Nature Reviews Neuroscience | Published online 9 Feb 2017

IN BRIEF

SYNAPTIC TRANSMISSION

Recycling regulators

The mechanisms that regulate synaptic vesicle recycling are unclear. Here, Li *et al.* show that Ca^{2+} sensors that drive vesicle fusion also influence vesicle endocytosis. High-resolution recordings of fluorescently tagged vesicles revealed a role of synaptotagmin 1 in the regulation of the endocytosis of single synaptic vesicles. Furthermore, whereas synaptotagmin 1 and other canonical vesicle-fusion machinery components facilitate rapid multivesicular endocytosis after repetitive stimulation, synaptotagmin 7 promotes asynchronous neurotransmitter release and targets vesicles towards a slow endocytic pathway.

ORIGINAL ARTICLE Li, Y. C. *et al.* Synaptotagmin-1- and synaptotagmin-7-dependent fusion mechanisms target synaptic vesicles to kinetically distinct endocytic pathways. *Neuron* http://dx.doi.org/10.1016/j.neuron.2016.12.010 (2017)

SLEEP AND MEMORY

REM sleep targets new synapses

To support neural circuit stability and memory function, new synapses must be selectively pruned or maintained. Li *et al.* used two-photon imaging of the dendritic spines of mouse primary motor cortex pyramidal neurons to demonstrate the role of rapid eye movement (REM) sleep in these processes. They found that REM sleep deprivation impairs the pruning of new synapses and the strengthening and survival of maintained new synapses during development and after motor learning, and demonstrated a role for NMDA receptor-mediated dendritic Ca^{2+} transients in this effect.

ORIGINAL ARTICLE Li, W. *et al.* REM sleep selectively prunes and maintains new synapses in development and learning. *Nat. Neurosci.* http://dx.doi.org/10.1038/nn.4479 2017)

PAIN

Improving opioids

Chronic use of opioid analgesics can cause analgesic tolerance and opioid-induced hyperalgesia (OIH), contributing to dose escalation in patients. Corder *et al.* here provide evidence that, in mice, these unwanted effects, and the underlying altered circuit plasticity, are mediated by μ opioid receptors (MORs) expressed by primary nociceptors. Targeted genetic deletion of peripheral MORs or treatment with peripherally restricted MOR antagonists suppressed opioid tolerance and OIH without affecting analgesia, suggesting a possible strategy to improve pain management in patients.

ORIGINAL ARTICLE Corder, G. *et al.* Loss of μ opioid receptor signaling in nociceptors, but not microglia, abrogates morphine tolerance without disrupting analgesia. *Nat. Med.* http://dx.doi.org/10.1038/nm.4262 (2017)

NEUROMODULATION

A circuit modulator

The cytokine IL-17 is expressed in the brain, but its function is not known. Chen *et al.* found that the *Caenorhabditis elegans* orthologue of IL-17 regulates the aggregation of worms following a rise in environmental oxygen and that it mediates this effect by enhancing the responsiveness of the RMG interneuron that coordinates this response to presynaptic input from oxygen sensors. Thus, IL-17 might act as a neuromodulator that can potentiate neural circuit function.

ORIGINAL ARTICLE Chen, C. *et al.* IL-17 is a neuromoduator of *Caenorhabditis elegans* sensory responses. *Nature* http://dx.doi.org/10.1038/nature20818 (2017)