INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

OPEN ACCESS

Review Article

Moringa oleifera: Phytochemistry, Pharmacological Potential and Its Role in Wound Healing – A Comprehensive Review

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ARTICLE INFO

Published: 25 Nov 2026
Keywords:
Moringa oleifera, phyto-chemistry, pharmacological activity, wound healing, antioxidant, anti-inflamma-tory
DOI:
10.5281/zenodo.24527994

ABSTRACT

Moringa oleifera Lam. (Moringaceae) is a fast-growing plant commonly known as drumstick or horseradish tree, valued for its nutritional and medicinal properties and used for a long time in traditional medicine in South Asia, Africa and other tropical regions. Its leaves, seeds, pods, flowers, bark and roots contain glucosinolates, isothio-cyanates, flavonoids such as quercetin, kaempferol, rutin and vicenin-2, phenolic acids, alkaloids, sterols, vitamins, minerals, essential amino acids and fatty acids. These con-stituents are responsible for reported antioxidant, anti-inflammatory, antimicrobial, antidiabetic, hepatoprotective, cardioprotective and tissue protective activities, and among these the potential role of *M. oleifera* in wound healing has attracted increasing research interest. Wound healing is a complex, coordinated process involving haemostasis, inflammation, proliferation and tissue remodelling; oxidative stress, in-fection, persistent inflammation and conditions such as diabetes can delay it. Experi-mental wound models have shown that extracts of *M. oleifera* and its bioactive constituents may enhance tissue repair via wound contraction, fibroblast proliferation and migration, collagen deposition, hydroxyproline formation and tissue strength, while reducing oxidative stress, inflammatory responses and microbial growth. At the molec-ular scale, these effects have been associated with modulation of TNF-α, IL-1β and IL-6, enhancement of endogenous antioxidant defences involving superoxide dismutase, catalase and glutathione, and regulation of the growth factors TGF-β1 and VEGF in-volved in tissue regeneration and angiogenesis. The wound healing potential of *M. oleifera* has also been evaluated in experimental models of diabetic wounds.

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

INTRODUCTION:

Wound care has been documented since before the time of Galen (AD 129–200), when burn injuries and warfare were among the main causes of wounds. Galen held that four components should be taken into account when treating any infection: God was the most important factor, followed in decreasing order of significance by the doctor, medication and patient. He is particularly famous for his "laudable pus" idea, which held that a wound that becomes infected and discharges pus should be allowed to continue in order to heal properly.[1] A clay tablet from 2200 BC, one of the oldest known medical manuscripts, revealed the "three healing gestures"—washing the wounds, making the plasters, and bandaging them—perhaps for the first time.[2]

If left untreated, wounds can become chronic and lead to serious problems, including amputation, which severely reduces quality of life. Chronic wounds take longer to heal because the stages of wound healing fail and do not occur regularly, and prolonged exposure to bacteria makes them more prone to microbial infections. Common pathogens such as the fungus *Candida albicans* and the bacterium *Staphylococcus aureus*[3] often infiltrate chronic wounds. Pressure ulcers, diabetic foot ulcers and non-healing traumatic wounds are general types of chronic wounds which cause a significant financial burden, a decline in health-related quality of life and a rise in emotional stress. It is therefore important to give proper wound care, which includes regular dressing and debridement.[4]

The *Moringa oleifera* plant is a fast-growing medicinal plant of the Moringaceae family, typically known as the drumstick tree, horseradish tree, miracle tree, or mother's best friend tree. Its therapeutic use in the Indian subcontinent dates back to antiquity. In spite of the widely held "belief" in its health benefits, the interest of the international biomedical community in its medicinal potential has been rather tepid.[5] Moringa has long been used in herbal medicine by Indians and Africans, and the presence of phytochemicals makes it a good medicinal agent.[6]

2. WOUNDS AND THEIR CLASSIFICATION:

2.1. Wound

A wound is a disruption or damage to the normal anatomical structure and function. It can be as simple as a break in the epithelial integrity of the skin or a more complex injury involving other structures such as tendons, muscles, arteries, nerves, parenchymal organs or even bone, extending into subcutaneous tissue. Wounds can be caused by pathological processes that begin internally or externally within the implicated organ. Irrespective of cause and form, wounding damages the tissue and disrupts the local environment within it; the physiological response to the noxious factor results in bleeding, vessel contraction with coagulation, activation of complement and an inflammatory response.[7]

Abrasions are typically caused by friction, such as falling on rough surfaces. Deep cuts or tears caused by sharp objects are particularly prone to infection because of their depth, surgical cuts are made by medical professionals, and avulsion injuries can result in significant tissue loss. The impact of blunt or sharp objects can result in cuts, bruises or abrasions. Symptoms depend on the type and severity of the wound: pain can range from mild discomfort to severe pain depending on the extent of tissue damage, and active bleeding may occur in open wounds, particularly in cuts or lacerations that damage blood vessels.[8]

Wounds can be classified according to various criteria. Time is an important factor in injury management and wound repair; thus, wounds are clinically categorized as acute or chronic according to their time frame of healing.

2.2. Acute Wounds

Wounds that repair themselves and proceed normally by following a timely and orderly healing pathway, with the end result of both functional and anatomical restoration, are classified as acute wounds. The time course of healing usually ranges from 5 to 10 days, or within 30 days. Acute wounds can be acquired as a result of traumatic loss of tissue or a surgical procedure.[7] For example, an operation to remove a soft tissue tumour located in the skin and underlying parenchyma can result in a large but non-contaminated wound that

cannot be healed by primary intention, due to the large defect within the tissue. Traumatic wounds may involve only the soft tissue or be associated with bone fractures. These combined injuries have been classified by the AO Foundation (Arbeitsgemeinschaft fuer Osteosynthesefragen/Association for the Study of Internal Fixation) system, one of the most comprehensive and widely used, which covers closed and open fractures with assessment of skin, muscle, tendon and neurovascular injuries. Its benefit is that the extent of damage to muscles and tendons is taken into account, as it determines the prognosis of the injured limb.[7]

2.3. Chronic Wounds

Chronic wounds fail to progress through a normal, orderly and timely sequence of repair, or the repair process fails to restore anatomic and functional integrity after three months. Some chronic wounds can take decades to heal, contributing to secondary conditions such as depression and ultimately leading to isolation and family distress. The five-year mortality rate after developing a diabetic ulcer is approximately 40%; therefore, proper diagnosis and treatment of wounds and management of comorbidities are imperative. All wounds have the potential to become chronic. They are classified by etiology into four categories, each with its own typical location, depth and appearance: arterial, diabetic, pressure and venous ulcers. To appropriately treat a chronic wound, an understanding of its pathophysiology is critical. Despite differences in etiology, chronic wounds share certain features, including excessive levels of proinflammatory cytokines, persistent infections, formation of drug-resistant microbial biofilms, and senescent cells that do not respond to reparative stimuli.[9]

3. MORINGA OLEIFERA:

Figure 3.1: *Moringa Oleifera* Fruit, Flower, Leaves and Seeds.



