

WOUND HEALING GLOSSARY

Collagen is one of the principal components of connective tissue. Collagen is formed when tropocollagen molecules polymerize. The five different types are distinguished from one another by morphology, amino acid composition, and physical properties. Type III collagen is the type which appears in the wound initially, about 4 days after injury. However, it is quickly replaced by Type I collagen, which constitutes 90% of the total collagen in the body, and forms the major collagen type found in mature scar tissue.

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Fibrin is an essential component for blood clotting. Fibrin monomers are derived from an inactive precursor, fibrinogen. **Fibrinogen** becomes active, losing four low molecular weight peptides, in response to thrombin. The monomers immediately polymerize with similar molecules to form long fibrin threads which constitute the clot reticulum. Fibrin stabilizing factor, found in platelets, cause covalent bonds to form between fibrin monomer molecules and the multiple cross-linkages between adjacent fibrin threads.

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Kinins relax vascular smooth muscle, inciting vasodilation and increasing permeability and blood flow. Kinin is a general term for a group of polypeptides derived from kininogen. In the plasma enzyme cascade following injury, clotting factor XII (Hageman factor) activates the conversion of prekallikrein to plasma kallikrein. Plasma kallikrein cleaves kininogen into kinin.

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Macrophages are recruited to the wound area in the form of monocytes as a result of chemotactic stimuli and differentiation in tissues. A pivotal player in inflammatory reaction, macrophages are also responsible for bacterial phagocytosis and stimulation of other wound healing processes. They regulate lymphocyte responses and the coagulation/fibrinolytic pathway. Primary inflammatory mediators associated with macrophage activity are: lysosomal enzymes (acid hydrolases and neutral proteases), cationic proteins, phospholipase A₂, prostaglandins and leukotrienes, interleukin-1, and plasminogen activator.

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Mast cells are localized in the connective tissue of the body, and are especially prevalent in the dermis of the skin. They have receptors for IgE on their cell surface, and mediate a variety of allergic and inflammatory conditions. Their dense cytoplasmic granules contain histamine, acid mucopolysaccharides (including heparin), serine proteases, and chemotactic mediators for neutrophils and eosinophils.

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Platelets are round or oval disks formed from megakaryocytes which fragment either in the bone marrow or after entering the blood. Platelets have two types of granules: dense granules, which are a source of serotonin, histamine, calcium, adenosine diphosphate(ADP); and alpha-granules, containing fibrinogen, coagulation proteins, platelet-derived growth factors (PDGF), and other peptides and proteins. After injury, platelets release the arachidonic acid metabolite thromboxane A₂, which mediates smooth muscle constriction. Platelets adhere to exposed collagen in the vessel

wall, change form and aggregate to form a thrombus, and degranulate to induce changes in vascular permeability.

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Polymorphonuclear leukocytes (PMNs) are granulocytes distinguished by their multilobed nucleus. PMNs may be classified as neutrophils, eosinophils, and basophils. Neutrophils are by far the most abundant type of polymorph, and are among the first inflammatory cells to be recruited to the site of injury.

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Thrombin is derived from the unstable protein **prothrombin**, an alpha₂-globulin. Thrombin is an enzyme with proteolytic properties, cleaving fibrinogen molecules into fibrin monomers, a necessary step in blood coagulation. During clot formation, most of the thrombin adsorbs to the fibrin threads as they develop, preventing excessive spreading of the clot.

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Thromboplastin is present in tissues, platelets, and leukocytes. In the presence of calcium ions, it is necessary for the conversion of prothrombin to thrombin, a necessary step in the coagulation of blood.

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