Review Article

Phytochemical Investigation and Pharmacological Evaluation of *Plumeria rubra* and *Murraya koenigii* l. Leaves Extracts for Wound Healing Activity: A Review

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ABSTRACT

The study investigates the phytochemical composition and pharmacological efficacy of extracts from *Plumeria rubra* and *Murraya koenigii* L. leaves, known for their bioactive compounds and potential therapeutic applications. Unlike synthetic medications, which can be costly and ineffective, these natural extracts may hold promise for wound healing and antimicrobial activities. Various extracts, including acetone, methanol, and petroleum ether, were prepared and analyzed through preliminary phytochemical screening and TLC fingerprinting. Key compounds identified included flavonoids, tannins, saponins, alkaloids, and phenolic compounds, which have known antioxidant and anti-inflammatory properties. Notably, the methanol extract exhibited higher phenolic and flavonoid content, contributing to its superior antioxidant activity, as measured by the DPPH assay, compared to other extracts and standard ascorbic acid. Antimicrobial testing, conducted using the agar and cup plate method, demonstrated that both acetone and methanolic extracts significantly inhibited bacterial growth at 50 mg/ml, with their efficacy compared to that of Amoxicillin. In the wound healing activity evaluation using an excision model in rats, extracts showed marked improvement, particularly the acetone extract, which outperformed the positive control (Betadine). The findings underscore the therapeutic potential of these plant extracts in pharmaceutical research.

Keywords: *Plumeria rubra* and *Murraya koenigii* L. Phytochemical screening, Wound healing activity, antimicrobial.

INTRODUCTION

Wounds are defined as physical injuries that create an opening or break in the skin, disrupting normal anatomical structure and function. The loss of continuity can occur in the epithelium and potentially in the underlying connective tissue.[1] In the studied population, the prevalence of wounds is approximately 15.03 per 1000 individuals, with chronic wounds reported at 4.5 per 1000 and acute wounds at 10.5 per 1000.[2] Wound healing commences immediately and can vary in duration based on the severity of the wound. The healing process consists of three main stages: the inflammatory phase, the proliferative phase, and the remodeling phase, which ultimately affects the strength and appearance of the healed tissue.[3] Wound healing agents can promote fibroblast and keratinocyte proliferation, enhance collagen formation, and possess antimicrobial, antioxidant, and anti-inflammatory properties. For a substance derived from medicinal plants or natural products to be considered an effective wound healing agent, it typically needs to exhibit at least two of these beneficial properties.[4,5]

Various plants in multiple dosage forms are utilized by tribal communities and folk traditions for treating wounds, cuts, and burns. Numerous ethnomedicinal surveys, research studies, and publications emphasize the role of plant-based medications in wound healing management.[6] However, there remains a lack of comprehensive information detailing the specific plant parts used, other than leaves,[7] for their wound healing properties.

Classification of wound healing:

Wounds can be classified based on various factors such as etiology, location, type of injury, or symptoms.

1. Acute wound

2. Chronic wound

1. **Acute wounds:**

These are injuries that heal spontaneously through a well-organized healing process, resulting in functional and anatomical restoration. They can occur due to surgery or trauma and typically heal within 5 to 30 days. Acute wounds may involve full-thickness skin damage or be superficial injuries affecting only the epidermis and dermis. Examples include abrasions, lacerations, heat wounds, and surgical incisions.[8,9,10]

2. **Chronic wounds**:

These are defined as those failing to show healing signs within four weeks and often stagnating in specific healing phases. Various factors, such as exudation, necrosis, tissue hypoxia, infection, and elevated inflammatory cytokines, disrupt the healing process. Conditions like neuropathy, hypertension, burns, venous and arterial insufficiency, and vasculitis contribute to the development of chronic wounds. These wounds, including leg ulcers, pressure ulcers, severe burns, and diabetic ulcers, are challenging to manage due to their atypical healing patterns and persistence.[11,12]

Mechanism of wound healing:[13]

1. Coagulation and hemostasis

2. Inflammatory phase

3. Proliferative phase

4. Remodelling phase

1. **Coagulation and hemostasis**:

