J Bioequiv Availab 2018, Volume 10
DOI: 10.4172/0975-0851-C2-037

Icotinib inhibits EGFR signaling and alleviates psoriasis-like symptoms in animal models

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To investigate the effects of icotinib hydrochloride and a derivative cream on epidermal growth factor receptor (EGFR) signaling and within animal psoriasis models, respectively. The effect of icotinib on EGFR signaling was examined in HaCaT cells, while its effect on angiogenesis was tested in chick embryo chorioallantoic membranes (CAM). The effectiveness of icotinib in treating psoriasis was tested in three psoriasis models, including diethylstilbestrol-treated mouse vaginal epithelial cells, mouse tail granular cell layer formation, and propranolol-induced psoriasis-like features in guinea pig ear skin. Icotinib treatment blocked EGFR signaling and reduced HaCaT cell viability as well as suppressed CAM angiogenesis. Topical application of icotinib ameliorated psoriasis-like histological characteristics in mouse and guinea pig psoriasis models. Icotinib also significantly inhibited mouse vaginal epithelium mitosis, promoted mouse tail squamous epidermal granular layer formation, and reduced the thickness of the horny layer in propranolol treated auricular dorsal surface of guinea pig. We conclude that icotinib can effectively inhibit psoriasis in animal models. Future clinical studies should be conducted to explore the therapeutic effects of icotinb in humans.

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