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A Review on Wound Healing: Understanding its Phase and Mechanisms

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Abstract

Wound healing is a highly specialised, dynamic, multi-phase process that uses a complex mechanism to mend damaged or wounded tissues. Any disruption in the natural healing process of a wound leads to the production of aberrant scars and a chronic condition that makes the person more vulnerable to infections. The idea of wound healing as well as molecular and cellular processes were covered in this article. Emphasise the elements that influence wound healing as well. Using keywords like wound healing, cellular processes, and molecular mechanisms, secondary material was gathered from databases including PubMed, Google Scholar, Science Direct, and ResearchGate. It concluded that wound healing is a highly coordinated biological process driven by the interplay of keratinocytes, fibroblasts, immune cells, cytokines, and growth factors that collectively regulate inflammation, proliferation, and tissue remodeling. Key signaling pathways, including NF-κB, MAPK, and STAT3, orchestrate cellular responses essential for effective repair.

Keywords; Wound Healing, Cellular Mechanisms, Molecular Mechanisms, Role of platelets, Cytokines Phases.

INTRODUCTION

Wounds continue to be a difficult clinical issue in routine pathology, with both early and late consequences frequently contributing to morbidity and death. In an effort to mitigate the burden of wounds, extensive attention has been directed towards comprehending the physiological mechanisms of healing and wound management, with particular emphasis on innovative therapeutic strategies and the ongoing advancement of technologies tailored for both acute and chronic wound care [1]. The high incidence of wounds in general and their rising frequency in the aged population have resulted in their enormous social and economic effect on a global scale. There are many chronic, difficult-to-heal wounds, such as arterial, venous, diabetic, and pressure ulcers, that are linked to illnesses and anomalies that either directly or indirectly result in damage to the epidermal covering [2]. There are also many acute wounds. As people age, these chronic wounds become more common. Chronic wounds, for instance, are thought to impact 120 out of every 100,000 individuals between the ages of 45 and 65, and that number jumps to 800 out of every 100,000 individuals over 75 [2]. Furthermore, because of the problems associated with acute wounds, they might develop into chronic wounds, which are more challenging to treat, if their healing does not proceed in a prompt and organised manner [3].

The environment surrounding a wound changes as a person's health does, making wound healing a dynamic and intricate process. The process by which a live creature replaces injured or destroyed tissue with newly formed tissue is known as wound healing [4]. The dermis, or deeper connective layer, and epidermis, or surface epithelial layer, provide a barrier of defence against the outside world in healthy skin. Upon barrier disruption, a regulated cascade of biochemical processes is initiated to facilitate repair [5]. The process of wound healing is not only intricate but also delicate, rendering it vulnerable to disruption or failure that can result in the development of persistent, non-healing chronic wounds. Chronic wounds that do not heal can be caused by diabetes, arterial or venous disease, infection, and age-related metabolic deficits [6].

Phases of Wound Healing

The process of wound healing is dynamic and intricate, requiring the cooperation of several cellular and molecular processes. Haemostasis, inflammation, proliferation, and remodelling are its four overlapping stages. The effective advancement of healing depends on each phase, which is controlled by a complicated interaction between cell types, growth factors, and cytokines [7].

1. Haemostasis

Haemostasis represents the primary response to injury, with the objective of halting haemorrhage and forming a transient barrier against the external environment. Immediately following an injury, vasoconstriction takes place, and platelet aggregation causes a fibrin clot to develop. The recruitment of inflammatory cells to the wound site depends on this clot, which serves as a scaffold for later cellular activities [8].

2. Inflammation

The arrival of inflammatory cells, such as neutrophils and macrophages, to the wound site marks the start of the inflammatory phase, which happens soon after damage. First responders, neutrophils aid in the phagocytosis of pathogens and debris. Derived from monocytes, macrophages are essential in the latter phases of the inflammatory phase because they release cytokines and growth factors that promote the development of endothelium and fibroblast cells [8].

3. Proliferation

Granulation tissue, which is composed of extracellular matrix, fibroblasts, and new blood vessels, is a sign of the proliferative phase. The main cell type that produces collagen, which gives the healing tissue its tensile strength, is called fibroblasts. Through angiogenesis, endothelial cells create new blood vessels that feed the developing tissue with enough oxygen and nutrients. Furthermore, keratinocytes move from the borders of the incision to create a fresh layer of epithelium, which seals the wound [9].

