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IL-6 influences the balance between M1 and M2 macrophages in a mouse model of irritant contact dermatitis

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Abstract

Irritant contact dermatitis (ICD) is an acute inflammatory response that is ranked the 2nd most prevalent occupational injury associated with workman's compensation. Interleukin-6 (IL-6), a pro-inflammatory cytokine produced in the skin, is closely associated with healing and decreased levels are associated with more severe ICD. The phenotypic nature of the macrophage infiltrate during skin inflammation can greatly influence damage and healing during ICD. The pro-inflammatory, classically activated (M1) macrophage produce cytokines such as IL-6, IL-1, and TNFα, while alternatively activated (M2) macrophages produce IL-10 and TGFβ, and are thought to be associated with tissue repair. IL-6 may play a regulatory role in mediating the M1 versus M2 phenotypic change in skin. To examine the role of IL-6 in ICD, IL-6KO and C57 mice were exposed to benzoalkonium chloride (BKC) for 7 days. IL-6KO mice displayed decreased expression of CD206 (M2) as compared to CD86 (M1) and the ration of CD86:CD206 was significantly increased in IL-6KO mice compared to control. Multiplex protein analysis showed expression of multiple M1-related cytokines such IFN-g, CCL3, CCL4, CCL5, and CCL7 significantly modulated in IL6-KO BKC treated mice compared to C57.

Additionally, M2 related cytokine/chemokine receptors IL-4R and CCR1 were significantly dysregulated in IL-6KO BKC treated mice. IL-6 treatment of RAW cells enhanced the expression of CD206 in manner similar to IL-4. Overall, these results further confirm a role for IL-6 in macrophage polarization and could eventually result in the development of therapies for dermatitis based on this cytokines activity.

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