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Healing of Wounds: Essential Physiological Process

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Description

In the end, they result in repair, which consists of the substitution of specialized structures brought about by the deposition of collagen, and regeneration, which corresponds to the process of cell proliferation and posterior differentiation through pre-existing cells in the tissue and/or stem cells. These mechanisms do not mutually exclude themselves, that is, after a skin lesion, in the same tissue, regeneration and repair can occur, depending on the cell strains compromised by the injury. Tissue regeneration and repair processes occur after the onset of the lesion. Be that due to the trauma or resulting from a specific pathological condition.

Cutaneous wound healing is an essential physiological process consisting of the collaboration of many cell strains and their products. Attempts to restore the lesion induced by a local aggression begin very early on in the inflammatory stage. One lesion is created by all of the stimuli that break the physical continuity of functional tissues. The stimuli that cause lesions can be external or internal, as well as physical, chemical, electric, or thermal. Moreover, the lesions can result in damage to specific organelles or to cells as a whole.

Inflammatory Stage

Normally, cell response is established within the first 24 hours and can extend for up to two days. A quick activation of the immune cells in the tissue may also occur, as happens with mastocytes, gamma-delta cells, and Langerhans cells, which secrete chemokine's and cytokines. Inflammation is a localized and protective tissue response that is unleashed by the lesion, causing tissue destruction. Inflammatory cells play an important role in wound healing and contribute to the release of lysosomal enzymes and reactive oxygen species, as well as facilitate the clean-up of various cell debris.

Tissue repair is a simple linear process in which the growth factors cause cell proliferation, thus leading to an integration of dynamic changes that involve soluble mediators, blood cells, the production of the extracellular matrix, and the proliferation of parenchymal cells. The skin healing process, according to Mitchel et al., illustrates the principles of repair for the majority of tissues. Cell response in the inflammatory stage is characterized by the influx of leukocytes in the wound area. Such a response is very quick and coincides with the key signs of inflammation, which are revealed by the edema and the erythema at the location of the lesion.

Parallel to all of the aforementioned events, the epithelial coating cells, through the action of specific cytokines, proliferate and migrate from the borders of the wound in an attempt to close it, which is called reepithelialization. Epidermal cells of hair follicles quickly remove the coagulation and damaged reported that the epidermal germ cells of the hair follicle, which create the hair bulb, serve as a reservoir for keratinocytes in the healing process. Approximately ten hours after the onset of the lesion, there is a development and stretching of the pseudopod projections of the keratinocytes, a loss of the extracellular matrix-cell and cell-cell contacts, a retraction of the tonofilaments, and the formation of actin filaments in the extremities of its cytoplasms. Buckley argues that the interaction of leukocytes and stromal cells during an acute inflammatory response resolves around the inflammatory focus.

Neutrophils are known for expressing many pro-inflammatory cytokines and a large quantity of highly active antimicrobial substances, such as Reactive Oxygen Species (ROS), cationic peptides, and proteases at the location of the lesion. The inflammatory response continues with the active recruitment of the neutrophils in response to the activation of the complement system, platelet degranulation, and bacterial degradation products. These are attracted by many inflammatory cytokines produced by activated platelets, endothelial cells, and degradation products of pathogenic agents. In this manner, the neutrophils are the primary activated and recruited cells that play a role in the clean-up of the tissue, as well as contribute to the death of invading agents.

The reepithelialization of a wound by keratinocytes is performed by the combination of the proliferative stage with the migration of cells near the lesion. The migration of keratinocytes occurs in the direction of the remaining skin of the lesion to its extremities.

The epithelial cells undergo an epithelial-mesenchymal transition (EMT) and migrate to the organs to differentiate themselves in their mesenchymal components, including fibroblasts, smooth muscle cells of blood vessels, and, even more likely, pericytes. The skin, as well as the intestines, liver, lungs, and glandular tissues, contains epithelial and mesenchymal cells. When the migration ceases, possibly due to a result of the inhibition caused by contact, the keratinocytes are reconnected to the substrate and reconstruct the basal membrane. There is then the culmination of its differentiation process to generate the newly stratified epidermis.

Epithelial-Mesenchymal Transition

Considering that the hedgehog ligand can regulate angiogenesis, its signaling can also influence tissue remodeling. Though first studied in a tumor context, the expression of normal promoter inhibitor regulatory genes from cell growth, which are expressed in the cells present in the extracellular matrix, occurs in the healing process. Liu et al. described that the epithelial-mesenchyme transition can be regulated by microRNAs, as seen in miR-221, as well as by other oncogenes. The epithelial cells firmly adhere one to another, forming layers in which the basoapical polarity can be observed. The mesenchyme cells are non-polarized and are capable of movement, such as individual cells due to the loss of intercellular connections.

The biological process that occurs in the epithelial-mesenchyme transition makes it possible for a polarized epithelial cell to undergo molecular changes, acquiring a mesenchyme phenotype, with

migratory capacity through the extracellular matrix, resistance to apoptosis, and increase in the production of the components of the matrix.

Hedgehog is a family of secreted signaling molecules that are involved in many processes, including the role as key agents in the standardization of numerous tissues types. This family comprehends a cascade of proteins that regulate diverse biological processes, such as embryological development, homeostasis, tumor genesis, and tissue repair. Tome-Garcia et al. reported that the overexpression of the ras gene and the ERBB2 resulted in an increase in cell mobility in the extracellular matrix and in the metastatic potential of prostate cancer. Though these alterations have been described in the tumor microenvironment, such results can be observed in the healing process as well.