3.1. Taxonomical Classification

Table 3.1: Taxonomical Classification of *Moringa Oleifera* [10]

Rank	Taxon	Rank	Taxon
Kingdom	Plantae	Class	Magnoliopsida
Sub kingdom	Tracheobionta	Subclass	Dilleniidae
Super division	Spermatophyta	Order	Capparales
Division	Magnoliophyta	Family	Moringaceae
Genus	Moringa	Species	oleifera

3.2. Botanical Description

Table 3.2: *Moringa Oleifera* Botanical Description – Synonyms [11]

Language	Synonyms
English	Drumstick tree, Horseradish tree, Ben tree
Latin	*Moringa oleifera*
Marathi	Shevaga
Sanskrit	Subhanjana
Hindi	Saguna, Sainjna

Moringa oleifera is a small, fast-growing evergreen or deciduous tree, 5 to 10 m in height. It has three pinnate compound leaves with leaflets 12–18 mm long and a yellow or white petiole without red stripes. The flowers are bisexual and white or milky white, and the pods and seeds are nearly spherical. The optimum growth temperature is 25 to 35°C. The tree is hardly affected by drought, which accounts for its adaptation to a variety of soil types.

3.3. Morphology

3.3.1. Leaves: The leaves are bipinnate or commonly tripinnate, up to 45 cm long; the leaflets are hairy, green and almost hairless on the upper surface. The twigs are hairy and green; these are compound leaves with leaflets 1–2 cm long.

3.3.2. Flowers: The fragrant, bisexual, yellowish-white flowers are borne on hairy stalks in spreading or drooping axillary panicles 10–25 cm long. Individual flowers are approximately 0.7 to 1 cm long and 2 cm broad, with five unequal, yellowish-white, thinly veined, spathulate petals, five stamens with five smaller sterile stamens, and a pistil composed of a 1-celled ovary and slender style.

3.3.3. Fruits: Fruits are tri-lobed capsules referred to as pods; they are pendulous, brown, triangular, and split lengthwise into three parts when dry, 30–120 cm long and 1.8 cm wide. Fruit production mostly occurs in March and April. Fruits contain around 26 seeds during their development stage. Immature pods are green and turn brown on maturity.

3.3.4. Seeds: Seeds are round, 1 cm in diameter, with a brownish semi-permeable seed hull with three papery wings; hulls are brown to black but can be white if kernels are of low viability. Viable seeds germinate within 2 weeks; each tree can produce around 15,000 to 25,000 seeds/year. Average weight is 0.3 g/seed.[12]

4. PHYTOCHEMISTRY OF MORINGA OLEIFERA:

Table 4.1: Phytochemical Classes Reported from *Moringa Oleifera*

Class	Compounds	Representative constituents	Main plant parts
Flavonoids	36	Quercetin, kaempferol, isorhamnetin and their glycosides; apigenin, luteolin, myricetin; daidzein, genistein	Mostly leaves
Carbamates (including thiocarbamates)	35	Niazinins, niazimicin, niaziminins, marumosides, pterygospermin	Leaves, pods, seeds, root bark
Phenols	45	Chlorogenic and caffeoylquinic acids; gallic, ferulic and caffeic acids; vanillin; catechin, epicatechin; tocopherols	Leaves, seeds, stems, root bark
Glucosinolates, isothiocyanates and nitriles	27	Glucomoringin; 4-(α-L-rhamnosyloxy)benzyl isothiocyanate; niazirin; sulforaphane	Seeds, pods, leaves, roots
Steroids	4	β-Sitosterol and its glucosides; β-sitosterone	Seeds, roots
Carotenoids	2	β-Carotene; lutein	Leaves
Others (alkaloids, amino acids, volatiles, etc.)	14	N,α-L-rhamnopyranosyl vincosamide; pyrrolemarumine rhamnoside; moringine; methionine; cysteine; p-cymene	Leaves, seeds, pods, roots, flowers

4.1. Flavonoids

A total of 36 flavonoids have been reported from *M. oleifera*, comprising 34 flavonols and two isoflavones (daidzein and genistein); almost all occur in the leaves, mainly as glucosides, and quercetin and kaempferol are the representative compounds.[13,14,15] Flavonol glycosides (glucosides, rutinosides and malonyl glucosides) of quercetin, kaempferol and isorhamnetin, in decreasing order of abundance, are found in most parts of the tree.[16] Leaf contents range from 0.07 to 1.26% for quercetin and from 0.05 to 0.67% for kaempferol, and the Indian cultivars PKM-1 and PKM-2 had higher

totals than African samples.[17] In seven Pakistani cultivars, quercetin, apigenin and kaempferol derivatives made up on average 47.0%, 20.9% and 30.0% of the total flavonoids.[18]

Radical-scavenging activity has been reported for astragalin, isoquercitrin, isorhamnetin and rutin, and antioxidant activity for quercetin, kaempferol and several leaf diglucosides, acetylglucosides and apigenin glucosides.[19] A 70% ethanol leaf extract gave the highest extractable flavonoids (6.20 g isoquercitrin equivalents per 100 g extract), a DPPH-scavenging EC50 of 62.94 µg/mL, the highest ferric-reducing power, and a significant reduction in intracellular reactive oxygen species at 100 µg/mL. A leaf extract rich in quercetin and kaempferol also showed antiproliferative and apoptotic effects in KB tumour cells, with morphological changes, reduced viability, DNA fragmentation and ROS generation.[16]

4.2. Carbamates and Thiocarbamates

A total of 35 carbamates and thiocarbamates have been reported, which are among the most distinctive constituents of the plant and occur in the leaves, pods, seeds and root bark. Niazinin A, niazimicin and niaziminin A and B were reported to be effective in managing hypertension, and four thiocarbamate glycosides lowered arterial blood pressure in normotensive anaesthetised rats. S-Methyl-N-thiocarbamate showed anti-inflammatory activity, two seed carbamates were antibacterial at 5 mg/L, and pterygospermin was also reported to have antibacterial properties.[19]

