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HAIR DISORDERS

FIBROBLAST GROWTH FACTOR 20 STIMULATES THE GROWTH AND CYCLE TRANSITION OF MOUSE HAIR FOLLICLES

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Introduction: Recently, the important role of fibroblast growth factors (FGFs) in development, diabetes, tumorigenesis and metastasis has been paid more attention.

Objective: To detect the effects of FGF20 on hair growth and cycle transition, we set up hair follicle organ culture model and intradermal injection of FGF20 animal model.

Material and Methods: We construct a mouse vibrissal follicle organ culture model. The mouse vibrissal follicles were cultured in different concentration of FGF20. Sixty female 60-day-old C57BL/6 mice were randomly divided into 2 groups, the control group and the FGF20 group. Flow cytometry, PCR Array of Wnt signaling pathway, immunohistochemistry staining, immunofluorescence staining, qPCR, and Western Blot were performed to detect the regulation of FGF20 on the follicle cycle transition.

Results: In a certain range, the growth rate of the cultured vibrissal follicles increased with the increase of FGF20 concentration, and the growth of the FGF20 100ng/ml group was significantly increased. Ki67 immunofluorescence staining showed Ki67+ matrix cells in the FGF20 100ng/ml treated group was significantly higher. The mice in the FGF20 treated group entered anagen phase since 6 days post-treatment, which was significantly earlier than that of the control group. Flow cytometry showed that the proportion and proliferation of CD34+CD49f+ hair follicle stem cells in the FGF20 treatment group was significantly higher. PCR Array test identified that the expression level of Wnt5a, Fzd4, Lrp5, Lef1, and Tcf1 was up-regulated in the FGF20 treated group.

Conclusions: FGF20 can promote the growth of vibrissal follicles through upregulating the proliferation of hair matrix cells. FGF20 can promote the telogen-to-anagen transition of hair follicle in mice. FGF20 regulates the cycle of hair follicle by promoting the proliferation and differentiation of hair follicle stem cells. FGF20 up-regulated Wnt5a to activate the Wnt canonical signaling pathway and then regulate the cycle of hair follicles.





