IN BRIEF

PARASITE IMMUNITY

Dynamic imaging of T cell–parasite interactions in the brains of mice chronically infected with *Toxoplasma gondii*

Schaeffer, M. *et al. J. Immunol.* **182**, 6379–6393 (2009)

Robey and colleagues used *Toxoplasma gondii* infection of mice as a model system to directly visualize the interactions between a pathogen and the adaptive immune system *in vivo*. During chronic infection, the intracellular parasite *T. gondii* forms cysts in the brain that are controlled by an ongoing $CD8^+$ T cell response. Parasite-specific $CD8^+$ T cells in the brains of infected mice were found to accumulate and arrest near cells containing individual parasites but not near parasite-containing cysts, suggesting that parasite antigens were not presented by cyst-bearing host cells. The individual parasites were often found in aggregates of $CD11b^+$ myeloid cells that resembled granulomas. The parasite-specific T cells formed brief contacts with antigen-presenting cells in the $CD11b^+$ aggregates, only some of which contained intact parasites, which suggests that $CD11b^+$ cells in the brain might cross-present parasite antigens. These data provide new insight into the immune response to persistent pathogens in the brain.

AUTOIMMUNITY

The level of B7 homologue 1 expression on brain DC is decisive for CD8 Treg cell recruitment into the CNS during EAE

Zozulya, A. L. *et al. Eur. J. Immunol.* 7 May 2009 (doi:10.1002/eji.200839165)

The frequency, distribution and phenotype of dendritic cells (DCs) in the brain are rate-limiting factors for the induction and effector phase of experimental autoimmune encephalomyelitis (EAE). This study looked at the effect on EAE severity of DCs deficient for the inhibitory molecule B7-H1 (also known as PDL1), which has an important role in regulating T cell activation and tolerance. Intracerebral injection of B7-H1-deficient DCs presenting myelin oligodendrocyte glycoprotein (MOG) resulted in a delay in EAE onset and decreased peak disease score compared with injection of wild-type DCs presenting MOG. This effect correlated with increased proliferation and recruitment of $CD8^+CD122^+$ regulatory T cells to the brain. Understanding the functions of DCs in maintaining the balance between encephalitogenic and regulatory T cells in the brain could lead to the development of new therapies harnessing the tolerogenic properties of brain DCs.

LYMPHOCYTE MIGRATION

Cutting edge: natalizumab blocks adhesion but not initial contact of human T cells to the blood–brain barrier *in vivo* in an animal model of multiple sclerosis

Coisne, C. *et al. J. Immunol.* **182**, 5909–5913 (2009)

The α4 integrin-specific antibody natalizumab has been used in clinical trials to reduce disease severity in patients with multiple sclerosis. It was presumed that this antibody works by blocking the α4 integrin-mediated extravasation of inflammatory cells into the central nervous system. However, given the broader functions of α4 integrin it is possible that this antibody functions through other mechanisms. Using intravital fluorescence video microscopy this study provides the first direct *in vivo* evidence that natalizumab specifically blocks the firm adhesion of human T cells to the microvasculature of the spinal cord of mice with experimental autoimmune encephalomyelitis. However, T cell rolling and capture (the abrupt stop of T cells on the vessel wall for up to 7 seconds) was unaffected.