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Regulating AGE/RAGE signalling to revert macrophage dysfunction in wound microenvironment to assist healing in diabetic scenario

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Wound healing progress through four interconnected stages- hemostasis, inflammation, proliferation and remodelling. However, hallmarks of diabetic wound healing include a prolonged inflammatory phase that leads to a disturbed distribution of classically and alternatively activated macrophage population, altered protease equilibrium, degradation of the deposited ECM, and sustained bacterial infection leading to impaired healing. Several studies have reported that the chronic wound microenvironment also contains advanced glycation end product (AGE), reactive oxygen species (ROS), inflammatory cytokines, and low levels of growth factors (GF) over an extended duration of time. The accumulation of AGE and its binding to the receptor (RAGE) and repeated microbial infection enhances ROS production resulting in constant infiltration of inflammatory cells at the wound bed. The upregulation of AGE/RAGE signalling is also responsible for the increase in apoptotic bodies, decrease in the phagocytotic potential and loss of phenotypic switch of the wound macrophages. As a result, the apoptotic cell load increases in the wound bed and generates more proinflammatory stimulus and cytokines. Though the initial impulse helps recruit the inflammatory cells, however, their prolonged expression delays the progression of healing. Overexpression of proteases like matrix metalloproteinases (MMPs) degrades the essential molecules (GFs) necessary for the healing. As a result, the signal required for the migration and proliferation of fibroblasts, keratinocytes, along with the synthesis of collagen, is lowered in the chronic wound bed. Overall, the existing anarchy results in a situation that is completely reverse of the optimal conditions required for efficient healing and causes disoriented deposition of mechanically weak collagen fibres, reducing the strength and quality of the regenerated skin. Thus, our approach aims to regulate the microenvironmental anarchy by addressing the altered macrophage function to provide a holistic treatment alternative for chronic wounds.

Recent Publications

1. Kimball A, Schaller M, Joshi A, Davis F M, et al. Ly6CHi Blood Monocyte/Macrophage Drive Chronic Inflammation and Impair Wound Healing in Diabetes Mellitus. Arteriosclerosis, thrombosis, and vascular biology, (2018) 38(5), 1102–1114.

2. Dokumacioglu E, Iskender H, Sen T M, et al. The Effects of Hesperidin and Quercetin on Serum Tumor Necrosis Factor-Alpha and Interleukin-6 Levels in Streptozotocin-induced Diabetes Model. Pharmacognosy magazine, (2018) 14(54), 167–173.

3. Barman P K & Koh T J. Macrophage Dysregulation and Impaired Skin Wound Healing in Diabetes. Frontiers in cell and developmental biology, (2020) 8, 528.

Speaker Biography

Ahana Banerjee is a PhD scholar at the Centre for Biomedical Engineering (CBME), which is a joint initiative of the Indian Institute of Technology, Delhi and the All India Institute of Medical Sciences, Delhi. She is a 5th year PhD scholar developing materials for the treatment of chronic wounds. Her research interest lies in developing biomimetic and sustainable therapies to modulate the chronic wound microenvironment to elevate the standard of living in patients suffering from diabetic foot ulcers by improving the standard of care and reducing cases of amputations. She explores various biomaterials and drug delivery techniques to address the altered physiological conditions associated with chronic wound healing and pivot the therapeutic effect around regulating different signal transduction pathways to achieve the desired result.

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