

## IN BRIEF

## GLIA

## Glial messaging

A new study provides evidence for a glia-mediated form of long-term potentiation (LTP) in the rat spinal cord. Activation of astrocytes and microglia induced LTP at spinal C-fibre synapses on lamina I neurons *in vitro*. Blocking glial metabolism or signalling induced by D-serine, which is released by astrocytes, prevented LTP induction in spinal C-fibres after high-frequency stimulation (HFS) of the sciatic nerve. Finally, spinal superfusate from mice that underwent such HFS induced LTP in C-fibres in naive animals, and this was blocked by inhibiting signalling by D-serine or tumour necrosis factor, also released by glia.

**ORIGINAL ARTICLE** Kronschläger, M. T. *et al.* Gliogenic LTP spreads widely in nociceptive pathways. *Science* **354**, 1144–1148 (2016)

## LEARNING AND MEMORY

## The cannabinoid connection

How acute changes in the activity of neuronal mitochondrial affect learning and memory is unclear. By precluding mitochondrial localization of cannabinoid receptor 1 (CB1) in the mouse hippocampus, the authors prevented CB1-agonist-induced inhibition of excitatory transmission in hippocampal slices and of the poor performance of animals in the novel-object recognition test. Moreover, activation of mitochondrial CB1 decreased neuronal mitochondrial respiration. Together, these data show that acute alterations in mitochondrial activity can regulate memory formation.

**ORIGINAL ARTICLE** Hebert-Chatelain, E. *et al.* A cannabinoid link between mitochondria and memory. *Nature* **539**, 555–559 (2016)

## CELL BIOLOGY OF THE NEURON

## Endocytic mediators

Whether actin has a role in endocytosis in mammalian cells is unclear. Wu *et al.* showed that, in mice, knockout of the  $\beta$ -actin gene or  $\gamma$ -actin gene impaired various types of endocytosis in calyx-type and hippocampal synapses. The deficits observed in hippocampal boutons could be rescued by the expression of wild-type actin isoforms but not by polymerization-deficient mutant actin. Moreover, knockout of the  $\beta$ -actin gene led to a reduction in the number of membranous pits in such boutons. These data suggest that polymerized actin is crucial for endocytosis at different synapses.

**ORIGINAL ARTICLE** Wu, X.-S. *et al.* Actin is crucial for all kinetically distinguishable forms of endocytosis at synapses. *Neuron* <http://dx.doi.org/10.1016/j.neuron.2016.10.014> (2016)

## GLIA

## An astrocytic influence

How astrocytic  $\text{Ca}^{2+}$  signalling interacts with neural activity *in vivo* and affects behaviour is not well understood. A new study in *Drosophila melanogaster* found that astrocytic knockdown of a transient receptor potential cation channel inhibited somatic astrocytic  $\text{Ca}^{2+}$  transients and impaired olfactory behaviour and touch-induced startle responses in larvae. It also identified a neuronal population that, via the release of octopamine (Oct) and tyramine (Tyr), induced such transients and showed that disruption of this Oct and Tyr signalling led to the behavioural deficits. Last, the study revealed that these astrocytes seem to influence behaviour through effects on dopaminergic neurons.

**ORIGINAL ARTICLE** Ma, Z. *et al.* Neuromodulators signal through astrocytes to alter neural circuit activity and behaviour. *Nature* **539**, 428–432 (2016)