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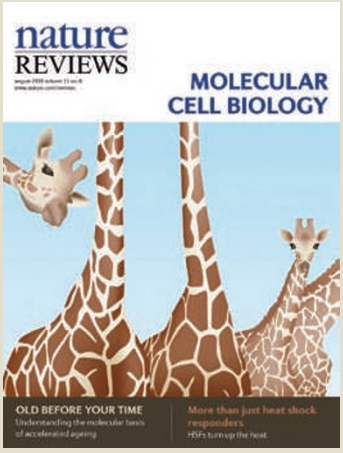

► **COVER:** 'It's all in the neck' by Vicky Summersby, inspired by the Review on p556.


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To survive exposure to stress, such as high temperatures and infection, eukaryotes depend on the heat shock response. This is mediated by heat shock factors (HSFs) — transcription factors that induce the expression of a family of molecular chaperones known as heat shock proteins (HSPs), which promote protein homeostasis. One such HSP, HSP70, is involved in a range of biological processes, including polypeptide folding and protein–protein interactions. As discussed by Harm H. Kampinga and Elizabeth A. Craig on page 579, this functional diversity is provided by a class of cofactors known as J proteins. These target HSP70 to specific locations in the cell or bind and deliver client proteins directly to HSP70.

Although HSFs are best known for promoting HSP transcription, some HSFs are also involved in development and ageing. On page 545, Malin Åkerfelt, Richard I. Morimoto and Lea Sistonen review the regulatory roles of HSFs in several developmental processes and key longevity pathways, such as insulin and insulin-like growth factor signalling. Thus, targeting HSFs could be beneficial for the treatment of age-related pathologies.

To develop targeted approaches for age-associated diseases, we need to better understand the molecular mechanisms underlying ageing and longevity. Studies on the premature ageing disease Hutchinson–Gilford progeria syndrome have revealed that it is caused by molecular changes similar to those in normal ageing, such as telomere attrition and defective stem cell homeostasis. On page 567 Christopher R. Burtner and Brian K. Kennedy explore the connection between progerias and ageing and suggest that the study of accelerated ageing and animal models of longevity will help to elucidate the molecular basis of the ageing process.

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