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IDO expressing fibroblasts suppress the development of imiquimod-induced psoriasis-like dermatitis

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Psoriasis is a common inflammatory disease of human skin characterized by raised cutaneous plaques with scaling and variable erythema. It is believed that psoriasis is triggered by specific stimuli, such as trauma or bacterial infections, which in turn induce resident dendritic cells (DC), macrophages and Th17 cells to produce cytokines that initiate a cascade of events. Recently it was shown that expression of cytokines by dendritic cells increase in the Psoriasis lesion and this induces the expression of IL-17 by $\gamma\delta^+$Tcell and Th17. We have previously shown that an immuno-modulatory enzyme, indole-amine 2, 3-dioxygenase (IDO) in dermal fibroblasts generates a tryptophan-deficient environment that selectively inhibits proliferation and induces apoptosis in $CD4^+$ T cells. Because of that we hypothesized that IDO-expressing fibroblasts can serve as source of the local immunosuppression treatment for Psoriasis. Injected IDO-expressing mice showed significantly reduced dermal thickness and erythema and scaling score as compared to untreated group. Analysis of immune cell infiltration in skin showed a significant reduction in the frequency ofneutrophils, $CD4^+IL\text{-}17^+$ Tcells and $\gamma\delta^+$ $IL\text{-}17^+$ T cell. This finding suggests that IDO-expressing fibroblasts might be a potential immune suppressive treatment for Psoriasis.

Biography

Sanam Salimi Elizei is a PhD student in the Experimental Medicine program of University of British Columbia, under the supervision of Dr. Aziz Ghahary since September 2011. He has examined the immune-regulatory effects of IDO expressing fibroblasts on pathogenesis of Psoriasis, an immune-mediated cutaneous disease.

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