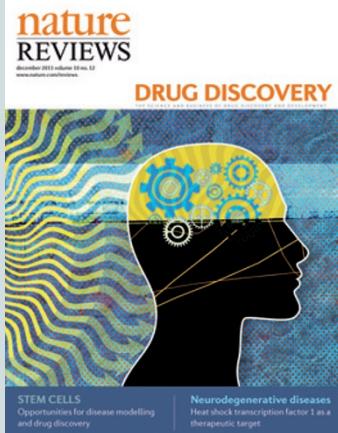




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► **COVER:** 'Heat shock transcription factor 1 and protein-folding diseases' by Susanne Harris, inspired by the Review on p930.

The generation of induced pluripotent stem cells (iPSCs) from human somatic cells has provided a new platform for studying disease pathogenesis and for cell-based phenotypic screening to identify novel therapies. In their Review, Daley and colleagues discuss key issues and recent advances associated with the generation and application of iPSCs in drug discovery, and highlight diseases that have been successfully modelled using patient-derived iPSCs, including spinal muscular atrophy, familial dysautonomia, long QT syndrome and Parkinson's disease. Neurodegenerative diseases such as Parkinson's disease are associated with the accumulation of misfolded proteins, which results in neuronal dysfunction and cell death. Thiele and colleagues review the therapeutic potential of enhancing cellular protein-folding capacity through elevated expression of chaperone proteins, which is regulated by heat shock transcription factor 1 (HSF1), discussing emerging strategies and challenges in the development of small-molecule HSF1 activators for the treatment of neurodegenerative diseases. Neuronal excitability and heart rate are controlled in part by hyperpolarization-activated cyclic nucleotide-gated (HCN) channels. Postea and Biel provide an overview of the HCN channel family and the roles of these channels in disease, focusing on their potential to be targeted in the development of novel anticonvulsant, anaesthetic, analgesic and bradycardic drugs. Cardiovascular disorders such as atherosclerosis, restenosis, thrombosis and hypertension are among the many diseases in which roles of members of the CCN family of extracellular matrix proteins have been implicated. In a final Review, Jun and Lau discuss the biological and pathological functions of CCN proteins and explore their potential as diagnostic markers and therapeutic targets in disorders linked to inflammation or chronic tissue injury.



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