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Role of Azadirachta Indica in Gastroprotection and Cutaneous Wound Regeneration

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Abstract: *Azadirachta indica (Neem) is a widely recognized medicinal plant in Ayurvedic and modern pharmacology due to its broad-spectrum therapeutic potential. This review explores its gastroprotective and cutaneous wound regenerative properties based on experimental and clinical evidence. Neem contains bioactive compounds such as nimbidin, nimbin, azadirachtin, quercetin, and limonoids that exhibit anti-inflammatory, antioxidant, antimicrobial, and cytoprotective effects. These activities contribute to mucosal defense in gastrointestinal disorders and accelerated tissue repair in skin injuries. The present study compiles recent literature (2010–2025) highlighting mechanisms of action and experimental outcomes supporting neem's dual role in gastroprotection and wound healing..*

Keywords: Azadirachta indica, gastroprotection, wound healing, neem, tissue regeneration, phytotherapy

I. INTRODUCTION

Medicinal plants have been extensively used in traditional systems of medicine for managing gastrointestinal disorders and skin injuries. Among them, Azadirachta indica (Neem), belonging to the Meliaceae family, is known as a "village pharmacy" due to its wide pharmacological applications. Modern research confirms that neem exhibits significant anti-ulcer, antioxidant, anti-inflammatory, and antimicrobial activities, making it a potential therapeutic agent for gastrointestinal and dermal diseases.

PHYTOCHEMICAL PROFILE OF NEEM

Azadirachta indica (Neem), belonging to the family Meliaceae, is one of the most extensively studied medicinal plants in traditional and modern pharmacology due to its exceptionally rich and diverse phytochemical composition. The phytochemical profile of neem is highly complex, containing more than 140 structurally distinct compounds distributed across different parts of the plant, including leaves, bark, seeds, flowers, and fruits, each exhibiting unique therapeutic properties. The most important classes of compounds present in neem include limonoids, terpenoids, flavonoids, alkaloids, tannins, phenolic compounds, saponins, and glycosides, which collectively contribute to its broad pharmacological spectrum such as anti-inflammatory, antimicrobial, antioxidant, antidiabetic, hepatoprotective, and wound healing activities.

Among these, limonoids are considered the most biologically active and characteristic constituents of neem, with compounds such as azadirachtin, nimbin, nimbidin, nimbolide, salannin, and gedunin being widely investigated for their medicinal potential. Azadirachtin, primarily found in neem seeds, is known for its strong insecticidal and antifeedant properties, but recent pharmacological studies also highlight its indirect role in modulating inflammatory pathways and oxidative stress. Nimbin and nimbidin, predominantly found in neem seed oil and bark extracts, exhibit significant anti-inflammatory and anti-ulcer activities by inhibiting prostaglandin synthesis and reducing histamine-mediated gastric secretion, thereby contributing to gastroprotective effects. Nimbolide, another major limonoid isolated from neem leaves and flowers, has demonstrated potent antioxidant, anticancer, and anti-inflammatory properties by regulating NF-κB signaling pathways and inducing apoptosis in abnormal cells.

In addition to limonoids, neem contains a wide range of flavonoids such as quercetin, kaempferol, rutin, and myricetin, which are powerful natural antioxidants responsible for scavenging reactive oxygen species (ROS) and protecting

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cellular components from oxidative damage. These flavonoids play a crucial role in maintaining gastrointestinal mucosal integrity and accelerating tissue repair in wound healing processes by enhancing collagen synthesis and fibroblast proliferation. The phenolic compounds present in neem, including gallic acid, ferulic acid, and catechin derivatives, further strengthen its antioxidant potential and contribute to antimicrobial defense by disrupting microbial cell walls and inhibiting enzyme activity in pathogens. Tannins, another important group of phytochemicals in neem, possess astringent properties that help in wound contraction and formation of protective layers over damaged tissues, thereby promoting faster epithelialization.

