Silencing keloid by modulating mechanotransduction

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Although the details of keloid and hypertrophic scar pathogenesis remain poorly understood, medical practitioners have empirically recognized that the formation of these scars is promoted by mechanical stress on the healing wound. To prevent this stress, taping or compressive materials are used but their effects are limited. There has been a great increase recently in research on mechanotransduction, which is the process by which cells sense and respond to mechanical stimuli by converting them to biochemical signals. In particular, the mechanotransduction-related activities of multiple cell types that accumulate in pathological scars (*e.g.* fibroblasts, myocytes, and endothelial cells) are being studied with the aim of identifying mechanotransduction targets that could be used to treat keloids and hypertrophic scars. To facilitate this promising and novel approach, we have developed a mechanical stress mouse model and an *ex vivo* co-culture model. These models can be used to assess the effect of mechanical stress on the global gene expression profile of the wound: this analysis will indicate the signaling molecules that are up- or downregulated by mechanical stress and that could be good candidates for keloid treatment. Here, we present the current results of our studies and discuss the prospects of these studies in the future.