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The survival of a species, be it human or virus, depends largely on its ability to evolve in the face of environmental and pathogenic insults. Numerous factors affect the ability of a species to survive. For humans, as Peter Parham describes on page 201, the inheritance of various combinations of the polymorphic HLA- and KIR (killer-cell immunoglobulin-like receptors)-gene-family members might influence our resistance to infections and our chances of successful reproduction. For a virus, the ability to escape host defence mechanisms is an important factor for ensuring viral persistence and effective transmission between hosts. The hepatitis viruses are good examples of persistent viruses. By comparing hepatitis B virus with hepatitis C virus, Barbara Rehermann and Michelina Nascimbeni (page 215) illustrate how these viruses have evolved different mechanisms of immune escape and survival, which probably reflect their differing disease pathogenesis.

At a cellular level, a fine balance between cell survival and cell death by apoptosis is crucial to prevent disorders such as autoimmunity, lymphoma and immunodeficiency. As discussed on page 189 by Andreas Strasser, the BH3-only proteins are key initiators of apoptosis during lymphocyte development and in response to cytokine signals, and are therefore important in maintaining the balance between life and death.

Elsewhere in this issue, Ronald Glaser and Janice Kiecolt-Glaser (page 243) describe how physiological stress can influence our immune responses and affect our ability to heal wounds. Bidirectional signals between the nervous, endocrine and immune systems modulate both cellular and humoral responses, so stress, as perceived by the brain, can lead to immune dysfunction and might contribute to premature ageing of a chronically stressed population.



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