4. Remodelling

The penultimate stage of wound healing, sometimes referred to as the maturation phase or remodelling phase, can take months or years. The newly created tissue goes through remodelling during this phase, which is typified by the reorganisation and cross-linking of collagen fibres to strengthen the scar tissue. Additionally, the wound contracts, decreasing its size and enhancing its aesthetic appeal. Restoring the tissue's structural integrity requires the remodelling phase [10].

Table 1: Phase of wound healing

Phase of healing	Time post Injury	Cells involved in phase	Function/ activity
Haemostasis	Immediate	Platelets	Clotting
Inflammation	Day 1-4	Neutrophils Macrophages	Phagocytises
Proliferation (granulation & Contraction)	Day 4-21	Macrophages Lymphocytes Anglocytes Neutrocytes Fibroblasts Keratinocytes	Fill defect Re-establish Skin function closure
Remodelling (maturation)	Day 21-2years	Fibrocytes	Develop Tensile strength

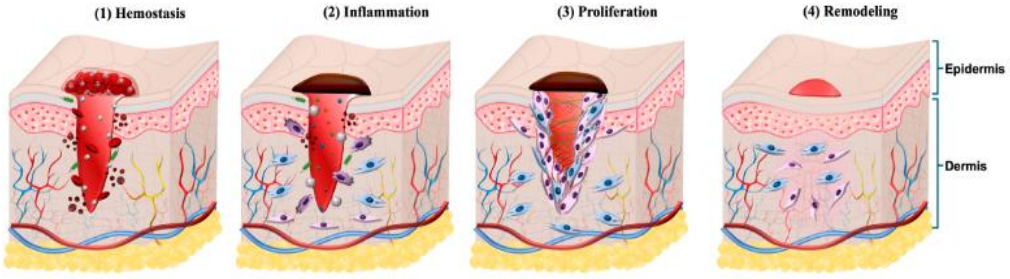

Figure 1: Illustration of four phases in the wound healing process.

Factors that affect wound healing

The pre-existing integrity of the injured skin due to age or medical treatments, comorbidities, medications, infection, hydration status, nutritional status, lifestyle choices, and pre- and post-operative care if surgery has been performed are some of the factors that may hinder the wound healing process [11].

The following are pertinent drugs, lifestyle variables, and comorbidities:

- **Diabetes:** A prevalent complication related to diabetes is peripheral neuropathy, which can result in foot ulceration. Peripheral ischaemia brought on by peripheral vascular disease is another consequence. Both issues impede wound healing overall and have an impact on the proliferative stage of healing [12].
- **Obesity:** Obesity is linked to a higher risk of ischaemia and insufficient oxygenation of the tissues, which can cause necrosis or delayed wound healing [13].
- **Necrosis:** Another condition that may prevent wound healing is unplanned tissue death, which necessitates debridement—the surgical removal of the damaged tissue—before healing can take place [14].
- **Poor nutrition:** Reduced blood vessel development, collagen synthesis, and fibroblast proliferation can result from malnutrition, which is commonly observed in older individuals. This eventually affects the healing process of wounds [15].
- **NSAIDs (non-steroidal anti-inflammatory drugs):** NSAIDs reduce pain by inhibiting PGE2, a mediator of inflammation. NSAIDs are known to prevent angiogenesis, which slows the healing of wounds. Additionally, NSAIDs promote the development of scars, especially when used during the proliferative stage [16].
- **Steroids:** Steroids' immunosuppressive and anti-inflammatory properties can impede wound healing by reducing collagen synthesis and fibroblast proliferation [16].
- **Radiation therapy:** The disintegration of skin tissue and slower healing of both new and old wounds can result from ionising radiation beams harming epithelial cells when they penetrate to the targeted tissues.
- **Chemotherapy:** Chemotherapeutic drugs have an impact on wound healing by reducing collagen formation and postponing the inflammatory stage of healing [16].
- **Smoking:** By producing vasoconstriction, cigarette smoking—more especially, the usage of nicotine—affects blood flow. Additionally, nicotine weakens the immune system, which may raise the risk of wound infection [17].
- **Alcohol:** Alcohol consumption is frequently linked to unhealthy eating practices, which may weaken the immune system. Alcohol may also hinder wound healing by reducing the production of collagen and angiogenesis, which weakens the creation of scar tissue and slows the healing process overall. Certain nutrients, including zinc and vitamin C, may also have a beneficial effect on wound healing [18].

Cellular and Molecular Mechanisms

(i) Role of platelets

Because they aid in haemostasis and the release of several growth factors and cytokines, platelets are essential in the early phases of wound healing.

