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Titel des Beitrags: IL-17 amplifies human contact hypersensitivity by licensing hapten nonspecific Th1 cells to kill autologous keratinocytes.

Abstract: Th17 is a newly identified lineage of effector T cells involved in autoimmunity and immune responses to pathogens. We demonstrate in this study the pathogenic role of IL-17-producing CD4(+) T lymphocytes in allergic contact dermatitis (ACD) to skin-applied chemicals. IL-17(+) T cells infiltrate ACD reactions and predominantly distribute at the site of heavy spongiosis. Skin IL-17(+) T cells were functionally and phenotypically heterogeneous: although pure Th1 prevailed in ACD skin, hapten responsiveness was restricted to Th1/IL-17 (IFN-gamma(+)IL-17(+)) and Th0/IL-17 (IFN-gamma(+)IL-17(+))IL-4(+) fractions, and to lesser extent Th2/IL-17 cells. In the IFN-gamma-dominated ACD environment, IL-17-releasing T cells affect immune function of keratinocytes by promoting CXCL8, IL-6, and HBD-2 production. In addition, compared with Th1, supernatants from Th1/IL-17 T cells were much more efficient in inducing ICAM-1 expression on keratinocytes and keratinocyte-T cell adhesiveness in vitro. As a consequence, exposure to combined IFN-gamma and IL-17 rendered keratinocytes susceptible to ICAM-1-dependent Ag nonspecific T cell killing. Thus, IL-17 efficiently amplifies the allergic reaction by rendering virtually all of the
T lymphocytes recruited at the site of skin inflammation capable to directly contribute to tissue damage.