Drosophila Histone Deacetylase 6 Protects Dopaminergic Neurons against α-Synuclein Toxicity by Promoting Inclusion Formation

Guiping Du, Xiang Liu, Xinpeng Chen, Mei Song, Yan Yan, Renjie Jiao, and Chih-chen Wang

dHDAC6 functions to suppress α-synuclein–induced neurodegeneration and locomotion defects in a Drosophila PD model through promoting α-synuclein–enriched inclusion formation while reducing the toxic oligomers.

Mol. Biol. Cell 21 (13), 2128–2137

Mitochondrial Fragmentation Leads to Intracellular Acidification in Caenorhabditis elegans and Mammalian Cells

David Johnson and Keith Nehrke

It is unclear how lesions in genes that regulate mitochondrial structural dynamics lead to disease. Here, tissue-specific biosensors expressed in the genetic model organism Caenorhabditis elegans are used to demonstrate that robust cellular acidification occurs when mitochondria are fragmented and may contribute to the etiology of morphology diseases in mammals.

Mol. Biol. Cell 21 (13), 2191–2201

MDCK cells expressing a constitutively active variant of ARF6 form cysts with multiple lumens. (Image: James Clancy and Crislyn D’Souza-Schorey, University of Notre Dame)
Fusel Alcohols Regulate Translation Initiation by Inhibiting eIF2B to Reduce Ternary Complex in a Mechanism That May Involve Altering the Integrity and Dynamics of the eIF2B Body

Eleanor J. Taylor, Susan G. Campbell, Christian D. Griffiths, Peter J. Reid, John W. Slaven, Richard J. Harrison, Paul F.G. Sims, Graham D. Pavitt, Daniela Delneri, and Mark P. Ashe

This study highlights a connection between the eIF2B body and the regulation of translation initiation as a response to stress in Saccharomyces cerevisiae. Fusel alcohols are involved in signaling nitrogen scarcity to the cell, and they inhibit protein synthesis by preventing the movement of the eIF2B body throughout the cell.

Mol. Biol. Cell 21 (13), 2202–2216

A Gαi–GIV Molecular Complex Binds Epidermal Growth Factor Receptor and Determines Whether Cells Migrate or Proliferate


Migrating cells do not proliferate and vice versa, but the mechanism involved remains unknown. This study reveals how this cellular decision is made by showing that a Gαi–GIV molecular complex interacts with EGF receptor and programs growth factor signaling, triggering migration when assembled and favoring mitosis when assembly is prevented.

Mol. Biol. Cell 21 (13), 2338–2354

Unregulated ARF6 Activation in Epithelial Cysts Generates Hyperactive Signaling Endosomes and Disrupts Morphogenesis

Jogender S. Tushir, James Clancy, Andrew Warren, Carolyn Wrobel, Joan S. Brugge, and Crislyn D’Souza-Schorey

This study shows that constitutive ARF6 activation during epithelial cyst morphogenesis promotes the formation of signaling endosomes that serve as platforms for hyperactive receptor signaling and leads to the generation of tumorigenic glandular phenotypes.

Mol. Biol. Cell 21 (13), 2355–2366

The Interferon-γ–induced Murine Guanylate-Binding Protein-2 Inhibits Rac Activation during Cell Spreading on Fibronectin and after Platelet-derived Growth Factor Treatment: Role for Phosphatidylinositol 3-Kinase

Angela F. Messmer-Blust, Sujata Balasubramanian, Victoria Y. Gorbacheva, Jonathan A. Jeyaratnam, and Deborah J. Vestal

IFN-γ and mGBP-2 inhibit the spreading of fibroblasts on fibronectin by inhibiting Rac activation. mGBP-2 is incorporated into a protein complex with the catalytic subunit of PI3-K, p110, and inhibits PI3-K activation during spreading. This is a novel mechanism by which IFN-γ can alter how cells respond to extracellular signals.

Mol. Biol. Cell 21 (14), 2514–2528

A lens from a transgenic mouse expressing the N-terminal nuclear receptor box of transcriptional coactivator Ncoa6 shows abnormal lens fiber cell differentiation and denucleation. (Image: Wei-Lin Wang, Department of Genetics, Albert Einstein College of Medicine)