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Titel des Beitrags:

Decreased pathology and prolonged survival of human DC-SIGN transgenic mice during mycobacterial infection.

Abstract:

Dendritic cell (DC)-specific intercellular adhesion molecule-3 grabbing nonintegrin (DC-SIGN: CD209) is a C-type lectin that binds ICAM-2,3 and various pathogens such as HIV, helicobacter, and mycobacteria. It has been suggested that Mycobacterium tuberculosis, the causative agent of pulmonary tuberculosis, interacts with DC-SIGN to evade the immune system. To directly analyze the role of human DC-SIGN during mycobacterial infection, we generated conventional transgenic (tg) mice (termed "hSIGN") using CD209 cDNA under the control of the murine CD11c promoter. Upon mycobacterial infection, DCs from hSIGN mice produced significantly less IL-12p40 and no significant differences were be observed in the secretion levels of IL-10 relative to control DCs. After high dose aerosol infection with the strain M. tuberculosis H37Rv, hSIGN mice showed massive accumulation of DC-SIGN(+) cells in infected lungs, reduced tissue damage and prolonged survival. Based on our in vivo data, we propose that instead of favoring theimmune evasion of mycobacteria, human DC-SIGN may have evolved as a pathogen receptor promoting protection by limiting tuberculosis-induced pathology.

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