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IN BRIEF

NEURODEGENERATIVE DISEASE

Clearing away Alzheimer's disease

The behavioural deficits and neurodegeneration that are observed in late-onset Alzheimer's disease are linked to high levels of amyloid-β (Aβ) in the brain, which result from a failure of Aβ clearance mechanisms. Here, the authors treated mice exhibiting Aβ-associated behavioural deficits and pathology with the retinoid X receptor (RXR) agonist bexarotene. RXR activation induces the expression of apolipoprotein E, which facilitates Aβ removal from the brain. Bexarotene treatment rapidly lowered soluble and insoluble levels of brain Aβ and improved cognitive, behavioural and olfactory deficits, indicating that RXR agonists may be useful in the treatment of Alzheimer's disease.

ORIGINAL RESEARCH PAPER Cramer, P. E. *et al.* ApoE-directed therapeutics rapidly clear β-amyloid and reverse deficits in AD mouse models. *Science* 9 Feb 2012 (doi:10.1126/science.1217697)

NEURAL DEVELOPMENT

Nogo to synapse formation

A new study reveals that Nogo receptors (NgRs) have key roles in restricting synapse formation. Wills *et al.* showed that loss of all three NgRs in mice promoted excitatory synapse formation, whereas overexpression of NgR1 in hippocampal slices decreased synaptic density. Furthermore, they showed that NgRs seemed to specifically limit synapse addition, partly through limiting dendritic growth. Interestingly, Wills *et al.* found that NgR expression was downregulated by neuronal activity, possibly facilitating activity-dependent synapse formation.

ORIGINAL RESEARCH PAPER Wills, Z. P. *et al.* The Nogo receptor family restricts synapse number in the developing hippocampus. *Neuron* **73**, 466–481 (2012)

LEARNING AND MEMORY

Memory consolidation gets murky

Memory consolidation is widely understood to involve *de novo* protein synthesis, as the administration of protein synthesis inhibitors such as anisomycin and cyclohexamide prevents the formation of long-term memory following initial learning. A new study shows that in anaesthetized rats, intrahippocampal infusion of anisomycin or cyclohexamide reduces (and can even abolish) overall hippocampal electrical activity. These findings indicate that the reported effects of protein synthesis inhibitors on memory consolidation may have been confounded by their silencing effects on neural activity.

ORIGINAL RESEARCH PAPER Sharma, A. V., Nargang, F. E. & Dickson, C. T. Neurosilence: profound suppression of neural activity following intracerebral administration of the protein synthesis anisomycin. *J. Neurosci.* **32**, 2377–2387 (2012)

SYNAPTIC PLASTICITY

Direct involvement for astrocytes in LTP?

Here, Navarrete *et al.* explored the direct involvement of astrocytes in a specific form of synaptic plasticity *in vivo*. In anaesthetized rats, tail-pinch-induced cholinergic activity led to calcium elevations in hippocampal astrocytes and long-term potentiation (LTP) of CA3–CA1 synapses. Interestingly, LTP required metabotropic glutamate receptor activation downstream of the astrocyte calcium signal. In rat hippocampal slices, stimulation of Schaffer collaterals in CA1 recapitulated the events observed *in vivo* and downregulation of the astrocyte calcium signal prevented LTP. Thus, astrocyte-derived glutamate release may be a necessary component of cholinergic signalling-induced plasticity of hippocampal synapses.

ORIGINAL RESEARCH PAPER Navarrete, M. *et al.* Astrocytes mediate in vivo cholinergic-induced synaptic plasticity. *PLoS Biol.* **10**, e1001259 (2012)