Niazimicin was an effective anti-tumour agent in a two-stage mouse skin carcinogenesis test, and niaziminin was a potent inhibitor of Epstein–Barr virus activation (IC50 1.3 µM), an effect attributed to its acetoxy group at the 4′ position. Aurantiamide acetate and 1,3-dibenzyl urea from the root inhibited production of TNF-α and IL-2, and marumoside A, together with niazirin and a sitosterol glucoside, improved psoriasis-like skin lesions in mice by suppressing Th17-related cytokines. Some carbamate and thiocarbamate derivatives from fruit and seed stimulated insulin release or lowered blood glucose, and deoxy-niazimicine from root bark was antibacterial and antifungal.[19] Thiocarbamates formed during the breakdown of glucosinolates are known for hypotensive and spasmolytic effects.[16]

4.3. Phenols and Phenolic Acids

A total of 45 phenols, including esters and glycosides, have been reported from the leaves, seeds and other parts; phenols are described as the predominant free-radical scavengers and chelating agents in the leaves. Chlorogenic acid and related caffeoylquinic acids (cryptochlorogenic, 4-O- and 5-O-caffeoylquinic acids) occur in the leaves; chlorogenic acid is described as antibacterial, antioxidant and cancer-suppressing, and as a regulator of glucose and lipid metabolism.[20] Two caffeoylquinic acid glucosides from the leaves inhibited neuraminidase and scavenged DPPH radicals.[21] Seed phenolics identified by HPLC (gallic, p-coumaric, ferulic, caffeic, protocatechuic and cinnamic acids, and vanillin) had antioxidant activity and were antibacterial against *Bacillus cereus*, *Staphylococcus aureus*, *Escherichia coli* and *Yersinia enterocolitica*, while epicatechin and catechin were antioxidant and bactericidal.[22] Two fruit phenolics were tested in cyclooxygenase (COX-1 and COX-2) inhibition assays and were reported to stimulate insulin release.[23] Extraction with 70% ethanol gave the highest extractable phenolics from leaves, and phenolic-rich seed flour extracts and seed flavonoid extracts were antimicrobial.[16]

4.4. Glucosinolates, Isothiocyanates and Nitriles

Liu et al. (2022) catalogue 27 glucosinolates and related nitriles and isothiocyanates. Saini et al. (2016) identify glucomoringin, 4-O-(α-L-

rhamnopyranosyloxy)-benzylglucosinolate, as the predominant glucosinolate in the stem, leaves, flowers, pods and seeds, whereas benzyl glucosinolate (glucotropaeolin) is most prominent in the roots; the highest glucosinolate contents are in the leaves and seeds. The endogenous enzyme myrosinase converts glucosinolates to isothiocyanates, nitriles and thiocarbamates, which are known for hypotensive and spasmolytic effects.[16,19]

Many of the biological activities attributed to the plant are linked to these breakdown products. Niazirin was reported to be anti-inflammatory, and a glucosylrhamnosyl phenylacetonitrile from fruit was antimicrobial.[24] Four pod isothiocyanates reduced nitric oxide production and iNOS expression and showed antibacterial activity.[25] The main isothiocyanate, 4-(α-L-rhamnosyloxy)benzyl isothiocyanate, inhibited Epstein–Barr virus early antigen activation (IC50 32.7 μM), was a potent inhibitor of STAT5 and NF-κB signalling, and protected against secondary injury in a spinal cord injury model[26]; it was also antimicrobial, with minimum bactericidal concentrations of 40 μmol/L for *Mycobacterium phlei* and 56 μmol/L for *Bacillus subtilis*. Sulforaphane was reported to have chemopreventive potential acting through STAT5[27], and isothiocyanates inhibited human platelet aggregation, which required the isothiocyanate group and a chain length of 6 or 8.[28] In Saini et al. (2016), an aqueous leaf extract containing 1.66% isothiocyanates and 3.82% total polyphenols reduced the expression of iNOS and IL-1β in RAW macrophages, suggesting a role in alleviating low-grade inflammation associated with chronic disease. An isothiocyanate-rich leaf extract was also reported to affect weight gain, insulin resistance and hepatic gluconeogenesis in mice.[16]

4.5. Steroids

Liu et al. (2022) report four steroids: β-sitosterol, two of its glucosides (including a 6′-O-oleoyl glucoside) from seeds, and β-sitosterone from roots. β-Sitosterol from the seeds was found to have hypotensive activity[29], and phytosterols from the leaves suppress intestinal cholesterol absorption[30]; increased faecal excretion of neutral steroids has been proposed to explain the cholesterol-lowering effect seen in hypercholesterolaemic rabbits.[31] Two sitosterol glucosides inhibited Epstein–Barr virus early antigen activation (IC50 about 70 μM) and were proposed as anti-tumour agents.[32] Saini et al. (2016) do not treat steroids as a separate class, although they list phytosterols among the bioactive compound groups responsible for the biological activities of the plant.

4.6. Carotenoids

Liu et al. (2022) identify β-carotene and lutein in the leaves.[33] β-Carotene is a precursor of vitamin A with reported anticancer, antioxidant and immune-enhancing activity, and it was linked to the hepatoprotective effect of leaf extracts[34,35]; lutein has anticancer and antioxidant activity, is useful in cardiovascular disease, and protects the eyes and prevents macular degeneration.[36] All-E-lutein is the major carotenoid, accounting for 53.6% of total carotenoids in foliage and 52.0% in immature pods, with minor amounts of all-E-luteoxanthin, 13-Z-lutein, all-E-zeaxanthin and 15-Z-β-carotene.[37] Total carotenoids are highest in leaves (44.30–80.48 mg/100 g fresh weight), followed by immature pods (29.66 mg/100 g) and flowers (5.44 mg/100 g); among Indian cultivars, Bhagya (KDM-1) had the highest all-E-zeaxanthin, all-E-β-carotene and total carotenoids. Dehydration significantly degrades carotenoids, whereas foliar application of chitosan, carboxy-methyl chitosan, methyl jasmonate and salicylic acid can enhance them in field-grown trees.[16]

5. PHARMACOLOGICAL POTENTIAL OF MORINGA OLEIFERA:

5.1. Wound Healing Activity: *Moringa oleifera* Lam. has demonstrated promising wound-healing potential in preclinical studies owing to its cell-proliferative, anti-inflammatory, antioxidant, antimicrobial and tissue-repair properties. In the 2026 scoping review by Chow and Chin, 47 studies published between 2014 and June 2025 were evaluated. Extracts of different plant parts, particularly the leaves, promoted wound contraction and fibroblast proliferation, and several studies reported increased tensile strength, improved collagen deposition and a reduced period of epithelialization. These effects may be attributed to constituents such as the flavonoids quercetin and kaempferol and phenolic acids, which may reduce oxidative stress and limit prolonged inflammation at the wound site. The

review also describes antibacterial activity against wound-associated microorganisms including *Staphylococcus aureus*, *Escherichia coli* and *Pseudomonas aeruginosa*, although effects varied among studies, together with increased collagen content and collagen type I expression in experimental wound models. Hydrogel formulations, ointments and extract-loaded nanofibrous dressings have been investigated as topical delivery systems. However, the evidence remains predominantly based on *in vitro* and animal studies, and further clinical research is required to establish efficacy, safety, optimal concentration and mechanism of action in human wound healing.[38]