Neem also contains various alkaloids, though in smaller quantities, which exhibit pharmacological effects such as analgesic and antipyretic activity by modulating central and peripheral nervous system pathways. Saponins present in neem contribute to its immunomodulatory and cholesterol-lowering effects by interacting with membrane sterols and enhancing immune cell activity. Glycosides in neem are also associated with cardioprotective and hepatoprotective effects, although their exact mechanisms are still under investigation. The synergistic interaction of these phytochemicals is considered a major reason behind neem's wide therapeutic applicability in traditional systems of medicine such as Ayurveda, Siddha, and Unani. From a gastroprotective perspective, neem phytochemicals act through multiple mechanisms including suppression of gastric acid secretion, enhancement of mucus production, reinforcement of gastric mucosal barrier, and reduction of oxidative stress-induced lipid peroxidation in gastric tissues.

Experimental studies have shown that neem leaf and bark extracts significantly reduce ulcer index in ethanol- and NSAID-induced gastric lesion models, mainly due to the presence of nimbidin and flavonoid-rich fractions. In terms of cutaneous wound healing, neem phytochemicals accelerate all three phases of wound repair hemostasis, inflammation, and proliferation by enhancing fibroblast migration, promoting angiogenesis, and increasing collagen deposition at the wound site. The antimicrobial compounds in neem also prevent wound infections caused by bacteria such as Staphylococcus aureus and Pseudomonas aeruginosa, which are common barriers to effective wound healing. Furthermore, neem's antioxidant compounds reduce oxidative stress in injured tissues, thereby minimizing cellular damage and promoting faster regeneration. Modern analytical techniques such as gas chromatography-mass spectrometry (GC-MS), high-performance liquid chromatography (HPLC), and liquid chromatography-mass spectrometry (LC-MS) have confirmed the presence and structural diversity of neem phytochemicals, validating traditional claims with scientific evidence.

Seasonal variation, geographical location, and extraction methods significantly influence the concentration and composition of these bioactive compounds, which is an important consideration in pharmaceutical standardization. Overall, the phytochemical profile of *Azadirachta indica* reflects a highly integrated system of bioactive molecules that work synergistically to produce multiple therapeutic effects. Its rich composition of limonoids, flavonoids, tannins, phenolics, alkaloids, saponins, and glycosides makes neem one of the most valuable medicinal plants in natural drug discovery and herbal medicine development. Recent pharmacological research (2010–2025) strongly supports its potential as a source of novel therapeutic agents for gastrointestinal disorders, infectious diseases, inflammatory conditions, and tissue regeneration therapies, although further clinical trials and standardized formulations are required to fully harness its medicinal potential in modern healthcare systems.

Neem contains more than 140 bioactive compounds, including:

1. Limonoids (azadirachtin, nimbolide)
2. Flavonoids (quercetin, kaempferol)
3. Triterpenoids (nimbin, nimbidin)
4. Polyphenols and tannins

These constituents are responsible for its pharmacological activities such as mucosal protection and skin regeneration.

MECHANISMS OF GASTROPROTECTION

Gastroprotection refers to the complex physiological and biochemical mechanisms that maintain the integrity of the gastric mucosa against a wide range of injurious factors such as gastric acid, pepsin, non-steroidal anti-inflammatory drugs (NSAIDs), ethanol, bile salts, Helicobacter pylori infection, and oxidative stress. The gastric mucosa is constantly exposed to aggressive luminal factors, yet it remains intact under normal conditions due to a highly coordinated defense

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system that includes mucus and bicarbonate secretion, epithelial cell renewal, adequate mucosal blood flow, prostaglandin synthesis, antioxidant activity, and immune regulation. One of the most important gastroprotective mechanisms is the mucus-bicarbonate barrier, which forms a gel-like protective layer over the epithe.

This mucus layer traps bicarbonate ions secreted by epithelial cells, creating a near-neutral pH microenvironment at the mucosal surface even when the gastric lumen is highly acidic. This prevents back-diffusion of hydrogen ions and protects epithelial cells from acid-induced injury. Mucins, the major glycoprotein components of mucus, also act as a physical barrier against mechanical and chemical damage, while simultaneously preventing bacterial adherence. Another crucial factor in gastroprotection is gastric mucosal blood flow, which ensures adequate oxygen and nutrient delivery to epithelial cells and facilitates rapid removal of toxic substances and hydrogen ions that may penetrate the mucus layer. Reduced blood flow is strongly associated with ulcer formation, whereas enhanced microcirculation promotes healing and resistance to injury. Prostaglandins, particularly those of the E2 and I2 series, play a central role in maintaining gastric mucosal defense.