Immediately after injury, coagulation and hemostasis take place in the wound. The mechanisms aimed at preventing exsanguination protect the vascular system and ensure the integrity of vital organ functions despite injuries. A secondary objective is to create a matrix for the cells necessary in later healing stages. Hemostasis relies on a dynamic equilibrium among endothelial cells, thrombocytes, coagulation, and fibrinolysis, influencing the deposition of fibrin at wound sites and affecting reparative processes. The coagulation cascade is initiated through extrinsic and intrinsic pathways, resulting in platelet aggregation and clot formation to minimize blood loss. When blood contacts exposed collagen and extracellular matrix components at injury sites, it activates clotting factors, forming a blood clot composed of fibronectin, fibrin, vitronectin, and thrombospondin. This blood clot not only facilitates hemostasis but also serves as a provisional matrix for cell migration during the hemostatic and inflammatory phases. Platelets, containing α-granules filled with growth factors and cytokines like platelet-derived growth factor (PDGF) and transforming growth factor-β (TGF-β), play a crucial role in wound healing by attracting neutrophils, macrophages, endothelial cells, and fibroblasts. Additionally, platelets release vasoactive amines such as serotonin, promoting vasodilation and increased vascular permeability, leading to tissue fluid extravasation, edema, and self-potentiation during the inflammatory phase. Eicosanoids and other metabolites from arachidonic acid also contribute significantly to the immediate inflammatory response following cell membrane injury.

2. **Inflammatory phase:**

The inflammatory phase of the healing response begins immediately after an injury, whether surgical or traumatic, which disrupts tissue architecture and causes bleeding. Blood fills the wound defect, and its exposure to collagen triggers platelet degranulation and activation of Hageman factor. This activation initiates several biological amplification systems, including the complement, kinin, and clotting cascades, as well as plasmin generation. These processes enhance the original injury signal, resulting in the formation of a clot that brings the wound edges together and facilitates the accumulation of mitogens and chemo attractants at the wound site. Production of kinins and prostaglandins contributes to vasodilation and increased permeability in wound areas, leading to edema, pain, and swelling soon after injury. Within 6 hours, circulating immune cells, particularly polymorph nuclear leucocytes (PMN), are the first to arrive at the wound site, peaking at 24-48 hours. Their primary role is phagocytosis of bacteria introduced during injury, though their presence is not essential for normal healing in the absence of infection. PMNs have a short lifespan and diminish post-day three. Following PMNs, macrophages, derived from monocytes through migration and chemotaxis, enter the wound between 48-96 hours post-injury, peaking around day three. Macrophages, with a longer lifespan, persist until healing is complete and play a critical role in the healing process, facilitating tissue decontamination and releasing growth factors necessary for granulation tissue formation. T lymphocytes appear around day five, with peak numbers by day seven, and are essential for healing along with macrophages. Macrophages and lymphocytes act as both phagocytes and producers of cytokines, which recruit additional inflammatory cells and assist in tissue repair.

3. **Proliferative phase:**

The proliferative phase of wound healing begins three days post-injury and lasts approximately two weeks. During this phase, fibroblasts migrate and deposit newly synthesized extracellular matrix, replacing the provisional network of fibrin and fibronectin. Macroscopically, this phase is marked by the abundant formation of granulation tissue.

Fibroblast migration

Fibroblasts and myofibroblasts play a critical role in wound healing, beginning their proliferation in the surrounding tissue within the first three days post-injury. They are attracted into the wound site by factors such as Transforming Growth Factor beta (TGF-β) and Platelet-Derived Growth Factor (PDGF), which are released by inflammatory cells and platelets. Fibroblasts appear at the wound site by the third day and undergo phenotypic modulation before proliferating and producing matrix proteins including hyaluronan, fibronectin, proteoglycans, and type 1 and type 3 procollagen. By the end of the first week, an abundant extracellular matrix supports further cell migration necessary for repair. Fibroblasts transition to a myofibroblast phenotype, characterized by thick actin bundles and extensions known as pseudopodia that anchor to fibronectin and collagen in the extracellular matrix. This leads to wound contraction, a vital process that brings the wound edges closer together. Following this, redundant fibroblasts undergo apoptosis, concluding their role in the healing process.