5.2. Antibacterial and Antifungal Activity: *Moringa oleifera* demonstrates antibacterial activity against *Escherichia coli*, *Staphylococcus aureus* and *Pseudomonas aeruginosa*, to which isothiocyanates and flavonoids contribute, and antifungal activity against *Aspergillus niger* and *Candida albicans*. This broad-spectrum antimicrobial action underscores its traditional use in wound care and infection management and its potential use in natural antimicrobial formulations. [39]

5.3. Antidiabetic Activity: *Moringa oleifera*, particularly its leaf extracts, exhibits promising antidiabetic activity, with evidence predominantly derived from preclinical studies. Experimental models show reduced fasting and plasma blood glucose, improved glucose tolerance and insulin sensitivity, and favourable modulation of lipid metabolism. These effects are associated with enhancement of endogenous antioxidant defences, including superoxide dismutase (SOD), catalase (CAT), glutathione (GSH), glutathione peroxidase (GPx) and glutathione-S-transferase (GST), with reduced oxidative stress markers such as malondialdehyde (MDA); with improved insulin signalling through the PI3K/AKT and AMPK pathways and increased expression of insulin receptor, IRS-1 and GLUT4; and with reduction of pro-inflammatory mediators including TNF-α, IL-1β, IL-6, COX-2, iNOS and MCP-1. These effects may contribute to improved diabetic wound healing, as leaf extracts have been reported to enhance wound contraction and tissue regeneration while reducing inflammatory cytokines and increasing VEGF expression in diabetic animal models. Clinical evidence remains limited and inconsistent, highlighting the need for larger, well-controlled clinical studies using standardized preparations.[40]

5.4. CNS Activity: *Moringa oleifera* exhibits promising neuroprotective activity through antioxidant, anti-inflammatory and neurotransmitter-modulating mechanisms, attributed to flavonoids, phenolic compounds and isothiocyanates. It increases the activities of SOD, CAT and GSH while reducing lipid peroxidation, and suppresses inflammatory mediators such as nitric oxide (NO), prostaglandin E_2 (PGE_2), IL-6, IL-1β, TNF-α, NF-κB, COX-2 and iNOS. Root extract has demonstrated central inhibitory and anticonvulsant effects by inhibiting penicillin-induced seizures and altering neurotransmitter levels, particularly increasing serotonin (5-HT) and decreasing dopamine (DA) in several brain regions. The plant also exhibits cholinergic activity by inhibiting acetylcholinesterase and increasing acetylcholine levels in the cortical–hippocampal region, suggesting potential memory-protective and cognitive-enhancing effects.[41]

5.5. Anti-ulcer Activity: Alkaloids, flavonoids, rutin, steroids and other phytoconstituents in *Moringa oleifera* leaves explain their gastroprotective qualities; quercetin and rutin have been associated with antiulcer and gastro-cytoprotective effects, respectively. Ethanolic extracts showed significant antiulcer activity in ethanol-induced and pylorus ligation-induced ulcer models, and in ibuprofen-induced gastric ulcer models bisphenols and flavonoids in the leaves decreased the ulcer index, duodenal ulceration and stress-induced ulcer development. The effects are linked to reducing free radicals, neutralizing stomach acid, increasing gastric juice pH and decreasing gastric acid production, while flavonoids can improve gastric mucosal microcirculation and capillary resistance. More research is needed to identify the specific bioactive compounds and how they work.[42]

5.6. Anti-Inflammatory Activity: *Moringa oleifera* exhibits anti-inflammatory activity owing to its flavonoids, polyphenols, isothiocyanates and phenolic acids. Quercetin and kaempferol may reduce inflammation by inhibiting cyclooxygenase (COX) and lipoxygenase (LOX) enzymes, and isothiocyanates may suppress the NF-κB signalling pathway, reducing

the release of TNF-α and IL-1β; the antioxidant properties also help reduce oxidative stress that can contribute to inflammation. Animal studies of different plant parts, particularly leaves and seeds, report reductions in inflammatory markers and improvements in experimental conditions such as colitis and liver inflammation, although further clinical studies are needed to confirm effectiveness in humans.[43]

5.7. Anti-Microbial Activity: The antibacterial activity of *Moringa oleifera* is mainly attributed to isothiocyanates such as moringin, formed from glucomoringin through enzymatic activity. A systematic review of 12 in-vitro studies reported variable antibacterial effects of leaf and seed extracts, depending on plant part, extraction solvent and concentration. Leaf extracts showed limited and inconsistent activity against bacteria such as *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, while seed extracts, particularly those containing the outer seed coat, showed stronger inhibitory effects in some studies. Moringin may act by damaging bacterial cell membranes and cell walls, inducing oxidative stress and interfering with DNA replication, and sterols and fatty acid derivatives may also contribute. Variations in extraction methods, phytochemical composition and experimental procedures make results difficult to compare, and current evidence is insufficient to establish effectiveness as a standalone antibacterial treatment.[44]

5.8. Hepatoprotective Activity: In a mouse model of acetaminophen (APAP)-induced liver injury, hydroethanolic leaf extract reduced elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT), while improving liver tissue architecture and reducing hepatic necrosis. Three major mechanisms were associated with this protection: reduced expression of cytochrome P450 enzymes (CYP2E1, CYP1A2 and CYP2A6) involved in forming the toxic acetaminophen metabolite NAPQI; enhanced antioxidant defence through activation of the Nrf2/ARE signalling pathway, with reduced oxidative stress and lipid peroxidation (quercetin, kaempferol and apigenin may contribute); and reduced inflammation through lower TNF-α, IL-1β and IL-6 with restored levels of the anti-inflammatory cytokine IL-10. The evidence is primarily based on animal studies, and further research is required to establish safety and efficacy in humans.[3]

