**IP-10 exacerbates the symptoms of mite induced-atopic dermatitis by prolonged Th2 response**

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Interferon-γ-inducible protein 10 (IP-10, also known as CXCL10) is a chemokine modulating the innate and adaptive immune response by recruiting immune cells such as neutrophils, mast cells, and T lymphocytes in inflammation site. This study aimed to reveal the role of IP-10 in atopic dermatitis (AD), one of the dominant type 2 immune diseases, and its regulation mechanism. The analysis using AD mouse models and the skin tissues of AD patients showing different house dust mite (HDM)-specific immunoglobulin (Ig)E levels revealed the close relationship of IP-10 with the disease severity. The functional study of IP-10 was performed using 2,4-dinitrochlorobenzene (DNCB)/*Dermatophagoides farinae* extract (DFE) induced-AD phenotypes comparing with that in IP-10 knock-out (KO) mice. The representative clinical phenotypes of AD and Th2 immune responses were remarkably reduced in IP-10 KO mice. On the contrary, subcutaneous injection of IP-10 reinforced those reduction. How the typical Th1 chemokine (IP-10) was involved in typical Th2 disease (AD)? Interestingly, we found that IP-10 not only promotes the secretion of IL-4 from Th2 cells, but also promotes the priming of CD4+ T cells into IL-4 secreting cells. Of the various cells capable of producing IP-10, only keratinocytes increased IP-10 production by DFE stimulation. IP-10 production in keratinocytes is regulated by co-activation of TLR3 and 4, under the mediation of two transcription factors, NF-κB and IRF3. Collectively, IP-10 acts as an important mediator for the AD-like cutaneous inflammation by acting as a stimulator of Th2 cells. Our finding suggests that IP-10 could be a new therapeutic target of AD and can be a useful selective option in combination therapy with conventional drug or selection for an alternative drug for the treatment of severe AD.