg/kg	↓ blood glucose level
Experimental diabetes						
Villarruel-López et al., 2018 (61)	Male Sprague- Dawley rats		<i>M. oleifera</i> preparation	8 weeks	50 mg	↓ blood glucose level ↑ Weight
Genetic type 2 diabetes						
Sun et al., 2019 (62)	Male db/db mice		Leaf extract	8 weeks	120 mg/kg	↓ body weight ↓FBG ↓TG ↓TC ↓LDL ↑ HDL
Chinedu et al., 2014 (63)	Diabetic rats	Streptozotocin- induced insulin resistance/diabetes	diabetic rats	-	250 mg/kg and 500 mg/kg	↓ blood glucose

Mechanisms of Action

Most of the studies have shown the positive effects of MO leaves on metabolic syndrome and its risk factors, but some have indicated its ineffectiveness. Several

mechanisms can be related to the effects of MO ingredients and the risk factors of metabolic syndrome, some of which are presented in [Table 4](#).

Table 4. The possible mechanism of effect of *Moringa oleifera* on each Metabolic syndrome factors

Obesity
MO, containing chlorogenic acid, acts as a potent fat burner, effectively balancing fat metabolism and reducing belly fat (64, 65). Additionally, MO polysaccharides play a crucial role in reducing fat accumulation caused by a high-fat diet, thus preventing weight gain (50). Moreover, MO leaves promote the apoptosis process in fat cells by upregulating genes associated with the apoptosis pathway, such as BAX and BCL2, leading to a reduction in fat cell formation (66). Furthermore, the aqueous extract of MO leaf aids in reducing inflammation associated with obesity caused by a high-fat diet by modulating intestinal bacteria. These effects may be attributed to increased levels of lactobacilli and interleukin-6, as well as decreased levels of intestinal bifidobacteria ($P < 0.05$). Consequently, they significantly contribute to the pathophysiological regulation of weight gain (53). By integrating these mechanisms, MO demonstrates its potential in combating obesity through various pathways, making it a promising candidate for addressing this crucial aspect of metabolic syndrome (44).
Diabetes
MO leaves contain fiber and flavonoids, exhibiting antioxidant activity that prevents hyperglycemia by inhibiting the activity of pancreatic alpha-amylase and intestinal alpha-glucosidase (67). This mechanism helps regulate blood glucose levels and contributes to improved diabetes management. Another important mechanism of MO leaf in improving diabetes is by increasing the activity of antioxidants against reactive oxygen species (ROS) and reducing oxidative stress (68). These antioxidant compounds, such as carotenoids, ascorbic acid, phenolics, and flavonoids (69, 70), play a crucial role in protecting pancreatic beta cells from oxidative damage and preserving their function. Additionally, they aid in reducing overall oxidative stress in the body, which is beneficial for diabetes management. Furthermore, MO leaf's chlorogenic acid inhibits glucose-6-phosphate translocase in the liver, leading to a decrease in glycogenolysis and hepatic gluconeogenesis (64, 71). This mechanism contributes to the regulation of hepatic glucose production, thus helping to maintain blood glucose levels within a healthy range. In summary, MO showcases multiple mechanisms that contribute to its potential in managing diabetes. Its antioxidant properties, inhibition of alpha-amylase and alpha-glucosidase activity, and regulation of hepatic glucose production make it a valuable natural resource for supporting diabetes control and prevention.
Blood Bressure
MO leaves contain substantial amounts of potassium, whose biological properties, including anti-inflammatory, anti-cancer, heart protection, and hypocholesterolemic effects, have been confirmed by numerous studies (72-77). Additionally, MO flavonoids play a vital role in maintaining heart health by combating free radicals, regulating nitric oxide, enhancing the effects of vitamins, and promoting optimal blood flow (78). The aqueous extract of MO has demonstrated its ability to suppress the production of O₂ in blood vessels, increase the antioxidant activity of CAT and SOD, and reduce the level of MDA in plasma and thoracic aorta. These properties effectively lower blood pressure in hypertensive rats using L-NAME (54). Furthermore, the aqueous extract of MO reduces the inhibitory effect of NO-sensitive guanylyl cyclase, 1H-[1,2,4]-oxadiazolo-[4,3-a]-quinoxalin-1-one. This relaxation of smooth muscles in the blood vessels contributes to lowering blood pressure (55). Moreover, MO bioactive peptides exhibit inhibitory effects on the activity of angiotensin-converting enzyme (ACE) and double renin inhibitory peptides Leu-Gly-Phe-Phe (LGF) and Gly-Leu-Phe-Phe (GLFF). Consequently, MO leaves are employed in the treatment of high blood pressure (79). Additionally, MO leaf extract can inhibit the influx of calcium mediated through the Gq signaling pathway and depolarization voltage-dependent calcium channels, resulting in a relaxing effect on the smooth muscles of blood vessels (p-value = 0.0008) (42). Furthermore, the methanolic extract of MO leaf inhibits ACE activity in the hippuryl-L-histidyl-L-leucine (HHL) substrate, indicating its potential as a natural blood pressure inhibitor (42, 80). By exhibiting a range of mechanisms, MO proves to be a valuable natural remedy in managing blood pressure and promoting cardiovascular health.
Hyperlipidemia
MO flavonoids reduce blood fat by inhibiting HMG-CoA reductase in the path of cholesterol synthesis (81). Additionally, phenolic acids can reduce triglycerides by increasing the activity of lipolytic enzymes such as liver lipase and plasma lipoprotein lipase (82). Moreover, MO flavonones can increase cholesterol receptors and reduce plasma LDL levels by inhibiting cholesterol acyltransferase in the liver (83). The presence of diverse biological phytochemical compounds in MO helps maintain lipid homeostasis. These useful phytochemical compounds include chrysin-7-glycoside, quercitrin, isoquercitrin, and more (59). Furthermore, MO can exhibit antihyperlipidemic activity by inhibiting lipogenesis through the activation of the AMPK signaling pathway (84). The methanolic extract of MO leaves (MEOL) significantly increases the levels of catalase and reduced glutathione, while reducing the levels of superoxide dismutase (SOD) and lipid peroxidation (LPO). As a result, MEOL proves to be an effective agent in the treatment of oxidative stress and hyperlipidemia (85). By harnessing these multiple mechanisms, MO demonstrates its potential in managing hyperlipidemia and promoting healthy lipid profiles.