5.9. Anti-Cancer Activity: The anticancer activity of *Moringa oleifera* Lam. in experimental studies is mainly attributed to glucosinolates, isothiocyanates, flavonoids and phenolic compounds, which may inhibit cancer cell growth through apoptosis, cell-cycle arrest and modulation of survival and proliferation pathways. Glucosinolates are converted into isothiocyanates by myrosinase when plant tissues are disrupted. Allyl isothiocyanate (AITC) induced G2/M-phase cell-cycle arrest and apoptosis in human prostate cancer cells, with reduced levels of CDK1, CDC25B and CDC25C, and benzyl isothiocyanate (BITC) reduced tumour growth in experimental pancreatic cancer models and influenced the PI3K/AKT and mTOR pathways. Leaf and bark extracts showed inhibitory effects against breast and colorectal cancer cells (MDA-MB-231 and HCT-8), with reduced proliferation, increased apoptosis and cell-cycle arrest. These findings are primarily laboratory- and animal-based; *M. oleifera* should be considered a potential source of investigational anticancer compounds rather than an established cancer treatment.[45]

6. ROLE OF MORINGA OLEIFERA IN WOUND HEALING:

6.1. Overview of the Wound Healing Process

Cutaneous wound repair is a dynamic and tightly regulated process involving cellular, humoral and molecular mechanisms, conventionally divided into four partly overlapping phases. Hemostasis establishes a platelet–fibrin clot that seals the wound and releases cytokines and growth factors. In the inflammatory phase, neutrophils and macrophages clear debris and microorganisms, generating reactive oxygen species (ROS) and pro-inflammatory cytokines. During proliferation, fibroblasts, endothelial cells and keratinocytes build granulation tissue, form new blood vessels (angiogenesis) and restore the epithelial barrier (re-epithelialisation). In remodeling, the provisional matrix is replaced by organised collagen, the wound contracts and tensile strength gradually increases. Disruption at any stage, whether through prolonged inflammation, excessive oxidative stress, infection or

hyperglycemia, can lead to chronic non-healing wounds or abnormal scarring. Because each phase is a potential point of failure, a multi-component botanical such as *M. oleifera* may in principle act at several points simultaneously, which is the rationale for evaluating it as a wound-healing agent.[46]

6.2. Plant Parts and Preparations Investigated

Wound-related research has not been distributed evenly across the plant. Table 6.1 summarises the plant parts studied and the preparations used; leaves dominate the literature, followed by seed oil, whereas data for other parts are sparse.

Table 6.1: Plant Parts of *Moringa Oleifera* Investigated in Wound-Related Research.

Plant part	Typical preparations	Wound-related use and findings
Leaves	Aqueous, methanolic, ethanolic and ethyl acetate extracts; standardised fractions; powders	The most extensively studied part. Used in excision, abrasion, infected, diabetic and oral (palatal and gingival) wound models, and incorporated into ointments, gels, films and nanomaterials [47,48,49].
Seeds and seed ("ben") oil	Cold-pressed seed oil; oleic acid-rich fraction	Seed oil shortened healing time in chronic (immunosuppressed and diabetic) skin wounds in mice and improved collagen content and myofibroblast numbers [50].
Flowers, bark, roots, sap and pods	Traditional preparations	Used in traditional medicine [51], but comparatively little wound-specific experimental data is available.

6.3. Bioactive Constituents Relevant to Wound Healing

Phytochemical profiling indicates that *M. oleifera* leaves are rich in vitamins, minerals, fatty acids and polyphenolic compounds, including quercetin, kaempferol and vicenin-2, which have been linked to the wound-healing process.[46] More broadly, a wide variety of polyphenols, phenolic acids, flavonoids and glucosinolates, and possibly alkaloids, are believed to underlie the biological activities of the plant[51], with carotenoids and alkaloids also reported as constituents of interest.[52] Hydrogel studies have highlighted gallic acid and rutin among the key constituents of wound-healing extracts.[53] The principal classes and their proposed relevance to tissue repair are summarised in Table 6.2.

Table 6.2: Major Bioactive Constituents of *M. Oleifera* and Their Proposed Relevance to Wound Healing.

Class	Representative compounds	Main source	Wound-relevant activity (reported or proposed)
Flavonoids	Quercetin, kaempferol, rutin, vicenin-2	Leaves	Free-radical scavenging, anti-inflammatory activity and support of proliferative events; quercetin, kaempferol and vicenin-2 have been specifically linked to wound healing in a review of the plant [46].
Phenolic acids	Gallic acid, chlorogenic acid	Leaves, flowers	Antioxidant and antimicrobial activity; gallic acid is highlighted as a marker compound in hydrogel formulations [53].
Glucosinolates and isothiocyanates	Glucomoringin and its hydrolysis product moringin	Seeds, leaves	Associated with anti-inflammatory and antimicrobial effects; glucosinolates are among the compound groups proposed to mediate the plant's activities [51].
Vitamins	Vitamin A (β-carotene), vitamin C, vitamin E	Leaves	Antioxidant protection; vitamin C is a cofactor for collagen synthesis and maturation [48]; vitamin A supports epithelial integrity.
Minerals	Calcium, iron, zinc, magnesium, potassium	Leaves	Iron and zinc act as cofactors in collagen formation and cell proliferation; iron and oxygen availability also influence collagen maturation [48].
Proteins and amino acids	Essential amino acids, protein fractions	Leaves, seeds	Provide substrates for new tissue and extracellular matrix formation.

Class	Representative compounds	Main source	Wound-relevant activity (reported or proposed)
Fatty acids	Oleic acid (abundant in seed oil)	Seeds	Seed oil and oleic acid accelerated chronic wound repair and increased collagen content in diabetic wounds [54].
Carotenoids and alkaloids	β-carotene, lutein	Leaves	Antioxidant activity and provitamin A function [52].

The chemical profile of *M. oleifera* is not fixed. The type and amount of constituents depend on the plant part, cultivar and geographic origin, harvest stage, post-harvest processing and extraction solvent, so that aqueous, methanolic and ethyl acetate extracts can differ considerably in composition and potency. Standardisation of products has been identified as a recurring problem for the field.[51]

6.4. Mechanisms of Action in Relation to the Phases of Wound Healing

Organising the available evidence by phase shows which stages of repair *M. oleifera* appears to influence and where the data remain thin. Table 6.3 provides an overview, and the sections that follow describe the findings in more detail.