They stimulate mucus and bicarbonate secretion, enhance mucosal blood flow, inhibit gastric acid secretion, and promote epithelial restitution after injury. NSAIDs induce gastric damage primarily by inhibiting cyclooxygenase enzymes (COX-1 and COX-2), thereby reducing prostaglandin synthesis and weakening mucosal defense. Therefore, preservation or restoration of prostaglandin levels is a key therapeutic strategy in gastroprotection. Epithelial cell regeneration and restitution also contribute significantly to mucosal defense. The gastric epithelium has a high turnover rate, and damaged cells are rapidly replaced through proliferation of stem cells located in gastric glands. Additionally, surviving epithelial cells can migrate to cover superficial lesions in a process known as restitution, which occurs within minutes to hours after injury. Growth factors such as epidermal growth factor (EGF), transforming growth factor-alpha (TGF-α), and fibroblast growth factors (FGFs) enhance these processes by stimulating cell proliferation, migration, and differentiation. Oxidative stress is another major contributor to gastric mucosal injury, particularly in conditions such as ischemia-reperfusion, ethanol exposure, and NSAID use.

Reactive oxygen species (ROS) such as superoxide anions, hydroxyl radicals, and hydrogen peroxide can damage lipids, proteins, and DNA in gastric epithelial cells, leading to cell death and ulcer formation. Gastroprotective systems therefore include both enzymatic antioxidants such as superoxide dismutase (SOD), catalase, and glutathione peroxidase and non-enzymatic antioxidants like glutathione, vitamin C, and vitamin E, which neutralize free radicals and reduce oxidative damage. Inflammation also plays a significant role in gastric mucosal injury, where activated neutrophils release proteolytic enzymes and ROS that worsen tissue damage. Anti-inflammatory mediators and inhibition of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 are therefore essential for maintaining mucosal integrity.

In addition, the gastric epithelial tight junction system, composed of proteins such as claudins, occludins, and zonula occludens (ZO-1), regulates paracellular permeability and prevents leakage of luminal acid into deeper tissue layers. Disruption of these tight junctions increases mucosal vulnerability and contributes to ulcerogenesis. Neural regulation, particularly through the vagus nerve, also influences gastric protection by modulating acid secretion and mucosal blood flow. Stress-related ulceration is often mediated by excessive vagal stimulation or sympathetic overactivity, which reduces blood flow and increases acid secretion. Moreover, the role of the immune system is increasingly recognized in gastroprotection, where mucosal immune cells such as macrophages, dendritic cells, and T-lymphocytes help maintain homeostasis by clearing pathogens and regulating inflammatory responses.

Another important dimension of gastroprotection involves the inhibition of gastric acid secretion itself. Parietal cells secrete hydrochloric acid through the H^+/K^+ ATPase proton pump, which is regulated by histamine, gastrin, and acetylcholine. Pharmacological or natural inhibition of these pathways reduces acid output and prevents mucosal erosion. In addition, endogenous nitric oxide (NO) is a key gaseous mediator that enhances mucosal blood flow, stimulates mucus secretion, and inhibits leukocyte adhesion to endothelial cells, thereby exerting strong protective effects. Disruption of nitric oxide synthesis leads to increased susceptibility to gastric injury. Recent research also highlights the importance of the gut microbiota in gastroprotection. A balanced microbial environment supports mucosal immunity, enhances epithelial barrier function, and prevents colonization by pathogenic bacteria such as H. pylori. Dysbiosis, or microbial imbalance, has been linked to increased inflammation and ulcer formation.

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Collectively, gastroprotection is not the result of a single mechanism but rather a dynamic interaction between physical barriers, biochemical mediators, cellular repair systems, vascular regulation, immune responses, and microbial balance. When these systems function in harmony, the gastric mucosa can withstand continuous exposure to aggressive factors. However, disruption of any component whether through drugs, infection, stress, or oxidative imbalance can shift the equilibrium toward injury and ulcer development. Therefore, therapeutic strategies for gastroprotection often aim to enhance endogenous defense mechanisms rather than simply suppressing acid secretion. Natural compounds, including plant-derived bioactives, have gained significant attention because they often act on multiple pathways simultaneously, including antioxidant, anti-inflammatory, cytoprotective, and antimicrobial mechanisms, thereby providing a holistic approach to maintaining gastric health.

