



HAIR DISORDERS

EFFICACY OF TOPICAL TOFACITINIB IN PROMOTING HAIR GROWTH IN NON-SCARRING ALOPECIA

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Introduction. Tofacitinib is a janus kinase 3 (JAK3) inhibitor that promotes hair growth; however, the efficacy and mechanism of this effect are not yet understood.

Objectives. This study aimed to evaluate the efficacy and mechanism of topical tofacitinib on hair growth in mice.

Materials and Methods. Eight-week-old male C57BL/6 mice were divided equally into four groups and treated topically with tofacitinib, minoxidil, or vehicle once daily for 21 days. Weekly photographs were taken to determine the area and rate of hair growth, and tissue samples were collected for histopathological evaluation. mRNA and protein expression of anagen-maintaining growth factors, including vascular endothelial growth factor (VEGF), insulin-like growth factor-1 (IGF-1), bone morphogenetic protein 4 (BMP4) and noggin were determined via RT-PCR and ELISA, respectively.

Results. Tofacitinib-treated mice exhibited more hair regrowth than either minoxidil-treated or control mice did between days 7 and 21 ($P < 0.05$). Topical tofacitinib also promoted more rapid hair growth rate than topical minoxidil or control did ($P < 0.001$). Histopathology showed a distinct increase in the number of hair follicles, mostly in the anagen phase, in the tofacitinib-treated group. Hair follicles in the minoxidil- and vehicle-treated groups were more often classified as catagen and anagen. Moreover, mice treated topically with tofacitinib showed an increased length of hair infundibulum and a reduced interfollicular epidermal thickness. VEGF mRNA and protein expression in the tofacitinib-treated group was significantly greater than those in the other groups ($P < 0.05$). IGF-1 mRNA expression was not upregulated in tofacitinib-treated mice. The expression of noggin and BMP4 mRNAs were significantly higher in the tofacitinib-treated group than in the minoxidil-treated





group.

Conclusion. Topical tofacitinib is effective in promoting hair growth, and the possible mechanism involves increased VEGF and noggin levels. This study will help develop a new therapeutic option for non-scarring alopecia.

