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Several articles this month relate to the theme of how our immune system is able to walk the fine line between fighting off infections and preventing bystander damage or autoimmune disease. In terms of promoting an effective immune response to infection, the newest player in this field is the T helper 17 ( $T_H17$ ) cell, which through the production of interleukin-17 (IL-17), IL-21 and IL-22 generally has a pro-inflammatory role. On page 337, Chen Dong discusses how the regulation of these cells differs from that of  $T_H1$  and  $T_H2$  cells and describes their unique gene-expression profile. The uncontrolled activation of  $T_H17$  cells is involved in autoimmune disease, so these cells and their products might be important therapeutic targets.

On the other side of the line to  $T_H17$  cells, three receptor protein tyrosine kinases of the TAM family — TYRO3, AXL and MER — prevent unrestrained pro-inflammatory signalling leading to chronic inflammation. As Greg Lemke and Carla Rothlin discuss on page 327, these TAM receptors are involved in inhibiting the inflammatory activation of dendritic cells and promoting clearance of the apoptotic debris that results from infection.

Charles Serhan and colleagues (page 349) also discuss how an inflammatory response can be terminated and resolved once it has carried out its function by an active process that involves lipid mediators such as the lipoxins, resolvins and protectins, which rapidly restore tissue homeostasis.

Finally, an interesting new concept in the regulation of autoimmune responses is proposed on page 391 in an Opinion article by Simon Fillatreau and colleagues. They put forward a model in which B cells produce the regulatory cytokine IL-10 in response to Toll-like-receptor ligands, such that the level of exposure to environmental microorganisms might determine the balance between pathogenic and protective B cells in autoimmunity.



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