IN BRIEF

B CELL RESPONSES

Self control key for maturation

Affinity maturation of B cell receptors (BCRs) is an important component of germinal centre (GC) responses. Direct competition between GC B cells for antigen is thought to select for high-affinity clones. However, this does not explain how BCR affinities can increase over the course of an infection, when large amounts of antigen are produced for prolonged periods of time. This study proposes a new model for affinity maturation, whereby GC B cells compete with their own secreted antibodies for access to antigen. Experiments with monoclonal antibodies of defined affinities provided support for the authors' model, which explains how a directional selection pressure can be maintained in the GC. Notably, this model indicates that communication between distant GCs might be facilitated by antibodies, rather than by B cell migration, and can also explain how the GC response is terminated: eventually, the 'masking' of antigen by high affinity antibodies is too strong to allow for B cell survival.

ORIGINAL RESEARCH PAPER Zhang, Y. *et al.* Germinal center B cells govern their own fate via antibody feedback. *J. Exp. Med.* **210**, 457–464 (2013)

IMMUNOMETABOLISM

ILC2s maintain metabolic homeostasis

Eosinophils and alternatively activated macrophages in visceral adipose tissue (VAT) promote insulin sensitivity and metabolic homeostasis. Molofsky *et al.* now show that interleukin-5 (IL-5) deficiency promotes obesity and insulin resistance in mice on a high-fat diet. VAT-resident group 2 innate lymphoid cells (ILC2s) were the main source of IL-5 and IL-13, and were required for the maintenance of VAT eosinophils and alternatively activated macrophages in the steady state. Infection of mice with the intestinal helminth *Nippostrongylus brasiliensis* increases VAT eosinophil numbers and improves metabolic homeostasis in mice on a high-fat diet. This study shows that helminth infection activates VAT ILC2s to produce IL-5 and IL-13, and that loss of these cells results in defective accumulation of VAT eosinophils. So, VAT-resident ILC2s contribute to metabolic homeostasis by maintaining VAT eosinophils and alternatively activated macrophages.

ORIGINAL RESEARCH PAPER Molofsky, A. B. *et al.* Innate lymphoid type 2 cells sustain visceral adipose tissue eosinophils and alternatively activated macrophages. *J. Exp. Med.* **210**, 535–549 (2013)

ANTIVIRAL IMMUNITY

Visualizing the defensive lines

Intravital multiphoton microscopy was used to visualize the spatiotemporal organization of immune cells following acute epicutaneous vaccinia virus infection in mice. Vaccinia virus infected keratinocytes in epidermal foci and $GR1^{mid}LY6G^{-}$ inflammatory monocytes that surround the lesions. These monocytes were motile and produced infectious virus. Uninfected $LY6G^{+}$ cells also accumulated at these sites, penetrated the lesions and produced reactive oxygen and nitrogen species. By contrast, virus-specific $CD8^{+}$ T cells remained at the periphery, where they killed outlying infected inflammatory monocytes. Inhibition of peroxynitrite generation allowed $CD8^{+}$ T cells to enter the viral lesions, which modestly reduced viral titres. However, depletion of $CD8^{+}$ T cells and $LY6G^{+}$ cells greatly increased viral titres, which indicates that these cells cooperate to clear infection. Thus, clearance of viral skin infection requires a spatially coordinated and synergistic attack by innate and adaptive immune cells.

ORIGINAL RESEARCH PAPER Hickman, H. D. *et al.* Anatomically restricted synergistic antiviral activities of innate and adaptive immune cells in the skin. *Cell Host Microbe* **13**, 155–168 (2013)