Gastroprotection refers to prevention of gastric mucosal injury caused by acid, drugs, or stress. Neem exerts gastroprotective effects through multiple mechanisms:

1. Inhibition of gastric acid secretion
2. Strengthening of mucosal barrier
3. Antioxidant scavenging of free radicals
4. Reduction of inflammatory mediators (TNF-α, IL-6)
5. Enhancement of mucus production

Studies show neem bark extract significantly reduces gastric lesions induced by ethanol and NSAIDs in experimental models, suggesting strong anti-ulcer potential.

The gastroprotective mechanisms of *Azadirachta indica* are mainly mediated through its antioxidant, anti-inflammatory, and cytoprotective actions. Neem bioactive compounds such as nimbidin and flavonoids reduce gastric acid secretion and enhance the production of protective mucus in the stomach lining. It scavenges free radicals, thereby preventing oxidative damage to gastric epithelial cells. Additionally, neem inhibits the release of inflammatory mediators like prostaglandins imbalance and cytokines, which are responsible for mucosal injury. It also strengthens the gastric mucosal barrier and improves blood flow to the stomach lining, promoting faster repair of damaged tissues and reducing ulcer formation effectively.

MECHANISMS OF CUTANEOUS WOUND REGENERATION

Cutaneous wound regeneration is a complex, highly coordinated biological process that restores the structural and functional integrity of the skin after injury. The skin, being the largest organ of the body, acts as the first line of defense against environmental insults, pathogens, and physical trauma. When this barrier is disrupted, the body initiates a dynamic cascade of overlapping phases hemostasis, inflammation, proliferation, and remodeling. Each phase involves multiple cell types, cytokines, growth factors, and extracellular matrix components working in a tightly regulated manner to achieve tissue repair. Although wound healing is often described in sequential stages, in reality these phases overlap and influence one another continuously, ensuring efficient regeneration and restoration of tissue homeostasis.

The first stage of wound healing, hemostasis, begins immediately after injury. Blood vessels constrict to reduce blood loss, and platelets aggregate at the injury site to form a temporary clot. This clot not only stops bleeding but also serves as a provisional extracellular matrix that supports cell migration. Platelets release several growth factors such as platelet-derived growth factor (PDGF), transforming growth factor-beta (TGF-β), and vascular endothelial growth factor (VEGF). These molecules play a crucial role in initiating the healing cascade by attracting immune cells and fibroblasts to the wound site. Fibrin, fibronectin, and other matrix proteins within the clot provide structural support for incoming cells, establishing the foundation for tissue repair.

Following hemostasis, the inflammatory phase begins and typically lasts for several days. This phase is essential for clearing the wound of debris, pathogens, and damaged tissue. Neutrophils are the first immune cells to arrive at the wound site, where they perform phagocytosis to eliminate bacteria and necrotic material. They also release reactive oxygen species (ROS) and proteolytic enzymes that help break down damaged tissue. After neutrophils, macrophages become the dominant immune cells in the wound environment. Macrophages are considered central regulators of wound healing because they transition between pro-inflammatory (M1) and anti-inflammatory (M2) phenotypes. M1 macrophages continue the defense against infection, while M2 macrophages promote tissue repair by secreting growth

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factors such as TGF-β, interleukin-10 (IL-10), and VEGF. These signals help resolve inflammation and initiate the proliferative phase. A balanced inflammatory response is crucial, as excessive or prolonged inflammation can lead to chronic wounds, while insufficient inflammation may result in poor healing.

The proliferative phase represents a major stage of tissue regeneration and typically occurs between days 4 and 21 after injury. This phase involves re-epithelialization, angiogenesis, fibroblast proliferation, and extracellular matrix (ECM) formation. Keratinocytes from the wound edges and hair follicles migrate across the wound bed to restore the epidermal barrier. This process is driven by growth factors such as epidermal growth factor (EGF) and keratinocyte growth factor (KGF). Simultaneously, angiogenesis occurs, in which new blood vessels are formed from pre-existing vasculature to supply oxygen and nutrients to the healing tissue. VEGF plays a central role in this process by stimulating endothelial cell proliferation and migration. Fibroblasts also migrate into the wound site, where they synthesize collagen type III, glycosaminoglycans, and other extracellular matrix components that form granulation tissue. This granulation tissue is highly vascular and serves as the scaffold for further tissue development. Myofibroblasts, differentiated from fibroblasts under the influence of TGF-β, contribute to wound contraction by generating contractile forces that reduce wound size.

