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# IN BRIEF

## AXON DEGENERATION

### A receptor for injury

Axonal injuries can trigger a process of axonal disintegration known as Wallerian degeneration, which is thought to be largely driven by intrinsic mechanisms in the axon. Here, knocking out the death receptor 6 (*Dr6*) gene in cultured mouse sympathetic or dorsal root ganglion neurons was associated with prolonged preservation of axons in an intact state following axotomy. Moreover, following sciatic nerve axotomy, mice lacking *Dr6* exhibited marked preservation of axons in sciatic nerves compared with wild-type mice. Finally, the absence of DR6 was associated with a lack of activation of a kinase implicated in Wallerian degeneration. This study indicates that a neuronal receptor has a key role in this process.

**ORIGINAL ARTICLE** Gamage, K. K. *et al.* Death receptor 6 promotes Wallerian degeneration in peripheral axons. *Curr. Biol.* **27**, 890–896 (2017)

## NEURAL CIRCUITS

### Itch transmission

Humans exhibit socially contagious behaviours, including contagious itching, but studying the underlying mechanisms has been difficult. Yu *et al.* found that they could socially transmit scratching to mice by allowing them to watch mice with chronic itch. The observer mice showed upregulated neuronal activity in the suprachiasmatic nucleus (SCN) after watching the mice that exhibited excessive scratching. Ablation of gastrin-releasing peptide (GRP) signalling in the SCN inhibited scratching transmission but not the ability of the mice to scratch, and optogenetic activation of GRP-expressing SCN neurons induced scratching. These data provide evidence for a circuit underlying a socially contagious behaviour in mice.

**ORIGINAL ARTICLE** Yu, Y.-Q. *et al.* Molecular and neural basis of contagious itch behavior in mice. *Science* **355**, 1072–1076 (2017)

## DENDRITES

### Probing plasticity

Two new studies provide new tools for examining mechanisms of plasticity in dendrites. Tang and Yasuda developed new fluorescence resonance energy transfer (FRET)-based sensors for extracellular signal-regulated kinase (ERK) and protein kinase A (PKA), which are implicated in synaptic plasticity, primarily by replacing the fluorophore pairs in existing FRET sensors. The new sensors were ~2–3-fold more sensitive and allowed ERK and PKA activity to be visualized in individual dendritic spines of hippocampal neurons during structural plasticity under two-photon fluorescence lifetime imaging microscopy. Murakoshi *et al.* developed a genetically encoded, photo-inducible calcium/calmodulin-dependent protein kinase type II (CaMKII) inhibitor. The induction of long-term potentiation (LTP) and structural plasticity in spines requires CaMKII, but it has been hard to assess the temporal requirement of its activation in these processes. In hippocampal slices, photoactivation of the inhibitor revealed that CaMKII activation is needed early in the process of LTP induction for LTP and structural plasticity to occur. Moreover, in mice in an inhibitory avoidance task, photoactivation of the inhibitor showed that CaMKII activity was necessary in amygdalar neurons during training, but not after training completion, for the formation of memories.

**ORIGINAL ARTICLES** Tang, S. & Yasuda, R. Imaging ERK and PKA activation in single dendritic spines during structural plasticity. *Neuron* **93**, 1315–1324 (2017) | Murakoshi, H. *et al.* Kinetics of endogenous CaMKII required for synaptic plasticity revealed by optogenetic kinase inhibitor. *Neuron* http://dx.doi.org/10.1016/j.neuron.2017.02.036 (2017)