

IN BRIEF

CORTICAL PHYSIOLOGY**Studying waves up close**

During sleep, brain state is controlled by slow, rhythmic waves of activity in cortical and thalamic neurons. Stroh *et al.* now show that optogenetic activation of fewer than 100 cortical layer 5 neurons in mice can initiate waves of activity that spread throughout the cortex before recruiting thalamic neurons. The authors used a novel optical fibre technique to allow simultaneous excitation and imaging of neurons, even deep within the brain, giving new insight into the generation of corticothalamic waves.

ORIGINAL RESEARCH PAPER Stroh, A. *et al.* Making waves: initiation and propagation of corticothalamic Ca^{2+} waves in vivo. *Neuron* **77**, 1136–1150 (2013)

ADDICTION**Turning down drug-seeking**

It has been proposed that deficits in prefrontal cortex function might be related to compulsive drug-seeking in individuals with addiction, but the cause and effects of such deficits are unclear. Now, Chen *et al.* show that prolonged cocaine self-administration in rats leads to decreased excitability of prefrontal neurons. When the authors used optogenetic techniques to stimulate or inhibit the affected areas of the cortex, compulsive drug-seeking was reduced or increased, respectively. Targeted stimulation of the prefrontal cortex might be a new approach to be pursued in addiction therapy research.

ORIGINAL RESEARCH PAPER Chen, B. T. *et al.* Rescuing cocaine-induced prefrontal cortex hypoactivity prevents compulsive cocaine seeking. *Nature* **3** Apr 2013 (doi:10.1038/nature12024)

NEURODEGENERATIVE DISEASE**Oligodendrocyte loss in ALS**

Amyotrophic lateral sclerosis (ALS) involves the progressive degeneration of motor neurons, but the disease also seems to involve glial cells such as oligodendrocytes and their precursors. Kang *et al.* now show that there is extensive degeneration of grey matter oligodendrocytes in the spinal cord of the superoxide dismutase 1 (*SOD1*) mouse model of ALS; although there is also increased proliferation and differentiation of oligodendrocyte precursors, these cells fail to mature into functioning oligodendrocytes. Selective removal of the mutant gene from oligodendrocytes in these mice delayed the progression of the disease, indicating that the genetic defect in ALS might cause motor neurons to degenerate by reducing the ability of oligodendrocytes to support them.

ORIGINAL RESEARCH PAPER Kang, S. H. *et al.* Degeneration and impaired regeneration of gray matter oligodendrocytes in amyotrophic lateral sclerosis. *Nature Neurosci.* **31** Mar 2013 (doi: 10.1038/nn.3357)

SYNAPTIC PHYSIOLOGY**Phosphoinositides at the synapse**

The lipid phosphatidylinositol-3,4,5-trisphosphate (PI(3,4,5)P₃) is found in small amounts in both the presynaptic and postsynaptic terminals. Postsynaptically, it helps to cluster receptors and other proteins at the terminal. Now, Khuong *et al.* use super-resolution imaging to show that PI(3,4,5)P₃ interacts electrostatically with syntaxin 1A in the presynaptic active zone and thereby generates clusters of syntaxin 1A, which are essential for neurotransmitter release.

ORIGINAL RESEARCH PAPER Khuong, T. M. *et al.* Synaptic PI(3,4,5)P₃ is required for syntaxin1A clustering and neurotransmitter release. *Neuron* **77**, 1097–1108 (2013)