Collagen synthesis

Collagens, primarily synthesized by fibroblasts, play a crucial role in all phases of wound healing. They serve as a foundational element for forming the intracellular matrix within the wound environment. In unwounded dermis, collagen composition is predominantly type 1 (80%) and type 3 (25%). In contrast, wound granulation tissue shows a significant increase in type 3 collagen, constituting 40% of its makeup.

Angiogenesis and granulation tissue formation

The modeling and establishment of new blood vessels, essential for wound healing, occur during all phases of the

reparative process. Key to this process are angiogenic factors, such as FGF, VEGF, PDGF, angiogenin, TGF-α, and TGF-β, which promote angiogenesis and attract immune cells like neutrophils and macrophages. Endothelial cells respond to various stimulatory and inhibitory factors that regulate their proliferation and migration. A four-step process in this context includes:

(i) production of proteases by endothelial cells to degrade the basal lamina for migration through the extracellular matrix

(ii) chemotaxis

(iii) proliferation

(iv) remodeling and differentiation of new vessels.

Initially, there is an absence of vascular supply in the wound center, necessitating perfusion from uninjured vessels and diffusion from surrounding tissues. New capillaries form through capillary sprouts invading the wound area, creating a microvascular network within days. Chemotaxis allows cells to move along a chemical gradient, facilitating angiogenesis and influenced by various mediators, including endothelial cell growth factors and TGF-α. Migration necessary for angiogenesis is driven by chemotactic activity and involves coordinated cellular processes, such as signal transduction and cytoskeletal organization**,** regulated through mechanisms like chemotaxis, mechanotaxis, and haptotaxis.

Epithelialization

Migration of epithelial cells initiates at the wound edges within hours of injury, leading to the formation of a single cell layer over the defect. This process is marked by increased mitotic activity of epithelial cells near the wound edges. As these cells migrate, they attach to the provisional matrix beneath. The migration ceases once the advancing epithelial cells converge, prompting the formation of the basement membrane**.**

4. Remodelling phase:

As the final phase of wound healing, the modeling phase is crucial for developing new epithelium and forming scar tissue. This phase coincides with the synthesis of the extracellular matrix during the proliferative and remodeling phases, lasting from 1 to 2 years or longer. It is regulated to maintain a balance between degradation and synthesis for optimal healing. During this phase, collagen bundles increase in diameter while hyaluronic acid and fibronectin are degraded, allowing collagen fibers to regain approximately 80% of their original strength. However, the tissue's original strength is never fully restored. Continuous synthesis and breakdown of collagen and extracellular matrix remodeling reach a steady state around three weeks post-injury

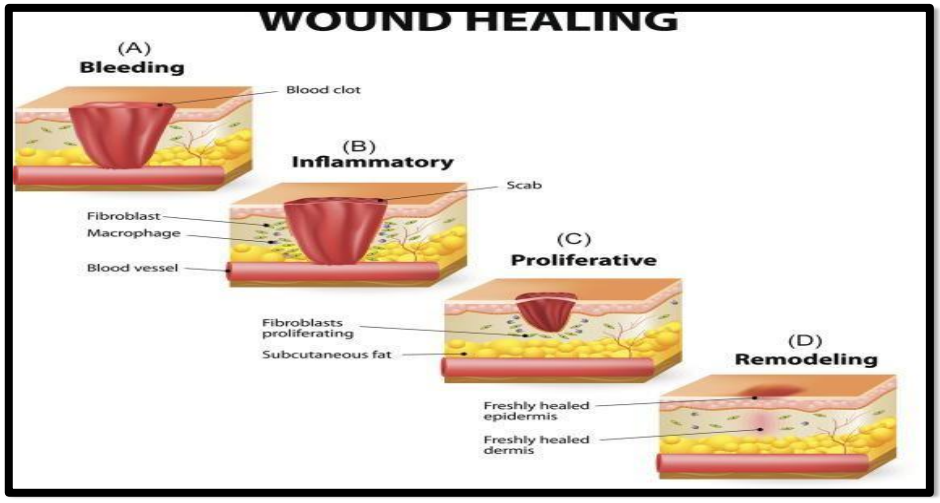

Figure 1: Stages of wound healing

Healing:

Tissue injury may result in cell death and tissue destruction. Healing is the body response to injury in an attempt to restore normal structure and function. Healing involves 2 distinct processes:

- Regeneration when healing takes place by proliferation of parenchymal cells and usually results in complete restoration of the original tissues.
- Repair when healing takes place by proliferation of connective tissue elements resulting in fibrosis and scarring.

