
C. Schlapbach

Human T\textsubscript{h}9 Cells: A subset of skin-tropic T\textsubscript{h} cells with autocrine and paracrine proinflammatory capacity

T helper type 9 (T\textsubscript{h}9) cells can mediate tumor immunity and participate in autoimmune and allergic inflammation in mice, but little is known about the T\textsubscript{h}9 cells that develop in vivo in humans. We isolated T cells from human blood and tissues and found that most memory T\textsubscript{h}9 cells were skin-tropic or skin-resident. Human T\textsubscript{h}9 cells co-expressed tumor necrosis factor-alpha and granzyme B and lacked coproduction of T\textsubscript{h}1/T\textsubscript{h}2/T\textsubscript{h}17 cytokines, and many were specific for Candida albicans. Interleukin-9 (IL-9) production was transient and preceded the up-regulation of other inflammatory cytokines. Blocking studies demonstrated that IL-9 was required for maximal production of interferon-gamma, IL-9, IL-13, and IL-17 by skin-tropic T cells. IL-9–producing T cells were increased in the skin lesions of both atopic dermatitis and psoriasis, suggesting that these cells may contribute to human inflammatory skin disease. Our results indicate that IL-9 is produced transiently by a discrete T cell subset and that these cells are tropic for the skin. Although these IL-9 producing T\textsubscript{h} cells may function normally to protect against extracellular pathogens, aberrant activation of these cells may contribute to inflammatory diseases of the skin.