1. **Haemostasis:** Platelets stick to the exposed collagen at the injury site to create a temporary plug to halt bleeding, and blood vessels constrict to lessen blood flow. Serotonin induces vasoconstriction, which further reduces blood flow, and von Willebrand factor (vWF), which promotes platelet adhesion, are released by platelets [19].
2. **Release of Growth Factors and Cytokines:** Upon activation, platelets release α-granules that are packed with cytokines and growth factors. By encouraging the migration and proliferation of "fibroblasts, smooth muscle cells, and endothelial cells, platelet-derived growth factor (PDGF)" aids in the development of new blood vessels and granulation tissue [7]. The synthesis of extracellular matrix, differentiation, and cell proliferation are all regulated by transforming growth factor-beta (TGF-β), which is necessary for tissue remodelling and healing [12]. Platelets additionally secrete inflammatory cytokines, including interleukin-1 (IL-1) and tumour necrosis factor-alpha (TNF-α), thereby recruiting immune cells to the injury location and facilitating the inflammatory response essential for initiating the healing cascade.
3. **Modulation of Inflammatory Response:** "Leukocytes and endothelial cells" can interact with platelets to affect the inflammatory response. They can facilitate the inflammatory stage of wound healing by improving leukocyte adherence to the endothelium and encouraging their migration into the tissue [20].

4. **Angiogenesis:** "Vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF)", which are released by platelets, encourage the development of new blood vessels (angiogenesis) in the wound bed and supply the nutrients and oxygen needed for tissue healing [14].

(ii) White blood cells, and other immune cells

Due to their ability to coordinate "the inflammatory response, remove debris, and facilitate tissue regeneration, white blood cells (WBCs) and other immune cells" are essential for wound healing. An explanation of their duties is provided below:

1. **Neutrophils:** Neutrophils constitute the initial immune cells to reach the site of injury, typically arriving within hours following the trauma. By phagocytosing bacteria, detritus, and apoptotic cells, they aid in the removal of pathogens and cellular debris from the wound. Additionally, neutrophils produce chemokines and cytokines that promote inflammation and draw additional immune cells to the wound site [19].
2. **Macrophages:** Macrophages are essential for both the reparative and inflammatory stages of wound healing. By generating pro-inflammatory cytokines such "interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α)", macrophages contribute to the amplification of the inflammatory response during the early inflammatory phase. Macrophages change into a pro-healing phenotype when the wound moves into the repair phase, encouraging tissue remodelling and repair. Growth factors including "platelet-derived growth factor (PDGF) and transforming growth factor-beta (TGF-β)" are secreted by pro-healing macrophages and promote collagen production and fibroblast proliferation [19].
3. **Lymphocytes:** T cells, B cells, and other lymphocytes regulate the healing of wounds. Through the production of cytokines that either stimulate or inhibit inflammation, T cells regulate the immune response. By generating antibodies that aid in the removal of infections, B cells aid in the healing of wounds [8].
4. **Mast Cells:** Early on in the healing process, mast cells have a role, especially during the inflammatory phase. In order to attract additional immune cells to the wound site, they produce histamine and other inflammatory mediators that enhance vascular permeability and encourage vasodilation [3].
5. **Other Immune Cells:** Additional immune cells, including dendritic cells and natural killer cells, also contribute to the wound healing process, although their particular functions remain less comprehensively elucidated. Dendritic cells may support tissue healing and aid in immune response coordination. The removal of contaminated or damaged cells from the wound site may be aided by natural killer cells.

(iii) Cytokines and growth factors

By controlling "inflammation, cell proliferation, migration, and extracellular matrix (ECM) formation", cytokines and growth factors are essential for wound healing. An explanation of their duties is provided below:

1. **Cytokines:**

- **Interleukin-1 (IL-1):** One important pro-inflammatory cytokine that is essential to the initial phases of wound healing is IL-1. It increases the production of other cytokines and growth factors, encourages inflammation, and draws immune cells to the wound site [9].
- **Interleukin-6 (IL-6):** Another pro-inflammatory cytokine that controls the immune system and promotes the synthesis of acute-phase proteins is IL-6. Additionally, it encourages distinct cell types involved in wound healing to proliferate and differentiate.
- **Tumor Necrosis Factor-alpha (TNF-α):** The powerful pro-inflammatory cytokine TNF-α is mostly generated by macrophages. It attracts immune cells, increases inflammation, and triggers the synthesis of more growth factors and cytokines [9].
- **Interleukin-8 (IL-8):** One chemokine that is essential for attracting neutrophils to the wound site is IL-8. It contributes to the early inflammatory response and aids in the removal of debris and germs from the site [15].

