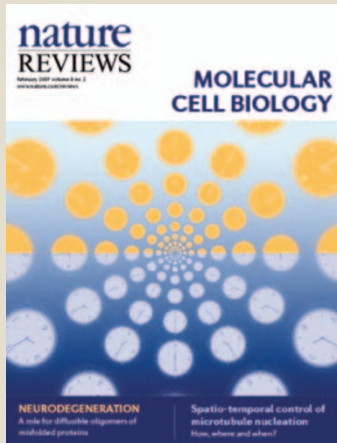




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► **COVER:** 'Clockwork' by Vicky Askew, inspired by the Review on p139



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Traditionally, the study of gene regulation has focused on understanding the control of transcription and has largely ignored the contribution of mRNA decay. But, as Nicole L. Garneau, Jeffrey Wilusz and Carol J. Wilusz discuss in their Review article on page 113, recent advances in the study of mRNA turnover have dispelled the misconception that transcript breakdown is simply a biological waste-disposal system and have validated mRNA decay as a tightly controlled cellular process that is as germane to gene regulation as is transcription.

The importance of mRNA decay is reflected in the many pathways that converge on mRNA turnover, in the identification of cytoplasmic compartments that seem to specialize in mRNA breakdown, and in the crosstalk of decay pathways with other aspects of RNA metabolism, including mRNA-surveillance systems that detect and destroy faulty transcripts, thereby preventing the synthesis of abnormal proteins.

Despite the many safeguards that prevent aberrant translation, defective proteins can be generated in other ways, for instance by misfolding. In Alzheimer's disease (AD), aggregates of amyloid β -protein (A β), a small peptide that is produced by the cleavage of a transmembrane precursor, are associated with neuropathology. However, the exact nature of the toxic species has remained controversial. Christian Haass and Dennis J. Selkoe (page 101) believe that this long-standing debate is on the brink of resolution. Recent studies clearly implicate soluble A β oligomers — intermediates in the aggregation process — in the impairment of long-term potentiation and in hippocampal cell destruction. Haass and Selkoe explain how these new insights into the molecular basis of AD have provided common ground for understanding other neurodegenerative disorders and are resulting in new treatment strategies for this ever more common disorder.

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