PLANT PROFILE:

***Plumeria rubra L*:**[14,15,16]

Plumeria rubra L., or frangipani, is a tropical flowering plant recognized for its fragrant flowers and ornamental value. It grows as a deciduous shrub or small tree, reaching heights of 2-8 meters. The plant features fleshy branches with grey bark and exudes irritating white latex. Its leathery leaves are dark green and clustered at branch tips, typically dropping in cooler weather. The highly fragrant flowers, mostly pink to red with yellow centres, bloom year-round and can produce elongated seed pods. It thrives in full sun and well-

drained soil, suitable for warm climates and showing tolerance to drought and salt, making it ideal for gardens and coastal settings.

Taxonomic classification:

Table 1: Taxonomy of *Plumeria rubra L.*

Scientific name	***Plumeria rubra L***
Kingdom	Plantae
Class	Magnoliopsida
Order	Gentianales
Family	Apocynaceae
Genus	Plumeria
Species	*Plumeria rubra* L
Common Names	Frangipani, Red plumeria, Temple tree.

Figure 2: *Plumeria rubra* L**.**

Chemical constituents:

Table 2: Chemical constituents present in different parts of *Plumeria rubra L.*

Sr. no	**Parts**	**Chemical constituents**
01	**Leaves**	❑ **Volatile compounds / essential oils** • (Z)-β-farnesene • α-patchoulene • limonene • (E)-β-farnesene • α-copaene • phytol (identified by GC-MS) ❑ **Secondary metabolites** • Flavonoids (e.g., *quercetin, quercitrin, kaempferol* glycosides) • Terpenoids (e.g., *lupeol, uvaol, ursolic acid*) • Iridoids (e.g., *plumieride*, *15-demethylplumieride*) • Sterols (e.g., *β-sitosterol*, *stigmasterol*) • Alkaloids (e.g., *plumerianine*)
02	**Stem Bark**	❑ Iridoids and glycosides: • *plumieride*, *allamacin*, *allamandin* • 13-O-trans-p-coumaroylplumieride ❑ Triterpenoids/steroids: • *rubrajaleelol, rubrinol* • *taraxasteryl acetate* • *oleanolic acid, β-amyrin acetate* ❑ Lignans (e.g., *liriodendrin*) ❑ Flavonoids ❑ Phenolics ❑ Other glycosides (e.g., flavan-3-ol glycosides)
03	**Flowers**	❑ **Essential oil / volatile constituents** • Benzyl salicylate • Benzyl benzoate • Geraniol • (E,E)-geranyl linalool • Linalool • (E)-nerolidol

		• Aliphatic alkanes (e.g., nonadecane, tricosane, pentacosane) These vary by region and extraction method. ❑ **Other identified classes** • Flavonoids: ○ narcissin, quercetin-3-O-α-L-arabinopyranoside ○ cyanidin glycosides ○ rubranonoside, plumerubroside • Phenolic acids (e.g., p-E-coumaric acid, 3-O-caffeoylquinic acid) • Fatty acids and derivatives • Alkaloids • Steroids (e.g., β-sitosterol) • Coumarins (e.g., scopoletin)
04	**Root**	❑ Iridoids: • plumieride, fulvoplumericin ❑ Dihydro derivatives: • β-dihydroplumericin, β-dihydroplumericinic acid ❑ Triterpenoids: • lupeol, oleanolic acid, stigmasterol ❑ Antibacterial terpenoids (e.g., rubrinol). Other glycosides and terpenoids

Pharmacological actions of *Plumeria rubra* include:[17,18,19]

1) leaves with anti-inflammatory, analgesic, and antimicrobial effects, used in treating fever, wounds, skin infections, and rheumatic pain;

2) flowers exhibiting antioxidant, antimicrobial, and mild sedative properties, utilized for stress, fever, cough, and cosmetic applications;

3) stem bark providing antidiarrheal, antimicrobial, and anti-inflammatory benefits, effective against diarrhea, dysentery, toothache, and skin diseases;

4) roots demonstrating antipyretic, purgative, and antimicrobial activities, used in fever, constipation, and some skin conditions;

5) latex presenting antimicrobial and caustic actions, applied externally for warts, corns, and toothache.

