IN BRIEF

HAEMATOPOIESIS

Role for NLRP1a inflammasome unravelled

The ligands and function of the NOD-, LRR- and pyrin domain-containing 1 (NLRP1) inflammasome have remained largely obscure. Now, studies in mice with an activating mutation in the gene encoding NLRP1a (termed *Nlrp1a*$^{Q593P/Q593P}$ mice) have revealed that the NLRP1a inflammasome assembles in an ASC-independent manner to promote caspase 1-dependent production of interleukin-1β (IL-1β). *Nlrp1a*$^{Q593P/Q593P}$ mice have neutrophilia and lethal systemic inflammation, and IL-18 or T cells partially suppressed this NLRP1a inflammasome-mediated inflammation. By contrast, *Nlrp1a*$^{Q593P/Q593P}$ *Il1b*$^{-/-}$ mice showed no signs of inflammatory disease but had caspase 1-dependent defects in the number and function of haematopoietic progenitor cells. Strikingly, NLRP1a activation as a result of the chemoablation or viral infection of haematopoietic cells led to progenitor cell death by pyroptosis and thus impaired haematopoiesis.

ORIGINAL RESEARCH PAPER Masters, S. L. *et al.* NLRP1 inflammasome activation induces pyroptosis of hematopoietic progenitor cells. *Immunity* **37**, 1009–1023 (2012)

INNATE-LIKE LYMPHOCYTES

An antifungal role for innate lymphoid cells

This study describes an essential role for interleukin-17 (IL-17)-secreting innate lymphoid cells (ILCs) in promoting protective immunity to an oral fungal infection. In mice infected sublingually with *Candida albicans*, the adaptive immune system was shown to be dispensable for the resolution of infection. Both MHC class II-deficient mice and *Rag1*$^{-/-}$ mice could control this infection. Furthermore, mice lacking γδ T cells or natural killer T cells could also clear the infection. By contrast, *Rag1*$^{-/-}$ mice that were treated with CD90-specific antibodies to deplete ILCs could not recover following *C. albicans* infection. Depletion of ILCs led to a marked reduction in the early induction of IL-17A and IL-17F in the tongues of *C. albicans*-infected mice. The authors suggest that ILCs mediate protection by upregulating IL-17 in response to IL-23 that is generated early following infection with *C. albicans*.

ORIGINAL RESEARCH PAPER Gladiator, A. *et al.* Cutting edge: IL-17-secreting innate lymphoid cells are essential for host defense against fungal infection. *J. Immunol.* **190**, 521–525 (2013)

INNATE-LIKE LYMPHOCYTES

Diet shapes the natural killer cell response

Caloric restriction can have beneficial effects in laboratory animals, such as extending their lifespan and decreasing the severity of autoimmune diseases and the incidence of cancer, but it can also increase their susceptibility to infection. Several studies have suggested that these outcomes are linked to the effects of caloric restriction on the immune system. This study describes how caloric restriction affects natural killer (NK) cell responses. Caloric restriction in mice was shown to alter the distribution of NK cell subsets to the peripheral tissues, spleen and lymph nodes. Mice subjected to caloric restriction had fewer mature NK cells and increased frequencies of CD127^{+} NK cells. This resulted in the mice on the restricted diet having NK cells that were less responsive to cytokine-mediated activation, although their NK cells could still mediate cytotoxic activity against target cells. Such altered NK cell function following caloric restriction in mice may contribute to some of the immunological phenomena observed in these animals.

ORIGINAL RESEARCH PAPER Clinthorne, J. F. *et al.* NK cell maturation and function in C57BL/6 mice are altered by caloric restriction. *J. Immunol.* **190**, 712–722 (2013)