

# Hormesis: Protecting neurons against cellular stress in Parkinson disease

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Protein folding stress is a salient feature of the most frequent neurodegenerative diseases. Although the accumulation of abnormally folded proteins is a well-characterized event underlying the pathology, the way cells respond to this phenomenon is not well understood. Signs of endoplasmic reticulum (ER) stress are a common marker of neurodegeneration in many diseases, which may represent two contrasting processes: cell protection events due to activation of adaptive programs, or a chronic stress state that culminates in apoptosis to eliminate irreversibly injured cells.

Autophagy has been proposed as a protective mechanism to overcome neurodegeneration that is also modulated by ER stress. In this issue of autophagy Bertrand Mollereau's group provides novel evidence indicating that engagement of nonharmful levels of ER stress protects against experimental Parkinson disease. At the mechanistic level, a homeostatic crosstalk between ER stress signaling and the autophagy pathway was propo