Elucidating of the Role of Reactive Oxidative Secies (ROS) in TGF-β1 Activation of TRPV1 in the Corneal Keratocyte

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Following stromal wounds, instrasomal cells tranform in myofibroblasts. The ensuing fibrosis results in fibrosis. Particularly in the cornea, TGFβ-1 regulated fibrosis may impair vision by compromising the transparency of the cornea. Globally neutralizing TGFβ 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 allows its modulation are required. The specific aim of the experiment was to identify the location , nuclear, cytosolic-microsomal or mitochondrial, nature, NOX subtype(s) involved and time course of the initial pSMAD2-mediated ROS burs 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 involve crosslinking of sulfhydryl groups on the TRPV1 polypeptide by hydrogen peroxide. By analyzing the mean peak fluorescence of nucHyPer dye in 293T cells, it was determined that TGF-B1 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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