The Journal of Immunology

# Immune phenotype and IL-6 deficiency modulate the inflammatory response in a mouse model of Irritant Contact Dermatitis

Kaitlin Calhoun, Lerin Luckett-Chastain and Randy Gallucci

J Immunol May 1, 2016, 196 (1 Supplement) 126.34;

## Abstract

Skin diseases are the second most common occupational illness with associated per annum costs exceeding $1 billion. Irritant contact dermatitis (ICD), the most common occupational skin disease, is characterized as an acute inflammatory response that manifests as a result of topical irritant exposure. Immune phenotype, such as Th1- and Th2- dominance, is postulated to contribute to the variability seen between patients. In addition, interleukin 6, which is known to have variably both Th1 and Th2 properties, has been shown to play a key role in 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 that IL-1β, TNF-α, TGF-β, CCL2, CCL3 and CXCL1, were elevated in mice exposed to BKC regardless of immune phenotype. In contrast, IL-1β, TNF-α, TGF-β, and CXCL1 varied between mouse strains when JP-8 was compared to control exposure. Flow cytometric analysis and

immunohistochemistry indicated neutrophil and monocyte presence within dermatitis lesions; however, IL-6KO showed a greater amount of neutrophils following BKC exposure and decreased neutrophil presence after JP-8 exposure when compared to C57 mice. Overall, it appears that immune phenotype and IL-6 deficiency alters the skin inflammatory response in ICD via differential cytokine and chemokine expression that influence the type of inflammatory cells that infiltrate into the skin.



Previous

Back to top

## In this issue

The Journal of Immunology
Vol. 196, Issue 1 Supplement
1 May 2016
Table of Contents