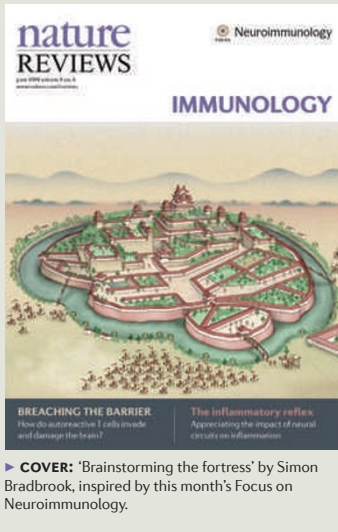




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**U**nderstanding the reciprocal relationship between the nervous and immune systems has become an important area of research and is the focus of this month's issue.

This relationship, influenced by neural circuits, can regulate innate immune responses, thereby preventing immune-mediated damage in the periphery, as described by Kevin Tracey (page 418). In addition, a controlled access of immune cells and mediators to the central nervous system (CNS) ensures necessary protection from infection, but the barriers that regulate this access can be breached, resulting in immune-mediated neurological disease.

One such disease — multiple sclerosis — is the topic of three articles in this issue. On page 393, Joan Goverman provides an in-depth overview of the infiltration, reactivation, effector functions and regulation of T cells that target the CNS. Lars Fugger and colleagues (page 408) describe the genetic determinants of multiple sclerosis and look to how we can identify the contributions of multiple genetic and environmental factors in disease pathogenesis. Lawrence Steinman (page 440) discusses the role of three key molecules in the relapse and remission of multiple sclerosis.

Besides T cells, other immune mediators can infiltrate the CNS and cause damage. As described in the Opinion article on page 449, circulating antibodies that crossreact with brain antigens might affect cognitive function when the barriers that normally prevent their entry are compromised.

In addition to these external influences, infection, ischaemic injury and endogenous toxic products (such as those associated with Alzheimer's disease) in the CNS can activate resident microglial cells through Toll-like receptors to initiate local innate immune responses, the results of which can not only be neurodestructive but also neuroprotective (page 429).

We hope that you enjoy this special Focus on Neuroimmunology.

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