

**THE INFLUENCE OF KERATINOCYTES ON MYOFIBROBLASTS
IN HYPERTROPHIC SCAR**

Ho Yun Chung¹, Tae Jung Kim¹, Eun Jung Oh², Hyun Ju Lim², Sang Woo Kim³

¹*Kyungpook National University, School of Medicine, Dept. of Plastic and Reconstructive Surgery, Daegu, Korea, Democratic People's Republic of,* ²*Kyungpook National University, Department of Advanced Organic Materials Science and Engineering, Daegu, Korea, Democratic People's Republic of,* ³*Pochon CHA University, Dept. of Plastic and Reconstructive Surgery, Seongnam, Korea, Democratic People's Republic of*

Aim: During wound healing, myofibroblasts play a central role in matrix formation and wound contraction. At the end of healing, there is evidence that myofibroblasts disappear via apoptosis. Hypertrophic scarring is a pathological condition that myofibroblasts persist in the tissue. It has been hypothesized that abnormalities in epidermal–dermal crosstalk explain this pathology. To find out how myofibroblasts respond to epithelial stimuli, we characterized myofibroblasts in monolayer co-culture with keratinocytes.

Methods: For initial assessment, human myofibroblasts from hypertrophic scar tissue (Hmyo) and TGF- β induced myofibroblasts from normal dermal fibroblasts (Imyo) were characterized by microarray. And then human keratinocytes co-culture was applied into several different experimental groups. Each group was analyzed by immunohistochemistry, RT-PCR, and Real-time PCR.

Results: On microarray, numerous extracellular matrix- and smooth muscle cell associated gene were up-regulated in Hmyo and Imyo respectively, suggesting Hmyo are fully differentiated. Decreased collagen type 1(COL1A1) gene expression was shown in keratinocytes co-cultured Imyo and Hmyo. Smooth muscle actin (SMA) gene expression in Imyo, which was not affected by exogenous TGF- β , increased by keratinocytes co-culture. SMA gene expression in Hmyo increased by TGF- β and decreased in keratinocytes co-cultured Hmyo.

Conclusion: These observations strongly suggest that keratinocytes play a role in the development of pathological fibrosis in hypertrophic scar by influencing the behaviour of dermal fibroblasts or myofibroblasts. It is expected that this analysis would provide the basis of hypertrophic scar pathophysiology and new therapeutic approaches.