2. **Growth Factors:**

- **Platelet-Derived Growth Factor (PDGF):** Platelets and macrophages release PDGF, which is essential for wound healing. It promotes the development of granulation tissue and new blood vessels by inducing the migration and proliferation

of endothelial, smooth muscle, and fibroblast cells [7].

- **Transforming Growth Factor-beta (TGF-β):** A multipurpose growth factor, TGF-β controls inflammation, cell division, proliferation, and the synthesis of extracellular matrix (ECM) in wound healing. It controls the activity of immune cells and fibroblasts and encourages the development of granulation tissue [12].
- **Vascular Endothelial Growth Factor (VEGF):** An important regulator of angiogenesis, VEGF encourages the development of new blood vessels in the wound bed. It is necessary to provide the wound site with nutrients and oxygen, which promotes tissue healing [7].
- **Fibroblast Growth Factor (FGF):** FGFs promote fibroblast, endothelial, and keratinocyte migration and proliferation. During wound healing, they are essential for angiogenesis, re-epithelialization, and ECM production [13].

LITERATURE REVIEW

(Ana Paula de Araújo Boleti et al., 2025) [21] examines the basic processes that underlie wound healing, including as the functions of growth factors and cytokines in the local microenvironment, with an emphasis on antimicrobial peptides (AMPs) as immune modulators and treatment agents for chronic wounds. Notably, it has been demonstrated that AMPs like LL-37 can decrease biofilm density by as much as 60%, underscoring their dual function in regulating host immune responses and thwarting bacterial infections that persist. Beyond conventional biological processes, it also looks at new technologies that are revolutionising the sector, including "gene therapy, regenerative medicine, smart dressings, 3D bioprinting, AI-driven treatments, and organoid models".

(Esad et al., 2025) [9] The biological process of wound healing is strictly controlled and includes tissue remodelling, inflammation, haemostasis, and proliferation. Wound healing may be postponed or becoming chronic if these stages are disturbed. The bioactive substances found in medicinal plants, including "flavonoids, alkaloids, tannins, and other phytoconstituents", have shown notable anti-inflammatory, antioxidant, and immunomodulatory properties that alter these pathways. Through metal-chelating action, "antioxidant enzyme activation, and reactive oxygen species (ROS) neutralisation", tannins aid in repair. "Tetrandrine, oxymatrine, and berberine" are examples of alkaloids that block NF-κB signalling, which lowers pro-inflammatory cytokines like TNF-α and IL-1β. In addition to defending against oxidative stress and promoting the proliferation of fibroblasts and keratinocytes—two essential processes in tissue regeneration—flavonoids also control inflammatory mediators and enzymes, such as COX and phospholipase A2. When combined, these substances lessen oxidative stress and encourage cellular migration and proliferation, which speeds up wound closure. Therefore, medicinal plants provide interesting complementary methods for managing wounds.

(Jin et al., 2025) [19] describes the abnormalities that explain disordered healing, summarises the cellular and molecular pathways of cutaneous repair, and assesses new ideas and translational approaches. The development of personalised therapies and precision wound medicine to enhance results and quality of life is supported by the integration of traditional models with new insights. When the biological process of wound healing is disrupted, it can result in fibrotic scarring or persistent wounds. Recent developments in biomaterials, regenerative medicine, and omics technology have changed how we think about repair and opened up exciting new treatment options. However, patient variability, concomitant illnesses, and targeted delivery restrictions make it difficult to translate these findings into successful therapeutic therapies.

(S et al., 2024) [8] gives an in-depth overview of the processes, variables, and treatments that affect wound healing, with an emphasis on managing chronic wounds and particular populations. The effect of many variables on wound healing, including systemic, local, and patient-related factors, on healing results is investigated. Numerous wound healing methods and therapies being investigated, such as dressings, operations, and new technologies like growth factor and stem cell therapy. The management of chronic wounds is discussed, with a focus on the difficulties and interdisciplinary methods necessary for successful treatment. Patients with certain disorders including "diabetes and autoimmune diseases", as well as paediatric and elderly patients, are taken into consideration due to their distinct wound healing demands and features.

