PSORIASIS

ASTILBIN AMELIORATES IMIQUIMOD-INDUCED PSORIASIS-LIKE SKIN LESIONS IN MICE BY INHIBITING INFLAMMATORY RESPONSES MEDIATED BY IL-17A-PRODUCING T CELLS

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Background: The flavonoid astilbin is the major active component extracted from the rhizome of Smilax glabra, which has been widely used in China to treat inflammatory and autoimmune diseases, Psoriasis is a common chronic inflammatory disease in which IL-17 and related cytofactors play an important role, provoking inflammation.

Objective: We aim to determine the immunoregulatory effects and the underlying mechanisms of Astilbin in psoriasis-related inflammatory responses.

Materials and Methods: We employed an imiquimod (IMQ)-induced psoriasis-like mouse model to investigate the effect of astilbin in inflammation. Mice were administered 25 to 50 mg/kg astilbin. Inflammation of psoriasis-like lesions was assessed by histology, circulating levels of T cells were assessed by flow cytometry and cytokines by bead-based immunoassay. Jak/Stat3 in isolated T cells was assessed by Western blotting and RORγt expression was assessed by RT-PCR.

Results: Administration of astilbin ameliorated IMQ-induced keratinocyte proliferation, infiltration of CD3+ cells to psoriatic lesions and ameliorated elevations in circulating CD4+ and CD8+ T cells and inflammatory cytokines (IL-17A, TNF-α, IL-6, IFN-γ and IL-2). In vitro, astilbin inhibited Th17 cell differentiation and IL-17 secretion of isolated T cells, and inhibited Jak/Stat3 signaling in Th17 cells, while up-regulating Stat3 inhibitor SCOSE3 expression in psoriatic lesions.

Conclusions: Astilbin likely alleviates psoriasis-like skin lesions by inhibiting Th17 related inflammation. Astilbin represents as an interesting candidate drug for immunoregulation of psoriasis.