clathrin-mediated endocytosis *in vivo*, showing that their roles are tightly intertwined in a common mechanistic pathway.

Using *Drosophila* as a model system, Verstreken *et al*. identified synaptojanin mutants and characterized their synaptic terminals from the ultrastructural and physiological standpoints. As the phenotypic abnormalities they found — defective neurotransmission and absence of synaptic vesicles in most terminals — were reminiscent of what had been described in endophilin mutants, the authors studied the combined effect of mutations in both proteins to test the nature of their interaction. As the phenotype of the double mutants was not different from that of either mutant alone, they concluded that synaptojanin and endophilin are functional partners that act on the same endocytic pathway.

Schuske *et al*. focused their attention on endophilin, and analysed synaptic structure and function in *Caenorhabditis elegans* mutants. These authors also noted the similarity between the endophilin and synaptojanin mutant phenotypes, and performed the same interaction test to arrive independently at the same conclusion: the interaction between the two proteins is crucial for their function in endocytosis *in vivo*.

These studies support the idea that endophilin acts as an adaptor that recruits synaptojanin to sites of endocytosis, enabling it to regulate the assembly and disassembly of the clathrin coat owing to the ability of endophilin to hydrolyse PtdIns(4,5)P_2. But whether the acyl transferase activity of endophilin is also relevant for its function *in vivo* is still uncertain. The characterization of endophilin mutants in which its protein–protein interactions are dissociated from its enzymatic activity might help to solve this problem.

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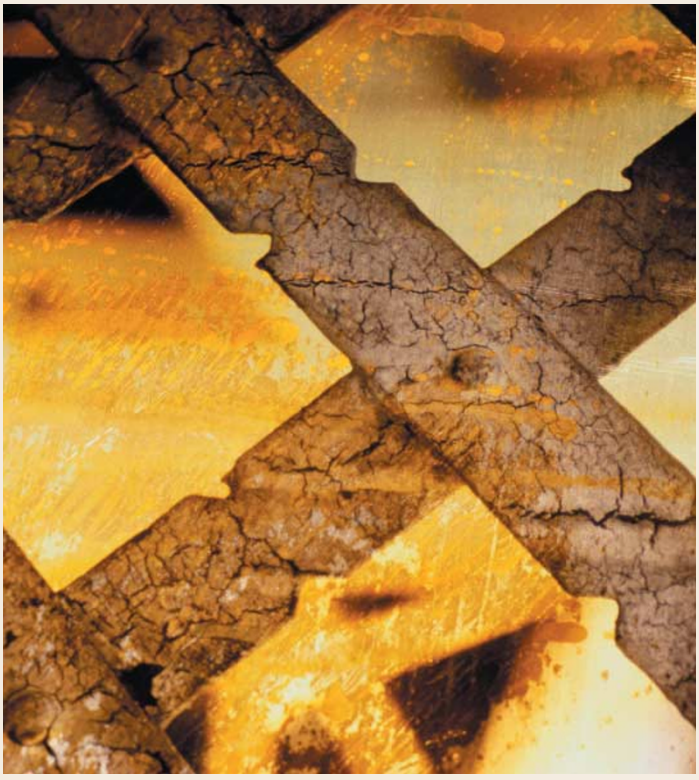
References and links

ORIGINAL RESEARCH PAPERS Verstreken, P. *et al.* Synaptojanin is recruited by endophilin to promote synaptic vesicle uncoating. *Neuron* **40**, 733–748 (2003) | Schuske, K. R. *et al.* Endophilin is required for synaptic vesicle endocytosis by localizing synaptojanin. *Neuron* **40**, 749–762 (2003)

FURTHER READING Slepnev, V. I. & De Camilli, P. Accessory factors in clathrin-dependent synaptic vesicle endocytosis. *Nature Rev. Neurosci.* **1**, 161–172 (2000)



IN BRIEF

SENSORY PROCESSING

Neuronal processing delays are compensated in the sensorimotor branch of the visual system.

Kerzel, D. & Gegenfurtner, K. R. *Curr. Biol.* **13**, 1975–1978 (2003)

How does the visual system deal with the fact that a moving object will continue to move during the delay between photons hitting the retina and information reaching the higher cortex? Although we could extrapolate the path of a moving object, we do not see such objects ahead of their final stopping point. Kerzel and Gegenfurtner find that perception of a final target position is accurate, but that reaching is directed beyond the final position, indicating that compensation for processing delays occurs late in sensorimotor processing.

NEUROTECHNIQUE

A rapid cellular FRET assay of polyglutamine aggregation identifies a novel inhibitor.

Pollitt, S. K. *et al. Neuron* **40**, 685–694 (2003)

This study describes a high-throughput system based on fluorescence resonance energy transfer that allows experimenters to measure intracellular agreggation of polyglutamine proteins, which are implicated in diseases such as Huntington's disease. The authors screened over 2,800 molecules for inhibitory activity and characterized one, an inhibitor of the Rho-associated kinase p160ROCK, in detail. Such compounds could provide potential new therapeutic agents and could also provide new insights into the pathogenic mechanisms of these polyglutamine diseases.

SENSORY TRANSDUCTION

Molecular basis for ultraviolet vision in invertebrates.

Salcedo, E. *et al. J. Neurosci.* **23**, 10873–10878 (2003)

The authors show that ultraviolet sensitivity in invertebrates arises as a result of a single amino-acid polymorphism (the replacement of an asparagine or glutamate in blue-sensitive rhodopsin with a lysine). The same polymorphism is responsible for ultraviolet vision in birds, but not in other animals, indicating that ultraviolet vision arose independently but by a similar molecular mechanism in birds and insects.

NEUROLOGICAL DISORDERS

Loss of m-AAA protease in mitochondria causes complex I deficiency and increased sensitivity to oxidative stress in hereditary spastic paraplegia.

Atorino, L. *et al. J. Cell Biol.* **163**, 777–787 (2003)

Paraplegin is one of a number of proteins in which mutations can cause hereditary spastic paraplegia (HSP). Atorino *et al*. show that this protein forms a complex in the mitochondrial inner membrane with a homologous protein, and that this complex is abnormal in patients with HSP. In cells that lack this complex, mitochondria show reduced complex 1 activity and increased sensitivity to oxidative stress, perhaps shedding new light on the pathogenesis of HSP.