FROM THE FDITORS











or goalkeepers playing in this month's World Cup, quick reactions are essential for stopping that ball from reaching the back of the net. Similarly, a rapid response by the innate immune system is crucial for protecting the body against incoming threats. To do so, the innate immune system has evolved a range of receptors and effector mechanisms that detect and respond quickly to tissue injury, cell stress or pathogens. One response — the production of interleukin-17 (IL-17) — has received much attention recently but mainly in terms of mature T cells. On page 479, Daniel Cua and Cristina Tato point out that many innate immune cell populations rapidly produce IL-17 on sensing stress, before T cells have had a chance to mature and gain this activity. This early production of IL-17 is thought to be crucial for protecting against pathogens and maintaining normal homeostasis of the body's tissues, particularly those that are exposed to the environment.

One of the main cell populations responsible for this quick-fire production of IL-17 is the $\gamma\delta$ T cell population. $\gamma\delta$ T cells are a unique T cell subset defined by their ability to recognize stress-induced molecules through the $y\delta$ T cell receptor and respond with innate cell-like kinetics. On page 467, Marc Bonneville and colleagues describe how the rapid effector functions of $\gamma\delta$ T cells are programmed during their development but can also be gained later according to the type of threat they face. This enables γδ T cells, by teaming up with 'players' of the innate and adaptive immune systems, to help establish appropriate responses to cell stress or pathogens. Agnès Lehuen and coauthors (page 501) explore such immune cell interplay further in the context of type 1 diabetes, describing how pathogens can influence these cellular interactions and therefore the onset of the disease.

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