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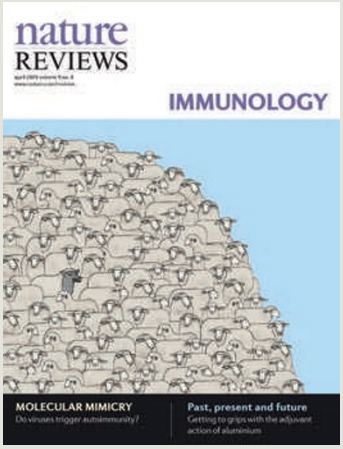

▶ **COVER:** 'A wolf in sheep's clothing' by Simon Bradbrook, inspired by the review on p246.


ELAINE BELL


LUCY BIRD


KIRSTY MINTON


OLIVE LEAVY

Viruses outsmart their hosts on many levels. Indeed, the success of chronic viral pathogens relies on their ability to outmanoeuvre host defences and lie low. Accordingly, the defensive measures mounted by the host often prove to be unsuccessful at eliminating the virus and instead can end up compromising the host in other ways. In HIV infection for example, unfettered attempts at eradicating the virus can leave the immune system hyperactivated and exhausted. This state of immune perturbation is likely to contribute to HIV-associated disease and ultimately death. It is well-established that T cells are the main targets of HIV-mediated immune dysfunction; however, extensive defects that affect B cells are also readily apparent in HIV-infected individuals. In their Review on page 235, Susan Moir and Anthony Fauci describe how HIV perturbs normal B-cell functions, leaving the host vulnerable to infection by opportunistic pathogens and B-cell-associated malignancies.

The long-standing suspicion that viruses are aetiological agents of autoimmune disease in genetically susceptible individuals points to another way in which viruses baffle the immune system. In the Review on page 246, Christian Münz and colleagues explain how viruses might trigger autoimmunity, diverting the attention of the immune system from viral proteins to self proteins through multiple mechanisms. The link between viruses and autoimmune disease is also supported by the recent findings (see In the News, page 224) that an enteroviral protein can be isolated from the pancreatic β-cells of patients with type 1 diabetes and that variation in a gene with a known role in antiviral immune responses influences susceptibility to type 1 diabetes.