Conclusion

Current evidence suggests that MO has biologically plausible effects on several metabolic abnormalities relevant to MetS; however, the clinical evidence remains insufficient to establish a consistent therapeutic effect on the syndrome as a whole. The available literature is characterized by substantial heterogeneity in botanical preparations, doses,

intervention durations, participant characteristics, and concomitant treatments. Importantly, much of the human evidence has been generated in patients with established T2D or in populations with isolated metabolic abnormalities and therefore represents indirect evidence for MetS.

Preclinical studies provide extensive mechanistic support for effects on glucose metabolism, lipid homeostasis, adiposity, oxidative stress, endothelial function, and blood pressure. Nevertheless, these findings should not be considered equivalent to clinical evidence. Recent randomized clinical evidence, including studies specifically involving patients with metabolic syndrome, warrants greater attention; however, the overall evidence remains limited by small samples, short follow-up, methodological heterogeneity, and uncertainty regarding the optimal preparation and dose.

Accordingly, MO should currently be regarded as a promising but insufficiently established adjunctive nutritional or phytotherapeutic candidate rather than an evidence-based treatment for metabolic syndrome. Future research should prioritize adequately powered, rigorously blinded randomized controlled trials in well-characterized MetS populations, using chemically characterized and standardized preparations, prespecified clinically relevant outcomes, longer follow-up, systematic adverse-event monitoring, and transparent reporting of concomitant medications and lifestyle interventions.

Limitations of the Current Evidence and of the Present Review

Several limitations constrain interpretation of the evidence. First, the number of well-designed human studies is small, and many have small sample sizes. Second, intervention periods are usually short, commonly lasting weeks rather than months or years, so long-term efficacy and safety remain uncertain. Third, most human studies do not directly evaluate clinically defined MetS; much of the evidence comes from diabetes or isolated risk factors and therefore has limited directness. Fourth, substantial heterogeneity exists in botanical preparation, extraction method, dose, intervention duration, baseline metabolic status, and concomitant treatment. Fifth, many studies provide incomplete information regarding randomization, allocation concealment, blinding, adherence, dietary intake, or adverse events.

The review itself also has limitations. The search was structured and transparent but was not registered as a systematic review or meta-analysis, and the original search was restricted to English-language peer-reviewed publications. Google Scholar was included as a supplementary source, which increases breadth but reduces reproducibility compared with database-specific systematic searches. Because the evidence was heterogeneous, no quantitative pooled estimate was generated in this review, and formal certainty grading was not performed across all the evidence included. Finally, literature continues to evolve rapidly, particularly with the publication of recent meta-analyses and direct MetS trials; therefore, the conclusions should be considered current to the updated literature identified for this revision rather

than definitive.

Clinical Implications and Future Directions

The review of current studies on the effects of MO on MetS risk factors presents promising findings. Despite limited human studies directly focusing on the impact of MO on metabolic syndrome, numerous mechanisms have been identified that suggest its potential in mitigating various risk factors associated with the syndrome.

Ethics approval and consent to participate

N.A

Consent for publication

N.A

Availability of data and materials

No datasets were generated or analyzed during the current study.

Competing interests

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Authors' contributions

N.T.T. and S.Y. contributed to the conceptualization of the study, literature search, and preparation of the initial draft. S.B. contributed to the literature review and data collection. M.R. contributed to the critical revision of the manuscript and interpretation of the literature. E.N.E. contributed to the conceptualization and supervision of the study and critically revised the manuscript. All authors read and approved the final version of the manuscript.

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