WOUND HEALING

TWEAK/FN14 SIGNALS MEDIATE BURN WOUND REPAIR

Yumin Xia [(1)] - Jing Liu [(1)] - Yale Liu [(1)] - Weihui Zeng [(1)]

The Second Affiliated Hospital, School Of Medicine, Xi'an Jiaotong University, Department Of Dermatology, Xi'an, China [(1)]

Background: Tumor necrosis factor-like weak inducer of apoptosis (TWEAK) acts by engaging with fibroblast growth factor-inducible 14 (Fn14) to regulate inflammatory responses, fibrosis, and tissue remodeling, which are central in the repair processes of wounds.

Objective: This study aims to explore the potential role of the TWEAK/Fn14 pathway in the healing of cutaneous burn wounds.

Materials and Methods: Third-degree burns were introduced in the wild-type and Fn14-deficient BALB/c mice, followed by evaluation of wound areas and histological changes. The downstream cytokines including growth factors were also examined in lesional skin. Moreover, human dermal microvascular endothelial cells (DMECs) and dermal fibroblasts were analyzed in vitro upon TWEAK stimulation.

Results: The healing of burn wounds was delayed in Fn14-deficient mice and was accompanied by the suppression of inflammatory responses, growth factor production, and extracellular matrix synthesis. Moreover, TWEAK/Fn14 activation enhanced the migration and cytokine production of both DMECs and dermal fibroblasts. TWEAK also facilitates the expression of α-SMA and palladin in dermal fibroblasts. Furthermore, transfection of Fn14 siRNA abrogated such promotion effect of TWEAK on these cells.

Conclusions: TWEAK/Fn14 signals mediate the healing of burn wounds, possibly involving TWEAK regulation of the function of resident cells.