***Murraya koenigii L.*:**[20,21]

Murraya koenigii L., or curry leaf tree, is an evergreen tree native to tropical regions of India and Sri Lanka, valued for its aromatic culinary and medicinal leaves. It belongs to the Rutaceae family, with scientific synonyms such as Bergera koenigii. The plant is categorized under Kingdom Plantae, Order Sapindales, Genus Murraya, and Species koenigii. It typically reaches heights of 4 to 6 meters, featuring a slender trunk of up to 16 cm in diameter and greyish bark. The leaves are pinnately compound, measuring about 30 cm long with 11-24 glossy, lance-shaped leaflets that are 1-5 cm long, releasing a strong citrus-anise aroma when crushed. The tree produces small, white, fragrant flowers in terminal cymes containing 60-90 blooms and fruits that are oblong and green, maturing to black, measuring 1-1.6 cm in length. *Murraya koenigii* thrives in moist, tropical to subtropical environments at elevations up to 1,600 meters, ideally in well-drained, slightly acidic soils with a pH of 5.5-6.5. Once established, it exhibits drought tolerance and grows at a medium pace in USDA zones 10 and higher, although it is sensitive to frost.

Figure 3: Curry leaves

Taxonomic classification:[22]

Table 3: Taxonomic classification of *Murraya koenigii* L.

Kingdom	**Plantae**
Scientific name	*Murraya koenigii* L.
Subkingdom	Tracheobionta
Division	Magnoliophyta
Class	Magnoliopsida
Subclass	Rosidae
Order	Sapindales
Family	Rutaceae
Genus	Murraya
Species	*Murraya koenigii* L.
Common name	Curry leaf plant

Chemical constituents:

Table 4: Chemical constituents present in different parts of *Murraya koenigii* L

Sr. no:	Parts:	Chemical constituents
01	Leaves	❑ **Carbazole alkaloids:** Mahanimbine, Murrayanine, Mahanine, Koenimbine, Koenigine ❑ **Essential oils**: α-pinene, β-pinene, sabinene, limonene, caryophyllene ❑ **Flavonoids.** ❑ **Phenolic compounds.** ❑ **Vitamins** (A, B, C) ❑ **Minerals** (iron, calcium, phosphorus)
02	Fruits	❑ **Carbazole alkaloids** ❑ **Flavonoids** ❑ **Sugars** ❑ **Organic acids**
03	Bark	❑ **Alkaloids:** Murrayanine, Mahanimbine ❑ **Tannins** ❑ **Glycosides**
04	Roots	❑ **Carbazole alkaloids:** Murrayanol, Isomahanimbine ❑ **Coumarins**
05	Seeds	❑ **Fixed oils** ❑ **Alkaloids (minor amounts)**

Pharmacological action:

1. **Leaves**: Possess antioxidant, antidiabetic, antimicrobial, anti-inflammatory, hepatoprotective, neuroprotective, and hypolipidemic activities.

2. **Bark**: Exhibits antimicrobial, analgesic, anti-inflammatory, and astringent effects.

3. **Roots**: Show anti-inflammatory, analgesic, and antimicrobial properties.

4. **Fruits**: Have antioxidant, antidiarrheal, and antimicrobial activities.

5. **Seeds**: Display antimicrobial and insecticidal effects.

USES IN TRADITIONAL MEDICINE:

***Plumeria rubra L*:[23]**

Fruit is consumed in the West Indies, whereas in India it is known to be an abortifacient. The root bark is classified as drastic and purgative, and is noted for its effects on blenorrhagia. The latex from the plant is utilized to alleviate toothaches and treat carious teeth. Additionally, the flowers are aromatic and possess bechic properties, leading to their use in a widely popular pectoral syrup.

