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# IN BRIEF

## COGNITIVE NEUROSCIENCE

### Conjunctive representation of position, direction, and velocity in entorhinal cortex.

Sargolini, F. *et al. Science* **312**, 758–762 (2006)

Grid cells in the medial entorhinal cortex (MEC) form part of a spatial coordinate system for navigation. Other cells in the MEC encode information about head direction. By studying the activity of MEC neurons in rats as they explored a two-dimensional environment, Sargolini *et al.* showed that these cell types are co-localized in layers III and V of the MEC, in which some grid cells are directionally tuned and some head direction cells have grid correlates. The authors propose that cells with conjunctive properties facilitate the integration of directional, positional and translational information during self-motion-based navigation.

## NEURODEGENERATIVE DISEASES

### *Drosophila pink1* is required for mitochondrial function and interacts genetically with *parkin.*

Clark, I. E. *et al. Nature* 3 May 2006 10.1038/nature4779

### Mitochondrial dysfunction in *Drosophila PINK1* mutants is complemented by *parkin.*

Park, J. *et al. Nature* 3 May 2006 10.1038/nature04788

Mitochondrial dysfunction has long been implicated in the pathogenesis of Parkinson's disease (PD) because environmental mitochondrial toxins cause PD-like pathology. The recent finding that mutations in PTEN-induced kinase 1 (PINK1), which encodes a mitochondrial kinase, cause familial PD, has added weight to this theory. Two groups now show that *Drosophila pink1* is crucial for mitochondrial function and acts upstream of another PD-associated gene, *parkin*. Clark *et al.* describe *pink1*-null flies with male sterility, apoptotic muscle degeneration and increased sensitivity to oxidative and other stresses. Mitochondria in these flies had fragmented cristae, with some appearing almost hollow. Expression of human *PINK1* rescued the *pink1*-null phenotype, indicating functional conservation. *pink1* loss-of-function mutants generated by Park *et al.* exhibited degeneration of muscle and dopaminergic neurons, as well as locomotive defects. Mitochondria were grossly enlarged and severely impaired. Interestingly, both groups noticed that *pink1*-mutant flies had a similar phenotype to flies lacking functional *parkin* — an E3 ubiquitin ligase — and that *parkin* overexpression could rescue the *pink1* mutant phenotype.

## CELL BIOLOGY OF THE NEURON

### $\alpha 3 Na^+/K^+$-ATPase is a neuronal receptor for agrin.

Hilgenberg, L. G. W. *et al. Cell* **125**, 359–369 (2006)

Agrin is required for the clustering of acetylcholine receptors at the developing neuromuscular junction, a function that is mediated by its interaction with the receptor tyrosine kinase MuSK. Agrin has also been implicated in synapse formation, calcium homeostasis and neuronal activity, but the receptor in the brain that mediates these functions has remained unknown. Now, Hilgenberg *et al.* show that agrin binds to and inhibits the $\alpha 3$ subunit of the $Na^+/K^+$-ATPase in CNS neurons, thereby providing a mechanism for agrin to regulate neuronal activity in the CNS.