6.4.1. Hemostasis and Inflammation

Dedicated studies of the effect of *M. oleifera* on hemostasis and clot formation are scarce, and most wound research has concentrated on the later phases. The anti-inflammatory evidence is stronger. In diabetic rats with excisional wounds, untreated animals showed higher expression of TNF-α, IL-1β and IL-6 than animals receiving aqueous leaf extract, and the extract-treated group achieved higher histopathological healing scores. Excessive or prolonged expression of these cytokines is a hallmark of chronic wounds, so dampening them may help move the wound from the inflammatory to the proliferative phase. Mechanistic work in other inflammatory models points towards modulation of NF-κB-related signalling, but wound-specific confirmation of this pathway is limited and should be regarded as a hypothesis rather than a demonstrated mechanism.[48]

6.4.2. Proliferation: Fibroblasts, Angiogenesis and Re-epithelialisation

The proliferative phase is where most of the positive effects of *M. oleifera* have been recorded. In a rat palatal wound model, three days after injury the fibroblasts of Moringa-treated wounds had repopulated the wound area significantly more than in povidone-iodine or vehicle controls, and a 4% leaf extract gel produced the highest fibroblast synthesis and collagen deposition.[55] The ethyl acetate fraction has also been examined for wound-healing properties in normal human dermal fibroblasts.[56] In MRSA-infected diabetic wounds, topical extract increased capillary density and shortened the period of epithelisation, with histology showing greater epidermal height, angiogenesis and collagen deposition compared with infected controls.[49] Vascular endothelial growth factor (VEGF) is the central regulator of angiogenesis, and a study in Wistar rats evaluated topical gels containing 5%, 10% and 15% leaf extract over eight days using wound closure rate and VEGF gene expression as endpoints.[57] Together these findings suggest that the extract supports granulation tissue formation and re-epithelialisation, although the molecular signalling responsible has not been fully mapped.

6.4.3. Remodeling: Collagen Deposition and Tensile Strength

Collagen content and wound strength are the principal endpoints of the remodeling phase. In diabetic rats treated with a standardised leaf extract hydrocolloid film, hydroxyproline (a surrogate marker of collagen) at day 21 was higher in the 0.5% film group (63.17 ± 8.69 µg/µL) than in diabetic untreated controls (46.85 ± 14.53 µg/µL).[58] Seed oil increased collagen content in diabetic wounds and restored myofibroblast numbers in immunosuppressed and diabetic animals[54], which is relevant to wound contraction. The scoping review additionally reports increased tensile strength after leaf extract treatment.[47] Long-term outcomes such as scar quality and mature collagen architecture have been studied far less than early closure rates.

6.4.4. Antioxidant Activity

Persistent ROS impair fibroblast function, degrade the extracellular matrix and prolong inflammation, particularly in diabetic wounds. Leaf extracts show the greatest antioxidant activity among the plant parts.[51] In infected diabetic wounds, leaf extract treatment was associated with increased activities of endogenous antioxidant enzymes alongside improved contraction and epithelisation.[49] The antioxidant effect is thought to arise from the combined action of flavonoids, phenolic acids and vitamins, and it is likely to contribute to healing across all phases.

6.4.5. Antimicrobial Effects

Infection is one of the commonest causes of delayed healing. Methanolic leaf extract improved healing in MRSA-infected diabetic wounds, but the effect was reduced when the wounds were infected with *Pseudomonas aeruginosa*[49], indicating that the antibacterial spectrum is not uniform. Antimicrobial activity has been exploited further by combining Moringa with other agents: electrospun PVA nanofibers containing Moringa water extract and zinc oxide nanoparticles were developed for antibacterial wound dressings[59], and silver nanoparticles synthesised using Moringa leaf extract and loaded onto bacterial cellulose produced marked distortion of bacterial cell morphology.[60] Antibiofilm activity has been suggested in the literature on this plant, but wound-specific evidence for it remains limited.

Table 6.3: Summary of Reported Effects of *M. Oleifera* Across the Phases of Wound Healing.

Phase	Key events	Reported effects of *M. oleifera*	Strength of evidence
Hemostasis	Clot formation; release of platelet-derived growth factors	Few dedicated studies; effects are largely uninvestigated.	Very limited
Inflammation	Neutrophil and macrophage infiltration; cytokine and ROS release; microbial clearance	Lower TNF-α, IL-1β and IL-6 in diabetic wounds [48]; antimicrobial activity against MRSA; antioxidant enzyme activity increased [49].	Moderate (animal)
Proliferation	Fibroblast proliferation and migration; angiogenesis; re-epithelialisation; granulation tissue	Fibroblast repopulation and collagen deposition [55]; increased capillary density and shorter epithelisation period [49]; VEGF-related endpoints studied [57].	Moderate (animal, limited *in vitro*)
Remodeling	Collagen maturation; wound contraction; tensile strength	Higher hydroxyproline content [58]; increased collagen and myofibroblasts [54]; increased tensile strength [47].	Moderate (animal)
Throughout	Oxidative stress; infection; hyperglycemia	Antioxidant, antibacterial and (indirectly) antihyperglycemic effects [47,51].	Moderate (animal); limited human data

6.5. Pathological Wounds: Diabetic and Infected Wounds

Diabetic wounds are impaired by hyperglycemia, neuropathy, vascular insufficiency and chronic inflammation. *M. oleifera* has a potential dual benefit here: it influences local repair and may also lower blood glucose. In the study by Azevedo et al., extract-treated diabetic rats had significantly reduced glycemia together with faster wound contraction.[48] Published human studies using powdered whole-leaf preparations have demonstrated antihyperglycemic and anti-dyslipidemic activity.[51] A bioactive aqueous fraction has been evaluated in a diabetic animal model[61], and combining topical conditioned medium of mesenchymal stem cells with oral *M. oleifera* extract produced glucose-lowering and wound-healing profiles comparable to the combination with metformin in diabetic rats.[50] These results support further investigation of *M. oleifera* in diabetic wound care, but should not be taken as proof of clinical benefit.

7. EXPERIMENTAL EVIDENCE:

7.1. In Vitro Studies

In vitro work relevant to wound healing has mainly used fibroblast and keratinocyte cultures and microbial assays. Normal human dermal fibroblasts have been

used to assess the ethyl acetate fraction of the plant[56], HaCaT keratinocytes were used for gene-expression analysis in a study of infected diabetic wounds[49], and human foreskin fibroblasts were included in the investigation of a chitosan–Moringa gel.[62] Typical readouts include cell viability and proliferation, scratch (migration) assays, collagen and growth-factor expression, and antimicrobial tests; scanning electron microscopy has shown morphological damage to bacteria exposed to Moringa-derived silver nanoparticles.[60] These assays are valuable for screening and narrowing down active fractions, but they cannot reproduce the multicellular, vascularised and microbially challenged environment of a real wound.

7.2. In Vivo Animal Studies

Most of the evidence comes from rodent models, including excision, incision, abrasion, infected, burn, oral mucosal and diabetic wounds. Key examples are summarised in Table 7.1 and discussed below.

