Possible effects of miR29a downregulation on keloid scar formation

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Abnormal wound healing can cause keloid scar formation. Keloids are fibrous benign tumours that continue growing beyond the wound boundaries. It is characterized by increased collagen accumulation. Therefore, targeting collagen synthesis may be the right approach for treatment. Evidence is emerging regarding the efficacy of the miR29 family in fibrotic diseases and extracellular matrix proteins are dominant among target molecules. In this study, we examined the role of miR29 family in keloids, HSP47 and LOX for collagen maturation and collagen synthesis through the TGF-b/Smad pathway. We determined the skin keloid and control tissue miR29 family gene levels via qPCR. After determining miR29 expressions in keloid scar tissues, we inhibited miR29a in primary keloid fibroblasts. We checked the protein levels related to collagen synthesis by using western blotting. TGF-b/Smad pathway gene levels were determined by using qPCR. Extracellular LOX activity was measured with a fluorescent kit, and TIMP-1 protein levels were assessed with ELISA. As a result, it has been found a significant increase in fibronectin, COL1A and LOXL2 protein levels, and LOX activity. These parameters proved that miR29a affects the collagen synthesis process and increases collagen synthesis. On the other hand, the downregulation of miR29a in keloids increased TGF-b/Smad pathway activity. TIMP-1 gene levels upregulated with this activity, but this was not reflected in the extracellular TIMP-1 protein level. This may indicate that miR29a affects TGF-b, TGF-b stimulates TIMP-1, but this stimulation may not be reflected in the protein level of TIMP-1 and may be a cause of the irregularity in keloid histopathology. These findings indicate that miR29a is effective in keloid formation and this activity is related to the expression level of miR29a.