constitutively active or inactive form of CAMKII had shorter and longer mitochondria (indicating higher and lower baseline fission rates) than did controls, respectively. Thus, CAMKII may activate DRP1 to drive local fission after stimulation.

Neurons expressing inactive DRP1 or inactive DYN2 showed less dendritic spine growth and AMPAR trafficking after cLTP stimulation than did controls. In mouse hippocampal slices, high-frequency stimulation (HFS) of Schaffer collaterals potentiated field excitatory postsynaptic potentials (fEPSPs) in CA1 dendritic spines, and mitochondria in CA1 of stimulated slices were shorter than those in slices that were stimulated but also treated with an NMDAR antagonist. Moreover, HFS of Schaffer collaterals in hippocampal slices expressing inactive DRP1 only weakly potentiated CA1 fEPSPs compared with those in controls slices. These results suggest that mitochondrial fission is necessary for structural and functional LTP.

Mitochondrial fission might evoke changes in Ca^{2+} handling that aid synaptic plasticity. In the dendritic mitochondria of cultured neurons expressing a mitochondrial Ca^{2+} sensor, stimulation generated transient increases in mitochondrial matrix Ca^{2+} levels, which the authors called mCaTs. Dendritic mitochondria in stimulated neurons expressing inactive DRP1 also showed mCaTs but, compared with control mCaTs, they were less frequent, smaller and shorter. Thus, mitochondrial fission may drive changes in Ca^{2+} regulation during LTP.

Together, these findings demonstrate NMDAR-dependent LTP requires fission of dendritic mitochondria that in turn depends on Ca^{2+}, CAMKII, DRP1 and DYN2. The authors note that the mitochondrial fission and mCaTs observed in activated dendrites might reflect or promote dendritic calcium integration and/or metabolic processes that are needed for LTP.

Natasha Bray

ORIGINAL ARTICLE Divakaruni, S. S. et al. Long-term potentiation requires a rapid burst of dendritic mitochondrial fission during induction. *Neuron* https://doi.org/10.1016/j.neuron.2018.09.025 (2018)

suggest that CD47 protects a specific synapse population from engulfment.

The authors next examined dLGN synapse number in mice at P60, when synaptic refinement in the retinogeniculate system is finished. They found that the number of dLGN synapses was lower in *Cd47*-KO mice than in the wild-type animals, indicating that CD47 loss leads to overpruning. In dLGN slices from P60 mice, there was a reduction in NMDA receptor-mediated currents in relay cells, indicating that there are fewer RGC inputs to these cells and, hence, that the overpruning has a functional effect.

How does CD47 inhibit pruning? In line with pruning levels, mRNA and protein levels of SIRPα were elevated in microglia at P5 and P10 but declined later (at P30). Moreover, mice lacking SIRPα had a similar increased engulfment phenotype to that of *Cd47*-KO mice. A series of synaptosome engulfment assays revealed that *Sirpa*-KO microglia engulfed more wild-type synaptosomes than did wild-type microglia, but both types of microglia consumed similar levels of *Cd47*-KO synaptosomes. Furthermore, wild-type microglia preferentially consumed *Cd47*-KO synaptosomes in assays containing both these and wild-type synaptosomes. Together, these data suggest that CD47 protects synapses from being engulfed by binding to microglial SIRPα.

The authors had previously shown that microglia preferentially engulf less-active inputs in the dLGN. Interestingly, here, the authors found that CD47 was more abundant at more active inputs, and that differential stimulation of RGC inputs had no effect on engulfment by *Cd47*-KO microglia. Thus, CD47 seems to protect active synapses from engulfment.

Together, these data show that synaptic CD47 acts as an activity-regulated 'don't eat me' signal during developmental synaptic refinement, by binding to its receptor on microglia.

Darran Yates

ORIGINAL ARTICLE Lehrman, E. K. et al. CD47 protects synapses from excess microglia-mediated pruning during development. *Neuron* https://doi.org/10.1016/j.neuron.2018.09.017 (2018)

IN BRIEF

SYNAPTIC TRANSMISSION

CA2 bursting

Oxytocin receptors (OXTRs) are expressed in hippocampal CA2; however, their functions in this region are unknown. Here, the authors combined optogenetics and electrophysiology to determine that activation of CA2 pyramidal cell OXTRs alters the shape of the spikes generated by these neurons, reducing their peak amplitude and after-hyperpolarization. This effect is the result of OXTR-driven stimulation of phospholipase C, which in turn activates KCNQ-containing M-channels and the protein kinase C pathway, both of which influence spike shape. The increase in CA2 bursting that results has implications for CA2–CA1 neurotransmission and plasticity.

ORIGINAL ARTICLE Tirko, N. N. et al. Oxytocin transforms firing mode of CA2 hippocampal neurons. *Neuron* https://doi.org/10.1016/j.neuron.2018.09.008 (2018)

NEUROREGENERATION

Boosting regeneration

Direct electrical stimulation of neural tissue close to a site of peripheral nerve injury has been shown to enhance regeneration. Here, the authors developed a bioresorbable, wireless electrical stimulator, which they secured to the rodent sciatic nerve and connected to a wireless receiver implanted subcutaneously. Passing radio frequency power through a transmission antenna placed near the receiver generated electrical impulses, the temporal pattern of which could be controlled. Rates of recovery following sciatic nerve transection were increased in rats receiving electrical stimulation compared with controls, suggesting that neuroregenerative bioelectronic approaches may have therapeutic potential.

ORIGINAL ARTICLE Koo, J. et al. Wireless bioresorbable electronic system enables sustained nonpharmacological neuroregenerative therapy. *Nat. Med.* https://doi.org/10.1038/s41591-018-0196-2 (2018)

SPATIAL NAVIGATION

Hexagonal pathfinding

In all animals, spatial navigation involves building a neural representation of the environment in a process mediated by cells of the hippocampal formation, including grid cells. Human functional MRI studies have detected grid-like signals in the entorhinal cortex during visual exploration. Staudigl et al. investigated visual space encoding in 35 healthy people viewing natural scenes by simultaneously recording eye-tracking data and hippocampal activity (via magnetoencephalography (MEG) or intracranial electroencephalography (iEEG)). Both MEG and iEEG data indicated that visual space is encoded by grid-like hexadirectional modulation of activity in the high frequency range (60–120 Hz). In a separate study, Chen et al. focused on low frequency oscillations in the theta band (4–8 Hz), performing iEEG recordings from the entorhinal cortex in people with epilepsy while they performed a virtual object-location memory task. They found that changes in movement direction induced a hexadirectional rotational modulation of theta power, which stabilized over time and showed sensitivity to boundaries in the environment. Together, these studies suggest that both high frequency and theta band oscillations in the human entorhinal cortex code space in a grid-like manner.

ORIGINAL ARTICLES Staudigl, T. et al. Hexadirectional modulation of high-frequency electrophysiological activity in the human anterior medial temporal lobe maps visual space. *Curr. Biol.* https://doi.org/10.1016/j.cub.2018.09.035 (2018) | Chen, D. et al. Hexadirectional modulation of theta power in human entorhinal cortex during spatial navigation. *Curr. Biol.* https://doi.org/10.1016/j.cub.2018.08.029 (2018)