

Elucidating of the Role of Reactive Oxidative Species (ROS) in TGF- β 1 Activation of TRPV1 in the Corneal Keratocyte

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Following stromal wounds, intrastromal cells transform into myofibroblasts. The ensuing fibrosis results in corneal opacity. Particularly in the cornea, TGF β -1 regulated fibrosis may impair vision by compromising the transparency of the cornea. Globally neutralizing TGF β blocks the fibroblast to myofibroblast conversion but also prevents cell repopulation and wound closure. Thus, innovative approaches that directly target the FTMT process in ways that allow its modulation are required. The specific aim of the experiment was to identify the location, nuclear, cytosolic-mitochondrial, nature, NOX subtype(s) involved and time course of the initial pSMAD2-mediated ROS bursts and establish whether further ROS production occurs as p38 is activated and whether the increase occurs by the same or different mechanisms by utilizing cell models similar to human keratocytes and to test the hypothesis that the activation of TRPV1 by ROS involves crosslinking of sulfhydryl groups on the TRPV1 polypeptide by hydrogen peroxide. By analyzing the mean peak fluorescence of nuchHyPer dye in 293T cells, it was determined that TGF- β 1 increased the oxidation and thus ROS generation within the cell over a timed course of 30 minutes, indicating that the ROS production in the human corneal fibroblast occurs as a p38 activated sequence.

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