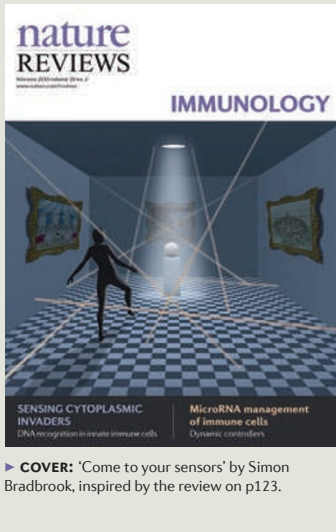




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Several articles this month look at the inputs to and outputs from innate immune cells that determine their instructive role in shaping adaptive immune responses.

The recognition of cytoplasmic DNA by innate immune cells is important for defence against viruses and many types of bacteria. On p123, Veit Hornung and Eicke Latz review the molecular mechanisms by which intracellular DNA can activate cells. This results in the transcriptional activation of pro-inflammatory genes, such as type I interferons and members of the interleukin-1 (IL-1) family, and the proteolytic activation of caspases, leading to the maturation of pro-cytokines such as pro-IL-1 $\beta$  and pro-IL-18.

John Sims and Dirk Smith (p89) discuss how this production of IL-1 $\beta$ , IL-18 and other members of the IL-1 family can amplify the innate immune response and link to adaptive immunity through the polarization of T helper (T<sub>H</sub>) cells. The main T<sub>H</sub> cell subsets are polarized by distinct members of the IL-1 family: IL-18 for T<sub>H</sub>1 cells, IL-33 for T<sub>H</sub>2 cells and IL-1 for T<sub>H</sub>17 cells.

IL-33 is the most recently identified member of the IL-1 family. As Foo Liew, Nick Pitman and Iain McInnes discuss (p103), IL-33 is expressed mainly by epithelial and endothelial cells and fibroblasts, indicating an important role in barrier defence and repair, and the IL-33 receptor is highly expressed by T<sub>H</sub>2 cells and mast cells, mediating protection against helminth infection and the pathogenesis of asthma and allergy.

Similarly, Toll-like receptor (TLR)-mediated recognition of the gut microbial flora also has a dual role in the repair of epithelial cell injury and protection from pathogens. Maria Abreu (p131) describes how TLR signalling in the gut for the proliferation of epithelial cells and recruitment of inflammatory cells can be usurped to support cancer development.

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