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Control of IL-23 Release in Dendritic Cells Protects Mice from Imiquimod-Induced Psoriasis

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Abstract : Psoriasis is a chronic inflammatory skin disease that affects about 2% of the world's population. IL-23 signaling plays a key role in the pathogenesis of psoriasis. Control of IL-23 release by small molecule compounds during developing psoriasis has not been well established. Here, we show that compound 1, a small molecule nature product, protected mice from imiquimod-induced psoriasis with improved skin lesions, reduced skin thickness, and reduced IL-23 mRNA expression in the skin tissue. FACS results showed compound 1 reduced the number of dendritic cells in the skin. Interestingly, compound 1 was not able to ameliorate IL-23-induced psoriasis-like skin inflammation in mice. Further, compound 1 inhibited MyD88-dependent IL-23 mRNA expression induced by LPS, CpG and imiquimod in BMDC cells, but not MyD88-independent CD80 and CD86 expression induced by LPS. The methods included real-time PCR, western blot, H & E staining, FACS and ELISA et al. In conclusion, compound 1 regulates MyD88-dependent signaling to control IL-23 release in dendritic cells, which improves imiquimod-induced psoriasis.

Keywords : dendritic cells, IL-23, toll-like receptor signaling, psoriasis
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