Extracellular matrix remodeling is another essential process that begins during the proliferative phase and continues into the remodeling phase. Matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) regulate the degradation and reorganization of ECM components. Initially, collagen type III is deposited in the wound, but it is gradually replaced by stronger collagen type I, which increases the tensile strength of the repaired tissue. The balance between ECM synthesis and degradation is critical for proper wound maturation. Dysregulation of this balance can result in excessive scarring or keloid formation.

The final stage of wound healing is the remodeling or maturation phase, which may last for several months to years. During this phase, the collagen fibers become more organized and cross-linked, improving the mechanical strength of the healed tissue. Vascular density decreases as unnecessary blood vessels are pruned, and cellularity within the wound reduces through apoptosis of excess cells. The scar tissue gradually becomes less cellular and more fibrous, resulting in a pale and stable scar. Although the repaired tissue regains strength, it rarely achieves the full functionality and elasticity of uninjured skin.

At the molecular level, wound regeneration is regulated by a network of signaling pathways including TGF-β/Smad, Wnt/β-catenin, Notch, and Hedgehog pathways. These signaling systems coordinate cell proliferation, differentiation, migration, and apoptosis. Growth factors such as PDGF, VEGF, EGF, and fibroblast growth factor (FGF) act as key mediators in intercellular communication during healing. In addition, the role of oxidative stress is significant, as controlled levels of reactive oxygen species function as signaling molecules, while excessive oxidative stress can delay healing and cause tissue damage.

Neural and immune interactions also influence wound regeneration. Nerve endings release neuropeptides such as substance P and calcitonin gene-related peptide (CGRP), which modulate inflammation and vascular responses. The immune system not only defends against infection but also regulates tissue repair through cytokine secretion and immune cell plasticity. Furthermore, stem cells derived from skin appendages, bone marrow, and adipose tissue contribute to regeneration by differentiating into keratinocytes and fibroblasts, enhancing tissue restoration.

In pathological conditions such as diabetes, chronic inflammation, or vascular insufficiency, the wound healing process becomes impaired. Reduced blood flow, impaired macrophage function, and decreased growth factor production lead to delayed healing and chronic ulcer formation. Conversely, excessive fibroblast activity and prolonged inflammation can result in hypertrophic scars and fibrosis. Therefore, understanding the mechanisms of cutaneous wound regeneration is crucial for developing therapeutic strategies that enhance healing and prevent complications.

Cutaneous wound regeneration is a highly regulated biological process involving hemostasis, inflammation, proliferation, and remodeling phases. It depends on a complex interplay of cellular activities, growth factors, cytokines, and extracellular matrix dynamics. Successful wound healing requires precise coordination among immune cells, fibroblasts, keratinocytes, endothelial cells, and molecular signaling pathways. Advances in biomedical research continue to uncover new therapeutic targets that can improve wound healing outcomes, particularly in chronic and non-healing wounds.

Wound healing involves inflammation, proliferation, and remodeling phases. Neem accelerates healing through:

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1. Stimulation of fibroblast proliferation
2. Increased collagen synthesis
3. Antimicrobial protection against wound infection
4. Enhanced angiogenesis
5. Reduced oxidative stress at wound site

Neem leaf extract has shown improved wound contraction rate and faster epithelialization in animal models.

PHARMACOLOGICAL EVIDENCE

Table 1: Experimental Evidence of Neem in Gastroprotection and Wound Healing

Study Type	Model	Neem Part Used	Observed Effect
In vivo ulcer model	Ethanol-induced gastric lesion in rats	Bark extract	Reduced ulcer index, mucosal protection
Experimental study	NSAID-induced gastric damage	Leaf extract	Increased mucus secretion, reduced acid damage
In vivo wound model	Excision wound in rats	Leaf extract ointment	Faster wound contraction and epithelialization
Review study	Tissue regeneration pathways	Whole plant	Promotes angiogenesis and collagen deposition

II. CONCLUSION

Azadirachta indica demonstrates significant gastroprotective and wound regenerative potential through multiple biological pathways including antioxidant defense, anti-inflammatory modulation, and antimicrobial activity. Its traditional use is strongly supported by modern scientific evidence. Further clinical studies are required to validate its therapeutic efficacy in humans.

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