IN BRIEF

INFLAMMATION

Interferon-λ2 limits neutrophil migration

The type III interferon (IFN) family comprises IFNλ1, IFNλ2 and IFNλ3, which are mainly known for their role in antiviral immunity. Now, Blazek *et al.* show that IFNλ2 has anti-inflammatory functions, and treatment with IFNλ2 halts and reverses the development of collagen-induced arthritis (CIA) in mice. After 4 days of IFNλ2 treatment — when progression of CIA was halted — the number of neutrophils was reduced in the joints of mice with CIA. This was associated with decreased levels of interleukin-1β, which is thought to be important for the amplification of CIA. Neutrophils expressed high levels of the IFNλ2 receptor, and the migration of these cells was restricted in response to IFNλ2. Interestingly, neutrophils are the most abundant immune cell in the synovial fluid of patients with rheumatoid arthritis. These results indicate that IFNλ2 or an IFNλ2 receptor agonist could be putative therapeutics for neutrophil-driven inflammation.

ORIGINAL RESEARCH PAPER Blazek, K. *et al.* IFN-λ resolves inflammation via suppression of neutrophil infiltration and IL-1β production. *J. Exp. Med.* http://dx.doi.org/10.1084/jem.20140995 (2015)

PARASITE IMMUNITY

Commensals regulate the schistosome response

To investigate the effect of repeated percutaneous exposure to schistosome larvae on immune regulation in the skin, Mountford and colleagues used a mouse model of skin infection with *Schistosoma mansoni*. Repeated infection resulted in interleukin-10 (IL-10) production by dermal $CD4^+$ T cells that do not express markers of classical regulatory T cells. In IL-10-deficient mice, neutrophil recruitment and T cell proliferation were increased, which suggests that the IL-10 response functions to limit inflammation and tissue damage. IL-10-producing dermal T cells were specific for both skin commensal bacteria and, later, schistosome antigens. The authors suggest that commensal bacteria gain access to the dermis during parasite penetration and elicit an early regulatory response. This could be a strategy used by the parasite to evade the host immune response.

ORIGINAL RESEARCH PAPER Sanin, D. E. *et al.* Helminth infection and commensal microbiota drive early IL-10 production in the skin by $CD4^+$ T cells that are functionally suppressive. *PLoS Pathog.* **11**, e1004841 (2015)

GENE REGULATION

Seasonal genes explain the winter blues

New research may explain why some inflammatory diseases are more common in the winter. According to Todd and colleagues, the activity of ~23% of human genes varies with the seasons. Genome-wide analyses of mRNA levels in peripheral blood mononuclear cells from European cohorts revealed a more inflammatory status in the winter compared with in the summer months. For example, genes involved in B cell receptor signalling and metabolic processes were more highly expressed in the winter, as were the pro-inflammatory factors soluble interleukin-6 receptor and C-reactive protein. In The Gambia in West Africa, the heightened inflammatory status correlated with the rainy season, from June to October, which is when infectious diseases peak in that region. So, seasonal gene regulation may provide readiness for better protection from infections, but it may also contribute to the higher frequencies of heart attacks, rheumatoid arthritis and type 1 diabetes that are observed in the winter months.

ORIGINAL RESEARCH PAPER Dopico, X. C. *et al.* Widespread seasonal gene expression reveals annual differences in human immunity and physiology. *Nature Commun.* **6**, 7000 (2015)