

## Autoaggressive effector cells in the immune privileged eye: Th17 or Th1?

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Experimental autoimmune uveitis (EAU) in animals serves as a model of human uveitis. EAU can be induced in mice by immunization with the retinal antigen IRBP in complete Freund's adjuvant (CFA) or by IRBP-pulsed *in vitro* matured dendritic cells. Experiments using these two alternative models of EAU as well as *in vitro* polarized retina-specific Th1 and Th17 cells, combined with genetic or antibody-induced deficiency of the reciprocal effector phenotype, demonstrated that disease can be driven either by a Th17 or by a Th1 effector response. The dominant effector response is at least in part affected by conditions present during the exposure to antigen, including the quality/quantity of innate receptor stimulation and/or type of antigen-presenting cells. This notion is supported by a shift in Th1/Th17 polarization *in vitro* as a result of Ag presentation by dendritic cells exposed to different maturation stimuli. Early IL-17 and IFN- $\gamma$  production by innate immune cells such as NKT may also affect the disease process. These data shed light on effector responses in autoimmune disease and provide balance to the prevailing Th1/Th17 paradigm. These findings may also help to explain the clinical heterogeneity of human uveitis observed in patients who respond to the same retinal antigen(s).

### References:

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