Regulation of NK cell function and Th17 responses by MAP kinase phosphatase 7

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Research Interest
Signaling transduction pathways that regulate innate and adaptive immune responses, and how dysregulated signaling can lead to inflammatory and autoimmune diseases.

Abstract
MAP kinase signaling pathways are evolutionary conserved immune regulators, having critical roles in innate and adaptive immune responses. The biological outcome of MAP kinase activation is determined by the duration and magnitude of their activation. It has been shown that members of MAP kinase phosphatase (MKP) protein family are major negative regulators of MAP kinase signaling. By controlling the activation of MAP kinases, MKPs play critical roles in immune responses. The functions of several MKP members, including MKP1, MKP5, and Pac-1, in immune responses have been studied. However, the functions of most of the MKP members in regulation of MAP kinases and in immune responses are not clear. In this study, we found that the deficiency of one MKP member, MKP7, led to an impaired function of NK cells in innate immunity. MKP7 deficient mice are defective in anti-intracellular bacterial infection. In adaptive immunity, MKP7 deficient mice have reduced Th17 responses, which resulted in the resistance of the mice to MOG-induced EAE disease. This study demonstrate that MKP7 has specific function in immune responses to infection and in autoimmune diseases.