- **Diabetic excision wounds.** In Wistar rats with diabetes, oral aqueous leaf extract (100 mg/kg for three weeks) together with topical 10% extract reduced blood glucose, increased the percentage of wound contraction on days 2, 5 and 10, lowered TNF-α, IL-1β and IL-6 expression and raised the histopathological score (198 ± 13.7 versus 145 ± 10.5 in saline-treated diabetic rats).[48] In a type 2 diabetic model (streptozotocin plus high-fat diet), a standardised extract-loaded hydrocolloid film at 0.5% and 1% raised day-7 wound closure to 77.67 ± 7.28% and 66.56 ± 12.67%, respectively, compared with 16 ± 3.82% in diabetic untreated controls.[58]
- **Infected wounds.** Methanolic leaf extract ointment improved contraction and reduced epithelisation time in MRSA-infected diabetic wounds, with histology showing increased epidermal height, angiogenesis and collagen deposition; the effect was weaker for *P. aeruginosa* infection.[49]
- **Oral and palatal wounds.** In 60 Sprague–Dawley rats followed for 14 days, 2% and 4% leaf extract gels were compared with povidone-iodine and vehicle gel on palatal donor-site wounds, with the 4% gel showing the greatest fibroblast activity and collagen deposition.[55] A separate rat study of gingival wounds found that 4% and 15% leaf extract gels were more effective than controls, with the 15% gel performing better.[63]
- **Chronic wounds and seed oil.** Moringa seed oil and oleic acid shortened the healing time of chronic wounds in immunosuppressed and diabetic mice and improved collagen content and myofibroblast numbers, although neither treatment changed skin cellularity during the inflammatory phase.[54]
- **Burn wounds and composite materials.** Nanoclay films loaded with amniotic fluid and Moringa were tested for antibacterial activity and in a burn wound model.[64] A carbopol-940 hydrogel containing Moringa extract accelerated healing in an excision wound model to a degree comparable with a marketed formulation.[53]

Dose–response relationships are not always linear. The 0.5% hydrocolloid film gave numerically higher day-7 closure than the 1% film[58], whereas in gingival wounds the 15% gel outperformed the 4% gel.[63] The optimal concentration therefore depends on the extract, vehicle and wound type and has to be established experimentally rather than assumed.

Table 7.1: Selected in Vivo Studies of *M. Oleifera* in Wound Healing.

Study	Model	Preparation and dose	Main findings
Azevedo et al., 2018 [48]	Diabetic Wistar rats; 1.5 cm² excision wounds (6 rats/ group)	Aqueous leaf extract, 100 mg/kg orally for 3 weeks; 10% topical (200 µL)	Reduced glycemia; greater wound contraction on days 2, 5 and 10; lower TNF-α, IL-1β and IL-6; higher histopathological score.

Study	Model	Preparation and dose	Main findings
Al-Ghanayem et al., 2022 [49]	MRSA- or *P. aeruginosa*-infected excision wounds in diabetic rats; HaCaT cells	Methanolic leaf extract ointment at two concentrations	Improved contraction, shorter epithelisation period, higher antioxidant enzyme activity, capillary density and collagen in MRSA infection; reduced effect with *P. aeruginosa*.
UKM PhD thesis [58]	STZ/high-fat diet diabetic rats; excision and abrasion wounds	Standardised aqueous leaf extract hydrocolloid film (0.1, 0.5, 1%)	Day-7 excision closure 77.67% (0.5%) and 66.56% (1%) versus 16% in controls; day-21 hydroxyproline 63.17 versus 46.85 µg/µL.
Amaliya et al., 2019 [55]	Palatal donor-site wounds in 60 Sprague–Dawley rats; 14 days	2% and 4% leaf extract gel	Early fibroblast repopulation; highest fibroblast synthesis and collagen deposition with 4% gel.
Gingival wound gel study [63]	Gingival wounds in rats	4% and 15% leaf extract gel	Better healing than control; 15% gel more effective than 4% gel.
Ventura et al., 2021 [54]	Physiological, immunosuppressed and diabetic wounds in mice	Seed oil and oleic acid, topical	Shortened healing time in chronic wounds; increased collagen in diabetic wounds; restored myofibroblast numbers.
VEGF study [57]	Wistar rats (30 animals), 8 days	Topical gels of 5, 10 and 15% leaf extract in CMC	Wound closure rate and VEGF gene expression evaluated as angiogenic endpoints.
Hydrogel study [53]	Excision wound model in animals	Carbopol-940 hydrogel with *M. oleifera* extract	Accelerated healing comparable to a marketed formulation; moist environment and antimicrobial action proposed.
Nanoclay film study [64]	Burn wound model	Amniotic fluid and Moringa-loaded nanoclay films	Evaluated for antibacterial activity and wound healing with molecular pathway analysis.

8. CLINICAL EVIDENCE:

Human data on the wound-healing effect of *M. oleifera* are very limited. A narrative review of 22 clinical trials and nine case reports published between 2015 and 2025 organised the human literature mainly into immunological and nutritional effects, metabolic and endocrine disorders, inflammatory and oxidative stress-related conditions, and maternal and child health, and called for standardised large-scale randomised controlled trials.[65] Earlier, Stohs and Hartman identified five human studies using powdered leaf preparations, which showed antihyperglycemic and anti-dyslipidemic effects without reported adverse events.[51] These studies are relevant to wound care mainly through their metabolic effects rather than as direct wound-healing trials.

One of the few controlled human studies of a Moringa-based topical product is a randomised, split-body study of a chitosan gel infused with hydroalcoholic extract of Moringa leaf powder after cosmetic breast surgery, in which one side received the gel and the other served as control; the gel was reported to improve wound healing and scar appearance, including elasticity. Such findings are encouraging, but a single study in an aesthetic surgery setting cannot be generalised to chronic, diabetic or infected wounds. Well-designed randomised trials in clinically relevant wound types, such as diabetic foot ulcers, venous ulcers, burns and surgical wounds, are needed.[62]

9. FORMULATIONS AND DELIVERY SYSTEMS:

The delivery system strongly influences how effectively an extract reaches the wound bed. A good dressing should retain the active compounds, release them in a controlled manner, maintain a moist wound environment, protect against microbial contamination and be non-toxic and easy to remove. Crude extracts are poorly retained on open wounds and may be rapidly lost, so a range of vehicles and carriers has been

explored (Table 9.1). A moist environment and the antimicrobial properties of the extract were suggested as the reasons for the performance of a carbopol hydrogel[53], and standardised extracts have been incorporated into hydrocolloid film dressings for diabetic wounds.[58]

Nanotechnology adds further possibilities. Silver nanoparticles incorporated into wound dressings have shown antibacterial activity against both Gram-positive and Gram-negative bacteria and have inhibited biofilm formation.[66] Green synthesis uses phytochemicals in plant extracts to reduce metal salts to nanoparticles, and *M. oleifera* leaf extract has been used in this way to prepare silver nanoparticles incorporated into bacterial cellulose as an antimicrobial wound-healing membrane.[60] Electrospun PVA nanofibers containing Moringa extract and zinc oxide have also been developed, in which the Moringa is intended to provide healing activity and the zinc oxide the antibacterial effect.[59] Because metallic nanoparticles carry their own cytotoxicity, dose and regulatory questions, such composites require careful safety evaluation before clinical use.

