The effect of TRPV1 gene ablation on wound healing of corneal stroma in mice

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Purpose: We determined in mice if loss of transient receptor potential vanilloid 1 (TRPV1) affects the wound healing in corneal stroma in response to an incision-injury in mice.

Methods: TRPV1-null (KO) mice or C57BL/6 wild-type (WT) mice were used. A full-thickness penetrating incision injury (limbus-to-limbus) was produced in a central cornea by using a micro-surgical blade. Healing was evaluated at day 3, day 5 and day 10. The eyes were processed for histology, immunohistochemistry or real-time RT-PCR for expression of wound healing-related components.

Results: Primary healing (wound closure) of incision injury in corneal stroma was delayed in a KO mouse as compared with a WT mouse at day 5. Immunohistochemical examination showed less population of α-smooth muscle actin (αSMA)-labeled myofibroblasts in KO mice. Fibroblasts were populated in the anterior stroma of a WT cornea, while the cells were mainly observed in the posterior stroma of a KO cornea. The loss of TRPV1 also suppressed upregulation of mRNA expression of αSMA and collagen I at day 3.

Conclusion: Loss of TRPV1 impaired primary wound healing in corneal stroma in response to an incision-injury in mice. The mechanism of action might include suppression of fibrotic reaction and macrophage invasion in the injured tissue by lacking TRPV1. The distribution of fibroblasts depend on the distance from the wound healing position of the epithelium. In KO corneal stroma, fibroblast could be mostly appeared around the invaginated area of epithelium with delayed healing.

Key words: TRPV1, wound healing