Type XVII collagen suppresses interfollicular epidermal proliferation in neonatal and aged skin, and helps rejuvenate epidermis

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Introduction

Type XVII collagen (COL17) is one of the hemidesmosomal proteins located at basement membrane zone. COL17 deficiency in human and mice shows premature aged phenotype in hair (Tanimura S, et al., Cell Stem Cell 2011; Matsumura et al., Science 2016) although the role of COL17 in regulating stemness and aging in interfollicular epidermis (IFE) has not been fully elucidated. In this study, we aimed to reveal how COL17 engages in the epidermal maintenance in IFE.

COL17 deletion induces transient epidermal proliferation in IFE via Wnt-β-catenin signaling

COL17 deficiency leads to epidermal proliferation in neonate

COL17 stabilizes Wnt signaling *in vitro* and *in vivo*

Induction of human COL17 to Col17a1-/- epidermis reverses the phenotype

Physical aging alters membranous COL17 distribution, which affects epidermal proliferation

Aging abolishes membranous COL17 and leads to IFE proliferation

Inhibition of atypical PKC alters COL17 distribution, simulating aged IFE

Overexpression of human COL17 restores the phenotype in aged epidermis

Discussion/Conclusion

These findings demonstrates that COL17 maintains IFE proliferation of neonatal and aged skin

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