IN BRIEF

SLEEP

The *Drosophila* fragile X mental retardation gene regulates sleep need

Bushey, D. *et al. J. Neurosci.* **29**, 1948–1961 (2009)

Periods of increased synaptic plasticity have been associated with heightened sleep need, but the molecular mechanisms that link the two are largely unknown. Loss-of-function and gain-of-function mutations in the *Drosophila melanogaster* fragile X mental retardation gene, *Fmr1*, are linked to defects in synaptic plasticity, and the authors show here that these mutations also increase or decrease, respectively, daily sleep amounts. Furthermore, the mutants lose the homeostatic pressure that normally follows sleep deprivation. Thus, FMR1 may provide a molecular link between synaptic plasticity and sleep regulation.

NEURAL CIRCUITS

The interscutularis muscle connectome

Lu, J. *et al. PLoS Biol.* **7**, e100032 (2009)

The hope that understanding connectivity will provide insights into nervous system function has fuelled ongoing efforts to generate complete wiring diagrams (connectomes) of many neural circuits. Here, the authors used mice expressing a fluorescent protein in their motor neurons to reconstruct the complete peripheral neuromuscular circuit that is associated with an individual interscutularis muscle. By comparing the connectomes of individual muscles, they were able to characterize some general organizational principles and identify areas of significant variability between connectomes.

SYNAPTIC PLASTICITY

Neto1 is a novel CUB-domain NMDA receptor-interacting protein required for synaptic plasticity and learning

Ng, D. *et al. PLoS Biol* . **7**, e1000041 (2009)

More than 70 auxiliary proteins have been associated with the NMDAR (*N*-methyl-D-aspartate receptor). Here, the authors show that a newly-discovered NMDAR-interacting protein, neuropilin and tolloid-like 1 (NETO1), is essential for maintaining the abundance of NR2A subunit-containing receptors in the postsynaptic density. Mice that lack NETO1 have reduced synaptic plasticity at the hippocampal Schaffer collateral–CA1 synapse and demonstrate impaired spatial learning. These findings support the idea that auxillary proteins can regulate NMDAR function.

NEURODEGENERATIVE DISEASE

Synchronous hyperactivity and intercellular calcium waves in astrocytes in Alzheimer mice

Kuchibhotla, K. V. *et al. Science* **323**, 1211–1215 (2009)

Amyloid plaques disrupt Ca^{2+} homeostasis in neurons; however, their effects on astrocytes were unclear. Using multiphoton fluorescent lifetime imaging microscopy, the authors showed that resting levels of Ca^{2+} and spontaneous activity are raised throughout the astrocyte network in a mouse model of Alzheimer's disease. Furthermore, coordinated changes in Ca^{2+} levels between distant astrocytes, as well as Ca^{2+} waves initiated by astrocytes located close to amyloid plaques, were also observed. These findings may explain how localized amyloid deposits are linked to global disruption of cortical function.