

## IN BRIEF

**ANTIBODIES****Antibodies protect against intracellular bacteria by Fc receptor-mediated lysosomal targeting**

Joller, N. *et al. Proc. Natl Acad. Sci. USA* 3 Nov 2010 (doi:10.1073/pnas.1013827107)

This study describes a new mechanism by which antibodies can mediate protection against intracellular pathogens. Antibody engagement with Fc receptors on host cells was shown to render the cells non-permissive to infection with *Legionella pneumophila* by preventing the bacteria from forming the replication-permissive vacuoles that enable them to evade lysosomal degradation. Complement was not required for antibody-mediated protection against infection with *L. pneumophila*; instead, opsonized *L. pneumophila* were targeted to lysosomes as a result of Fc receptor triggering and hence were unable to replicate intracellularly. Activation of Fc receptors on macrophages before exposure to non-opsonized bacteria also resulted in the cells being non-permissive for bacterial replication, indicating that this is not a direct effect on the phagocytic pathway. Instead, the mechanism depends on the intracellular kinase signalling pathway downstream of the Fc receptor, but does not require *de novo* protein synthesis by host cells.

**T CELL RESPONSES****Macroautophagy regulates energy metabolism during effector T cell activation**

Hubbard, V. M. *et al. J. Immunol.* 8 Nov 2010 (doi:10.4049/jimmunol.1000576)

Macroautophagy is a form of autophagy in which cytosolic proteins and organelles are targeted for lysosomal degradation, and it has been shown to be important for preventing the accumulation of damaged proteins within a cell. However, it has been unclear whether this process contributes to the regulation of adaptive immune responses. Hubbard *et al.* have now shown that macroautophagy is induced in effector T cells and is necessary for full T cell activation. Macroautophagy-deficient T cells showed defects in proliferation and cytokine production after stimulation, but these functions could be restored by providing the cells with an exogenous energy source. Autophagosome formation differed between resting and activated T cells: the latter actively excluded mitochondria from their autophagosomal cargo, and this seems to be important for meeting the energy requirements of activated T cells.

**INNATE IMMUNITY****Regulation of cytokine secretion in human CD127<sup>+</sup> LTI-like innate lymphoid cells by Toll-like receptor 2**

Crellin, N. K. *et al. Immunity* 4 Nov 2010 (doi:10.1016/j.immuni.2010.10.012)

Among immunologists, there is a growing interest in populations of innate-like immune cells that can produce a range of effector cytokines but do not belong to any classical leukocyte lineage. Lymphoid-tissue inducer (LTI)-like cells are a subset of innate-like immune cells that have previously been shown to secrete lymphotoxin, tumour necrosis factor, interleukin-17 (IL-17) and IL-22 upon activation. This study shows that human LTI-like cells can also produce IL-2 and the T helper 2-type cytokines IL-5 and IL-13 following activation by Toll-like receptor 2 (TLR2) ligands and common cytokine receptor  $\gamma$ -chain cytokines. Individual clones of LTI-like cells showed unique cytokine-producing profiles and the authors suggest that, similarly to T helper cells, distinct functional subpopulations of innate-like immune cells may develop in response to environmental cues and contribute to both protective immunity and disease pathology.