Table 9.1: Formulation Strategies for *M. Oleifera* in Wound Care.

Formulation	Examples in the literature	Potential advantages	Considerations
Extracts (oral or topical)	Aqueous leaf extract given orally with topical application [48]	Simple to prepare; allows systemic and local effects (for example glucose lowering)	Poor retention on open wounds; composition varies between batches
Ointments and creams	Methanolic leaf extract ointment [49]	Familiar dosage form; occlusive vehicle	May be less suitable for heavily exuding wounds
Gels and hydrogels	CMC gels [57]; HPMC gels [55]; carbopol-940 hydrogel [53]; chitosan gel [62]	Moist healing environment; easy application; good for irregular and oral wounds	Limited mechanical protection; stability and preservative needs
Film and hydrocolloid dressings	Standardised leaf extract hydrocolloid film [58]	Controlled release; protects wound; suited to standardised extracts	Needs mechanical, swelling and release testing
Nanofibers	PVA/Moringa/ZnO electrospun nanofibers [59]	High surface area; mimics extracellular matrix; combined antibacterial action	Scale-up and reproducibility; mainly early-stage research
Green-synthesised nanoparticles	Moringa-mediated silver nanoparticles on bacterial cellulose [60]	Antibacterial and antibiofilm potential [66]	Nanoparticle toxicity and regulatory questions
Composite and nanoclay films	Amniotic fluid and Moringa-loaded nanoclay films [64]	Multifunctional; tested in a burn wound model	Complex composition; difficult to attribute effects
Seed oil	Moringa seed oil [54]	Emollient; effective in chronic wound models	Different mechanism and chemistry from leaf extracts
Combination therapy	Topical stem-cell conditioned medium with oral Moringa extract [50]	Targets both local repair and glycemic control	Complex regimen; early-stage evidence

10. SAFETY AND TOXICITY:

Safety studies in animals using aqueous leaf extracts indicate a high degree of safety. Oral doses of up to 2000 mg/kg were reported to be safe, and the lethal dose in mice was greater than 6.4 g/kg.[51] An aqueous-methanolic leaf extract was likewise associated with an LD50 above 2000 mg/kg in female Wistar rats.[67] No adverse effects were reported in the human studies reviewed by Stohs and Hartman. Nevertheless, rare adverse reactions occur: a case of cutaneous toxicity attributed to Moringa powder resolved after the powder was discontinued and symptomatic treatment was given. The same report notes that plant products sold as supplements do not need to demonstrate quality, efficacy or safety before commercialisation. Most safety data derive from oral exposure, so dermal tolerance on broken skin, repeated application and systemic absorption through large or chronic wounds still require specific study.[68]

SUMMARY:

The drumstick or horseradish tree, *Moringa oleifera* Lam. (Moringaceae), is a fast-growing tropical tree long used in traditional African and South Asian medicine. This review examines its taxonomy, botany, phytochemistry and pharmacological potential, with particular emphasis on wound healing. Its leaves, seeds, pods, flowers, bark and roots contain flavonoids (quercetin, kaempferol, rutin), phenolic acids, glucosinolates and isothiocyanates, carbamates, carotenoids, sterols, vitamins and minerals, whose content is affected by plant component, cultivar, provenance, processing and extraction solvent. These compounds underlie reported antioxidant, anti-inflammatory, antimicrobial, antidiabetic, hepatoprotective, neuroprotective, antiulcer and anticancer activities.

Wound healing combines haemostasis, inflammation, proliferation and remodelling, and can be hampered by oxidative stress, infection, chronic inflammation and diabetes. Preclinical research, primarily in rodent models, shows that leaf extracts and seed oil improve wound contraction, fibroblast proliferation, angiogenesis, collagen deposition, hydroxyproline content and tensile strength. They also enhance natural antioxidant enzymes, decrease TNF-α, IL-1β and IL-6, and inhibit wound-associated organisms such as MRSA and *Staphylococcus aureus*, although effectiveness against *Pseudomonas aeruginosa* is less reliable. Benefits have been documented in diabetic and infected wounds, where glucose-lowering actions may enhance local tissue healing. Gels, hydrogels, hydrocolloid films, electrospun nanofibers and green-synthesised nanoparticles have been studied as delivery systems. Animal studies indicate a large safety margin after oral exposure, but human data remain extremely limited.

CONCLUSION:

Moringa oleifera is a promising multi-target herb for the treatment of wounds. Because poor healing is multifactorial, its antioxidant, anti-inflammatory, antibacterial and pro-regenerative qualities act on multiple stages of tissue repair simultaneously. Its apparent dual effect in diabetic wounds, combining glycaemic management with local repair, is particularly intriguing. Leaf extracts are the most researched preparation, followed by seed oil, and new developments in topical formulations and nanotechnology-based dressings offer additional therapeutic possibilities.

However, most of the information now available is preclinical, and a number of limitations prevent practical application. There are few clinical trials in wound care; the chemical makeup of extracts varies greatly and standardisation is uneven; dose–response relationships are not always linear, and ideal doses, vehicles and treatment durations are unknown. Because most safety data come from oral exposure, dermal tolerance on damaged skin, repeated application and systemic absorption through large or chronic wounds require particular assessment, and nanoparticle-based composites raise additional toxicological and regulatory concerns.

The priorities for future research should therefore be standardised and chemically characterised preparations, mechanistic investigations to validate the signalling pathways involved, and carefully planned randomised controlled trials in clinically relevant wounds such as burns, venous ulcers, diabetic foot ulcers and surgical wounds. Until such data become available, *M. oleifera* should be considered a prospective candidate for wound-healing treatments rather than a proven treatment.

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HOW TO CITE: Atharv Mankar, Varsha Kharat, *Moringa Oleifera*: Phytochemistry, Pharmacological Potential and Its Role in Wound Healing – A Comprehensive Review, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 11, 4612-4630. https://doi.org/10.5281/zenodo.24527994