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# CELLULAR MECHANISM OF WOUND HEALING ON SKIN

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There are four phases of wound healing:

- Haemostasis – establishes the fibrin provisional wound matrix and platelets provide initial release of cytokines and growth factors in the wound.
- Inflammation – mediated by neutrophils and macrophages which remove bacteria and denatured matrix components that retard healing, and are the second source of growth factors and cytokines. Prolonged, elevated inflammation retards healing due to excessive levels of proteases and reactive oxygen that destroy essential factors.
- Proliferation – fibroblasts, supported by new capillaries, proliferate and synthesize disorganized ECM. Basal epithelial cells proliferate and migrate over the granulation tissue to close the wound surface.
- Remodelling – fibroblast and capillary density decreases, and initial scar tissue is removed and replaced by ECM that is more similar to normal skin. ECM remodelling is the result of the balanced, regulated activity of proteases.

Cellular functions during the different phases of wound healing are regulated by key cytokines, chemokines and growth factors. Cell actions are also influenced by interaction with components of the ECM through their integrin receptors and adhesion molecules. MMPs produced by epidermal cells, fibroblasts and vascular endothelial cells assist in migration of the cells, while proteolytic enzymes produced by neutrophils and macrophages remove denatured ECM components and assist in remodelling of initial scar tissue.

**1. Haemostasis**

Haemostasis occurs immediately following an injury.To prevent exsanguination, vasoconstriction occurs and platelets undergo activation, adhesion and aggregation at the site of injury. Platelets become activated when exposed to extravascular collagen, which they detect via specific integrin receptors, cell surface receptors that mediate a cell's interactions with the extracellular matrix. Once in contact with collagen, platelets release the soluble mediators and adhesive glycoproteins, which signal them to become sticky and aggregate. The key glycoproteins released from the platelet alpha granules include fibrinogen, fibronectin, thrombospondin, and von Willebrand factor. As platelet aggregation proceeds, clotting factors are released resulting in the deposition of a fibrin clot at the site of injury. The fibrin clot serves as a provisional matrix. The aggregated platelets become trapped in the fibrin web and provide the bulk of the clot. Their membranes provide a surface on which inactive clotting enzyme proteases are bound, become activated and accelerate the clotting cascade.

Growth factors are also released from the platelet alpha granules. Neutrophils and monocytes are then recruited by PDGF and TGF-β from the vasculature to initiate the inflammatory response

**2. Inflammation**

*Inflammation*, the next stage of wound healing occurs within the first 24 hours after injury and can last for up to 2 weeks in normal wounds and significantly longer in chronic non-healing wound. Mast cells release granules filled with enzymes, histamine and other active amines, which are responsible for the characteristic signs of inflammation around the wound site. Neutrophils, monocytes,

and macrophages are the key cells during the inflammatory phase. They cleanse the wound of infection and debris and release soluble mediators such as proinflammatory cytokines that are involved in the recruitment and activation of fibroblasts and epithelial cells in preparation for the next phase in healing.

**3. Proliferative phase**

The milestones during the *proliferative phase* include replacement of the provisional fibrin matrix with a new matrix of collagen fibers, proteoglycans, and fibronectin to restore the structure and function to the tissue. Another important event in healing is angiogenesis, the in-growth of new capillaries to replace the previously damaged vessels and restore circulation. Other significant events in this phase of healing are the formation of granulation tissue and epithelialization. Fibroblasts are the key cells in the *proliferative phase* of healing.

***Remodelling***

Remodelling is the final phase of the healing process in which the granulation tissue matures into scar and tissue tensile strength is increased . The maturation of granulation tissue also involves a reduction in the number of capillaries via aggregation into larger vessels and a decrease in the amount of glycosaminoglycans and the water associated with the glycosaminoglycans (GAGs) and proteoglycans. Cell density and metabolic activity in the granulation tissue decrease during maturation. Changes also occur in the type, amount, and organization of collagen, which enhance tensile strength. Initially, type III collagen was synthesized at high levels, but it becomes replaced by type I collagen, the dominant fibrillar collagen in skin. The tensile strength of a newly epithelialized wound is only about 25% of normal tissue. Healed or repaired tissue is never as strong as normal tissues that have never been wounded. Tissue tensile strength is enhanced primarily by the reorganization of collagen fibers that were deposited randomly during granulation and increased covalent cross-linking of collagen molecules by the enzyme, lysyl oxidase, which is secreted into the ECM by fibroblasts. Over several months or more, changes in collagen organization in the repaired tissue will slowly increase the tensile strength to a maximum of about 80% of normal tissue.