***Murraya koenigii* L.[24]**

Commercially, raw *Murraya koenigii* leaves are being sold in vegetable markets, alongside various seasoned products like sambar powder, curry powder, and chutneys. The use of mosquito repellents is noted across all zones, with *Murraya koenigii* traditionally employed for insect repulsion. A study by Jamil et al. (2016) reported 100% effectiveness in repellence, a finding corroborated by current research indicating that all surveyed individuals use fumigation methods with these leaves. Historically, Namrata et al. (1997) explored the fumigation method in laboratory conditions. The primary usage of curry leaves is culinary, but they also serve medicinal, traditional, and commercial roles, earning them the title of "Magical Plant of Indian Spice," as described by Nishan and Subramanian (2015).

SAFETY AND TOXICITY:[25]

Acute toxicity assessment involves estimating the LD50, with studies indicating that *Plumeria rubra* and *Murraya koenigii* L. leaf extracts are safe at a dose of 2000 mg/kg, following OECD Guidelines for Acute Toxicity Study: Class Method (423). Research showed that administration of these extracts at doses up to 2000 mg/kg did not result in significant behavioral changes in animals, including alertness, motor activity, breathing, restlessness, diarrhoea, convulsions, coma, or appearance alterations. Hence, no lethality was observed in rats at this dosage, confirming the safety of *Plumeria rubra* and *Murraya koenigii* L. for biological studies, with a calculated maximum therapeutic experimental dose of 200 mg/kg.

CONCLUSION AND FUTURE PERSPECTIVES

Plumeria rubra and *Murraya koenigii* L. leaves were evaluated for pharmacognostical, phytochemical, and pharmacological properties. The ash, extractive values, and loss on drying complied with Ayurvedic Pharmacopoeia standards. Preliminary phytochemical analysis revealed that acetone extracts contained carbohydrates, glycosides, and

flavonoids, while methanolic extracts contained glycosides, carbohydrates, steroids, tannins, and flavonoids. In vitro studies demonstrated significant antibacterial activity at 50 mg/ml against Escherichia coli (10 mm zone of inhibition) and Staphylococcus aureus (8 mm). In vivo studies indicated significant wound healing activity with 1% and 2% ointment concentrations, with the 2% methanol extract proving most effective compared to standard Betadine in a rat model. Overall, the study supports the traditional medicinal use of these leaf extracts for wound healing.

Wound healing research is advancing rapidly with new technologies and treatments aimed at enhancing outcomes for patients with acute and chronic wounds. Innovations include biological therapies, regenerative medicine, smart dressings, gene therapy, nanotechnology, and bioinformatics.

Advancements in Wound Healing Research:

Advancements in wound healing research include biological therapies such as stem cell therapy, which aids tissue regeneration and modulates inflammation. Growth factors like PDGF and FGF are being investigated for their roles in cell proliferation and tissue repair. Regenerative medicine employs tissue engineering techniques, including skin substitutes and 3D bioprinting, to create functional tissue constructs that enhance integration and recovery. Smart wound dressings equipped with monitoring capabilities provide real-time feedback on healing progress. Additionally, gene therapy is being explored to deliver therapeutic genes that enhance cellular responses like angiogenesis and extracellular matrix formation.[26]

