Abstract #46

IMMUNE PHENOTYPE AND INTERLEUKIN-6 DEFICIENCY INFLUENCE THE CYTOKINE PROFILE AND INFLAMMATORY CELL PRESENCE IN MURINE SKIN IN RESPONSE TO IRRITANT CONTACT DERMATITIS

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Irritant contact dermatitis (ICD) is an acute inflammatory response induced by exposure to topical irritants, and is labeled as the most common occupational skin disease by the Centers for Disease Control and Prevention. Currently, identifying potential therapeutic targets and predicting an individual's response to irritant exposure is hindered by the interpatient variability associated with the manifestation and severity of ICD. However, immune phenotype, such as Th1- and Th2-domiance, is postulated to contribute to this variability. In addition, interleukin 6 (IL-6), which displays both Th1 and Th2 properties, has previously been shown to play a role in the severity of ICD. C57BL/6J ("Th1 dominant"), Balb/c ("Th2 dominant"), and IL-6KO mice were utilized to investigate ICD induced by exposure to the occupational irritants, benzoalkonium chloride (BKC) and JP-8 jet fuel. Histopathology revealed epidermal thickening and dermal cellular infiltration in dermatitis lesions, with IL-6KO skin exhibiting the most severe damage. Analysis of skin cytokine and chemokine protein expression showed elevated levels of IL-12 and IL-15 in C57 mice exposed to irritants as compared to Balb/c mice. Interestingly, flow cytometric analysis also revealed greater infiltration of $CD11b^+$, $Ly6G^+$ cells in dermatitis lesions from C57 mice. However, Balb/c mice expressed more Th2-associated cytokines following irritant exposure than C57 mice. In response to BKC, IL-6KO mice exhibited elevated IL-1α expression compared to C57 mice. Also, JP-8 exposure in IL-6KO mice resulted in down-regulation of IL-18 and TGF- β as well as decreased $CD11b^+$, $Ly6G^+$ cellular presence in comparison to C57 mice. Overall, it appears that immune phenotype and IL-6 deficiency alter the skin inflammatory response in ICD via differential cytokine and chemokine expression which influences the type of inflammatory cells that infiltrate into the skin. These findings provide a better understanding of the pathophysiology of ICD and insight into potential targets for therapeutic immunomodulation.

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