Synopsis of Original Research Paper

Regulation mechanism of collagen XVII: elucidation by CRISPR/Cas9-mediated genome-wide screening

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Collagen XVII (COL17) is one of the major components forming hemidesmosomes in basal keratinocytes, which functions adhesion between the epidermis and the dermis. Loss of function of COL17 due to gene mutations coding *COL17A1* or autoimmunity to COL17 both read to blistering diseases including epidermolysis bullosa and bullous pemphigoid. In addition, COL17 is known to play important roles for keeping stemness in keratinocytes. Therefore, identification of genes regulating COL17 expression must be helpful for developing new therapies for blistering diseases and elucidating regulation mechanisms of keratinocyte stemness. In this study, we tried to identify key genes upregulating COL17 expression by CRISPR/Cas9-mediated genome-wide screening. First, as a positive control study, collagen VII (COL7) promoter was introduced upstream of the destabilized GFP (dscGFP) gene in HaCaT immortalized keratinocytes. This method was able to visualize COL7-related GFP expression in HaCaT keratinocytes. However, COL17 gene expression was not high enough for performing FACS-sorting when COL17 promoter was introduced upstream of the dscGFP gene in HaCaT keratinocytes. Therefore, we are analyzing HaCaT keratinocytes in which COL17 promoter was introduced upstream of the hygromycin resistant gene.