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REFERENCES

1. Ramzi SC, Vinay K, Stanley R. Pathologic Basis of Diseases. 5th ed. Philadelphia, PA: WB Saunders Company; 1994. p. 86.
2. Gupta N, Gupta SK, Shukla VK, Singh SP. An Indian community-based epidemiological study of wounds. J Wound Care 2004;13:323-5.
3. Sumitra M, Manikandan P, Suguna L. Efficacy of Butea monosperma on dermal wound healing in rats. Int J Biochem Cell Biol 2005;37:566-73.
4. Houghton PJ, Hylands PJ, Mensah AY, Hensel A, Deters AM. In vitro tests and ethnopharmacological investigations: Wound healing as an example. J Ethnopharmacol 2005;100:100-7.
5. Agyare C, Asase A, Lechtenberg M, Niehues M, Deters A, Hensel A, et al. An ethnopharmacological survey and in vitro confirmation of ethnopharmacological use of medicinal plants used for wound healing in Bosomtwi Atwima-Kwanwoma area, Ghana. J Ethnopharmacol 2009;125:393-403.
6. Kadhirvel K, Ramya S, Sudha TP, Ravi AV, Ethnomedicinal survey on plants used by tribals in Chitteri hills environ. Int J Sci Tech 2010;5:35-45.
7. Sharma T, Bhide B, Acharya R. Ethnomedicinal claims on wound healing activity of certain leaf drugs a review. Int J Ayurvedic Med 2018;9:42-78.
8. Velnar T., Bailey T. and Smrkolj V. The Wound Healing Process: an Overview of the Cellular and Molecular Mechanisms. The Journal of International Medical Research 2009; 37: 1528 – 1542.
9. Robson MC, Steed DL, and Franz MG. Wound healing: biologic features and approaches to maximize healing trajectories. Curr Probl Surg 2001; 38: 72 – 140.
10. Li, J.; Chen, J.; Kirsner, R. Pathophysiology of Acute Wound Healing. Clin. Dermatol. 2007, 25, 9–18. [CrossRef] [PubMed]
11. Swezey L. The difference between acute and chronic wounds (2019).
12. K. Jung, S. Covington, C. K. Sen, M. Januszyk, R. S. Kirsner, G. C. Gurtner and N. H. Shah, Wound Repair Regen., 2016, 24, 181–188.
13. Sanjay S, Sivakumar G, Jaya Prakash S, Mohamed Shalim Khan M. Wound Healing: A Review of Mechanisms, Interventions, and Future Trends. IJSDR. 2024 Mar;9(3)
14. Barbosa Nogueira Freitas L, de Freitas CD, Ayala AP, de Moura TA, de Oliveira JS, Gonçalves JF, Ramos MV. Identification and Characterisation of Organic Calcium Crystals in the Plumeria pudica and *Plumeria rubra* Latexes. ACS omega. 2025 Jul 11;10(28):30879-85.
15. Rahman H, Reddy VB, Ghosh S, Mistry SK, Pant G, Sibi G. Antioxidant, cytotoxic and hypolipidemic activities of Plumeria alba L. and *Plumeria rubra* L. American Journal of Life Sciences. 2014;2(6):11-5.
16. Zaheer Z, Konale AG, Patel KA, Subur KW, Farooqui MN. *Plumeria rubra* Linn., An Indian Medicinal Plant. International Journal of Pharmacy & Therapeutics. 2010;1(2):116-9.
17. Verma S. Multipurpose ornamental plant *Plumeria rubra* Linn (Apocynaceae). Int J Sci Res Sci Eng Technol. 2016;2(4):646-9.
18. Suryanarayanan TS, Thennarasan S. Temporal variation in endophyte assemblages of *Plumeria rubra* leaves. Fungal Diversity. 2004 Feb 1;15:197-204.
19. Deepa Iyer DI, Devi PU. Phyto-pharmacology of *Murraya koenigii* (L.).
20. Handral HK, Pandith A, Shruthi SD. A review on *Murraya koenigii*: multipotential medicinal plant. Asian Journal of pharmaceutical and clinical research. 2012 Nov;5(4):5-14.
21. Kesari AN, Kesari S, Singh SK, Gupta RK, Watal G. Studies on the glycemic and lipidemic effect of *Murraya koenigii* in experimental animals. Journal of Ethnopharmacology. 2007 Jun 13;112(2):305-11.
22. DiPietro LA. Angiogenesis and wound repair: when enough is enough. Journal of Leucocyte Biology. 2016 Nov;100(5):979-84.
23. Kalantri M, Aher AN. Review on traditional medicinal plant: *Plumeria rubra*. J Med Plants Stud. 2016;4(6):204–7.
24. Nayana MD, Ramana P, Muruli KV, Rashmita HR. Traditional uses of wild curry [*Murraya koenigii* L.] growing in natural forests of Uttara Kannada. GreenariA [Internet]. 2024 Nov [cited 2026 Jun 13];2(11):174–9.
25. Gummalla P, Shaik S, Lakshmi K, Sailaja P. Anti-inflammatory and wound healing activity of hydroalcoholic extract of *Murraya koenigii* fruits in rats. Int J Herb Med. 2016;4(3):40–4.
26. Sanjay S, Sivakumar S, Jayaprakash S, Mohamed Shalim Khan M. Wound Healing: A Review of Mechanisms, Interventions, and Future Trends. IJSDR. 2024 Mar;9(3):982–91.