IN BRIEF

INFLAMMATION

Neutrophil primary granule proteins HBP and HNP1–3 boost bacterial phagocytosis by human and murine macrophages.

Soehnlein, O. *et al. J. Clin. Invest.* 11 September 2008 (doi:10.1172/JCI35740)

This study identifies a new antimicrobial function of neutrophil secretory proteins through the stimulation of phagocytosis by macrophages. The authors show that heparin-binding protein (HBP) and human neutrophil peptides 1–3 (HNP1–HNP3) are released from neutrophil primary granules during inflammation and stimulate the production of tumour-necrosis factor and interferon-γ by adjacent macrophages. In turn, these cytokines act in an autocrine manner to increase macrophage expression of the Fcγ receptors CD32 and CD64. Increased expression of these receptors enhances the phagocytosis of IgG-opsonized bacteria by the macrophages. This mechanism adds to the known effects of other neutrophil products on macrophage maturation and migration. HBP was shown to signal through β_2-integrins on macrophages, but the receptor for HNP1–HNP3 is yet to be determined.

VIRAL IMMUNITY

Apobec3 encodes *Rfv3*, a gene influencing neutralizing antibody control of retrovirus infection.

Santiago, M. L. *et al. Science* **321**, 1343–1346 (2008)

Rfv3 (recovery from Friend virus 3), which has long been known to confer resistance to retroviral infection, has now been shown to be encoded by the deoxycytidine deaminase family member *Apobec3*. Compared with wild-type mice, *Apobec3*-deficient mice were more susceptible to death induced by infection with Friend virus owing to markedly reduced levels of neutralizing antibodies. In addition, the investigation of several different strains of mice showed that those with increased susceptibility to Friend virus expressed an alternatively spliced form of *Apobec3* mRNA that was thought to result in absent or truncated expression of the protein. Although the exact mechanisms by which *Apobec3* acts as a viral resistance factor and promotes the production of neutralizing antibodies are not yet known, further studies of this gene have the potential to uncover new strategies to fight retroviruses, including HIV, in humans.

NATURAL KILLER T CELLS

The transcription factor PLZF directs the effector program of the NKT cell lineage.

Savage, A. K. *et al. Immunity* 14 August 2008 (doi:10.1016/j.immuni.2008.07.011)

Using a microarray technique, the transcription factor PLZF (promyelocytic leukaemia zinc finger) was identified as a transcriptional signature of natural killer T (NKT) cells. This protein, which is related to the $CD4^+$ T-cell-specifying factor Th-POK, was expressed early during NKT-cell development and was required for the intrathymic proliferation and acquisition of effector functions of these cells. Other immune-cell lineages, with the exception of mucosal-associated invariant T (MAIT) cells, were not found to express PLZF. Mice deficient for PLZF had marked irregularities in the number and tissue localization of NKT cells compared with wild-type mice, whereas transgenic expression of PLZF in $CD4^+$ T cells induced an effector phenotype and homing pattern that resembled that of developing NKT cells. Therefore, PLZF is uniquely important for specifying the effector function of NKT cells and probably of the closely related MAIT-cell subset.