(Freedman et al., 2023) [22] Millions of people's daily lives are still disrupted by skin injuries, which also lead to extended hospital admissions, infections, and fatalities. Clinical practice has been enhanced by advancements in wound healing devices, which have mostly addressed macroscale healing rather than the underlying microscale pathology. The lack of agreement on the best ways to treat

wounds with a variety of wound-healing products has spurred the development of novel treatments. We provide an overview of the latest developments in clinical trials and commercially available innovative pharmacological, biologic, and biomaterial treatments for wound healing. We also exchange ideas on how to successfully and quickly translate innovative integrated wound healing therapies.

(Ayavoo et al., 2021) [23] Due to their capacity to promote quicker tissue regeneration during wound repair, stem cells may be able to overcome the limitations of existing wound care procedures. Understanding the molecular principles behind stem cells has emerged as a key and exciting area of scientific research. The kinds and functions of stem cells in wound care management, as well as the current conventional and contemporary therapies for wound healing, are the main topics of this study. The basic molecular characterisation of stem cells and the variables affecting their molecular processes in wound healing are also included in this study. Stem cells have a lot of promise for wound healing and tissue regeneration. In a clinical setting, MSC treatment shown significant promise as a therapeutic method for wound care management. For contemporary regenerative drugs, the use of MSCs in tissue regeneration and wound healing is a significant difficulty.

(Wilkinson & Hardman, 2020) [24] A wide range of cellular processes underpin the intricate and dynamic process of wound healing, which requires close coordination in order to effectively restore injured tissue. Dysregulation of cellular processes associated with wound repair, such as those observed in diabetes and during the ageing process, can result in impaired healing and the development of chronic, non-healing lesions. Because of their high frequency and recurrence, these wounds represent a substantial economical burden. Therefore, a better molecular and clinical knowledge of the processes underlying wound healing is urgently needed. Here, we go over the cellular underpinnings of tissue restoration and talk about how new and existing knowledge of wound pathology may help build effective wound treatments in the future.

(Wang et al., 2018) [25] The physiological process of wound healing is crucial for preserving the skin's integrity following damage, whether intentional or the result of an accident. Three consecutive but overlapping phases—the haemostasis/inflammatory phase, the proliferative phase, and the remodelling phase—are involved in normal wound healing. Normal bodily function is hampered by abnormal "wound healing, such as excessive wound healing (hypertrophic scar and keloid) or chronic wound healing (ulcer)". Numerous advanced experimental investigations have shed light on wound healing. "(i) wound healing; (ii) wound healing in foetuses and adults; (iii) prostaglandins and wound healing; (iv) the pathogenesis of excessive wound healing; (v) the epidemiology of excessive wound healing; (vi) in vitro and in vivo studies for excessive wound healing; (vii) stem cell therapy for excessive wound healing; and (viii) the prevention strategy for excessive wound healing" are the main topics of this article, which focusses on information published after 2010.

RESEARCH OBJECTIVE

(i) To study the concept of wound healing and it's various phases.
(ii) To study the factors that affect wound healing.
(iii) To study the cellular and molecular mechanisms of wound healing.
(iv) To study the various researchers work on cellular and molecular mechanisms of wound healing.

RESEARCH METHODOLOGY

The research methodology for this review involved a systematic literature search across major scientific databases, including PubMed, Scopus, Web of Science, Google Scholar, and ScienceDirect. Keywords such as wound healing, cellular mechanisms, molecular mechanisms, were used. Studies published between 2014 and 2025 were screened based on relevance, scientific validity, and focus on cellular and molecular mechanisms. Data from peer-reviewed articles, and dissertations were extracted, compared, and synthesized. Publications lacking methodological clarity or reliable experimental evidence were excluded to ensure accuracy and quality of the review.

CONCLUSION

Wound healing is a highly coordinated biological process driven by the interplay of keratinocytes, fibroblasts, immune cells, cytokines, and growth factors that collectively regulate inflammation, proliferation, and tissue remodeling. Key signaling pathways, including NF-κB, MAPK, and STAT3, orchestrate cellular responses essential for effective repair. Medicinal plants and their bioactive constituents, such as flavonoids, alkaloids, and tannins, demonstrate significant potential to modulate these molecular mechanisms through anti-inflammatory, antioxidant, and profibrotic actions, thereby supporting cellular proliferation, migration, and extracellular matrix formation. Despite advances in omics technologies, biomaterials, and regenerative strategies, translating molecular insights into consistent clinical outcomes remains challenging due to

patient variability and comorbidities such as diabetes, obesity, malnutrition, and lifestyle factors. Understanding these intricate processes is therefore crucial for developing targeted, evidence-based therapeutic interventions. Continued research integrating molecular biology, phytotherapy, and clinical science will be vital for optimizing wound care and improving healing outcomes.

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