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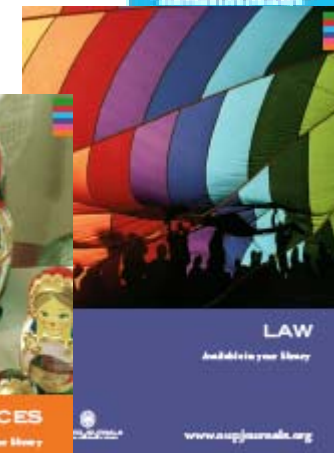
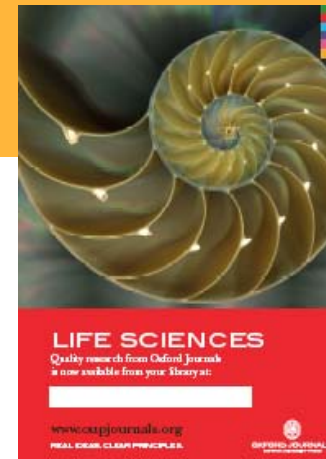
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### What clinical disorders tell us about the neural control of saccadic eye movements

Stefano Ramat<sup>1</sup>, R. John Leigh<sup>2</sup>, David S. Zee<sup>3</sup> and Lance M. Optican<sup>4</sup>

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## The role of autophagy-lysosome pathway in neurodegeneration associated with Parkinson's disease

Tianhong Pan<sup>1</sup>, Seiji Kondo<sup>2</sup>, Weidong Le<sup>1</sup> and Joseph Jankovic<sup>3</sup>

<sup>1</sup>Parkinson's Disease Research Laboratory, Baylor College of Medicine, <sup>2</sup>Department of Neurosurgery, University of Texas M. D. Anderson Cancer Center and <sup>3</sup>Parkinson's Disease Center and Movement Disorders Clinic, Department of Neurology, Baylor College of Medicine, Houston, TX, USA

Correspondence to: Joseph Jankovic, MD, Professor of Neurology, Director, Parkinson's Disease Center, and Movement Disorders Clinic, Baylor College of Medicine, Department of Neurology, 6550 Fannin #1801 Houston, TX 77030, USA. E-mail: [josephj@bcm.tmc.edu](mailto:josephj@bcm.tmc.edu)

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### Summary

The ubiquitin-proteasome system (UPS) and autophagy-lysosome pathway (ALP) are the two most important mechanisms that normally repair or remove abnormal proteins. Alterations in the function of these systems to degrade misfolded and aggregated proteins are being increasingly recognized as playing a pivotal role in the pathogenesis of many neurodegenerative disorders such as Parkinson's disease. Dysfunction of the UPS has been already strongly implicated in the pathogenesis of this disease and, more recently, growing interest has been shown in identifying the role of ALP in neurodegeneration. Mutations of  $\alpha$ -synuclein and the increase of intracellular concentrations of non-mutant  $\alpha$ -synuclein have been associated with Parkinson's disease phenotype. The demonstration that  $\alpha$ -synuclein is degraded by both proteasome and autophagy indicates a possible linkage between the dysfunction of the UPS or ALP and the occurrence of this disorder.

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