WOUND HEALING

# POLYI:C ACTIVATING TLR3 PROMOTES WOUND HEALING IN MURINE SKIN THROUGH INCREASED COLLAGEN I, α-SMA INVOLVING TGF-β/SMAD3

*Li Wang* [1] - *Jianmin Luo* [2]

*Shenzhen University General Hospital, Shenzhen University General Hospital/shenzhen University/dermatology, Sehnzhen, China* [1] - *Shantou University Medicla College, Shantou University Medical College/ Pathophysiology, Shantou, China* [2]

Introduction: Wound healing is a dynamic and highly coordinated series by regulating cytokines and signaling pathways. Innate immune responses are triggered after stimulation toll like receptors (TLRs) by their consanguine ligands; previous studies indicate that such interactions can facilitate wound healing. The detail mechanism need to be uncovered.

Objective: the work is to examine the effect of TLR3 agonist polyriboinosinic-polyribocytidylic acid (polyI:C) on mouse by compared wild and TLR3-deficient mice.

Materials and Methods: Histopathological analyses, RT-PCR, Western blot analysis were used to detect collagen I, α-SMA, TGF-β, Smad3 levels of RNA and protein in wild and TLR3 KO mice wound by polyI:C stimulating.

Results: polyI:C accelerated wound closure. PolyI:C application topically upregulated of collagen I, α-SMA, TGF-β expression and increased Smad3 phosphorylation. While polyI:C absent these effects on TLR3-deficient mice.

Conclusions: We suppose that polyI:C facilitates wound healing through increased collagen I, α-SMA involving TGF-β/Smad3 pathway. Stimulation via TLR3 performs a novel strategy to promote wound healing.