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The Guardians of the Skin Immune System

Editorial

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It has been well established that the epithelia are major components of the immune system. Together with secondary lymphoid organs (ig. lymph nodes, Payer´s patch, etc.), they comprise the activation sites of the Skin and Mucosal Immune Systems, also known as Peripheral or Tegumentary Immune System. Therefore, the epidermis is an essential place for immune modulation.

Based on a series of articles by Ralf Paus and collaborators from the University of Hamburg in Germany, in relation to hair follicle as a zone of immune privilege, we now know that a series of molecules produced by keratinocytes may play a key role in controlling skin homeostasis. These molecules are pleiotropic bioactive peptides, such as alpha-melanocyte-stimulating hormone (α-MSH), insulin-like growth factor 1 (IGF-1), indoleamine 2,3-dioxygenase (IDO), interleukin-10 (IL-10), IL-6, transforming growth factor-beta1 (TGF-β1) and neuroimmune regulatory proteins (e.g. CD200, CD47).

For instance, TGF-β1 induces stimulatory or inhibitory effects in human T cells, which is dependent on the T cell differentiation status and the stimulation conditions. Thus, TGF-β1 has distinct roles in T cell development, homeostasis, tolerance and differentiation.

The events that occur in wound-healing and the role of TGF-β1 may place these ideas into context. Recent evidences suggest that the release of enzymes generates by trauma, such as tryptases, promote epithelial cell division and produce essential guardians factors such as IL-6, TGF-β1, platelet-activating endothelial factor (PAF), α-MSH and IGF-I, which are crucial in enabling the recruitment of cells with active dermotropism.

Shortly after the initial injury coagulation occurs. There, platelets secrete platelet-derived growth factor (PDGF), TGF-β, and platelet factor IV, that initiate the wound-healing cascade by attracting fibroblasts, endothelial cells and macrophages into the wound. Platelets also secrete vasoactive neuropeptides, such as serotonin and histamine, which increase local microvascular permeability, allowing more inflammatory cells to infiltrate the wound.

In early inflammatory phase, the neutrophil is the predominant cell type, capable of preventing infection and removing debris by releasing proteinases and other enzymes into the wound. During late inflammatory response, macrophages, which are drawn to the wound by epithelial TGF-β, PDGF and various complement components, produce more TGF-β, PDGF, IGF-1 and basic fibroblast growth factor (bFGF). These cytokines regulate the production and organization of the extracellular matrix by fibroblasts, and the proliferation of smooth muscle cells and endothelial cells necessary for angiogenesis. The recruited fibroblasts begin to synthesise collagen starting in the remodelling phase of wound repair.

It is important to keep an eye on these epithelial guardians of the immune homeostasis, more that looking into inflammatory responses that most probably are an epiphenomenon.

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