IN BRIEF

SYNAPSE FORMATION

Thrombospondin 1 accelerates synaptogenesis in hippocampal neurons through neuroligin 1

Xu, J. *et al. Nature Neurosci.* 15 Nov 2009 (doi:10.1038/nn.2459)

Thrombospondin 1 (THBS1), secreted by astrocytes, has been shown to promote synaptogenesis in some neurons; however, the cellular mechanisms underlying its effects were largely undetermined. The authors showed that THBS1 increases the rate of the early stages of synapse formation in cultured rat hippocampal neurons. This effect was inhibited when the interaction between THBS1 and neuroligin 1 (NL1) was blocked or when NL1 expression was knocked down, suggesting that some of the synaptogenic effects of THBS1, like those of neurexins, are mediated by NL1.

LEARNING AND MEMORY

Nogo receptor 1 regulates formation of lasting memories

Karlén, A. *et al. Proc. Natl Acad. Sci. USA* 13 Nov 2009 (doi:10.1073/PNAS.0905390106)

Previous studies aimed at elucidating the molecular regulators of long-term memory had shown that neuronal activity decreases the expression of Nogo-receptor 1 (NgR1; also known as RTN4R) in brain regions associated with memory. Here, the authors showed that preventing NgR1 downregulation by overexpressing it in adult mice had no effect on long-term potentiation or short-term memory but impaired long-term memory. These findings suggest that activity-driven downregulation of NgR1 promotes the formation of long-lasting memories.

NEUROLOGICAL DISORDERS

Balance between synaptic versus extrasynaptic NMDA receptor activity influences inclusions and neurotoxicity of mutant huntingtin

Okamoto, S.-I. *et al. Nature Med.* 15 Nov 2009 (doi:10.1038/nm.2056)

Huntington's disease is caused by an expansion of the CAG repeat in the gene encoding huntingtin (HTT). Here the authors investigate what makes striatal and cortical neurons particularly vulnerable to the effects of mutant HTT. In a mouse model of the disease, synaptic *N*-methyl-D-aspartate receptor (NMDAR) activation triggered the formation of mutant-HTT inclusions that made neurons more resistant to death. By contrast, stimulation of extrasynaptic NMDARs increased neuronal vulnerability by preventing inclusion formation. Selective inhibition of extrasynaptic NMDARs could be an attractive therapeutic strategy in Huntington's disease.

DEVELOPMENT

Regulation of radial glial motility by visual experience

Tremblay, M. *et al. J. Neurosci.* **29**, 14066–14076 (2009)

In the developing optic tectum, radial glia have a key role in guiding retinal axons. By using multiphoton live imaging in *Xenopus laevis* tadpoles, the authors show for the first time that visual stimulation influences the motility of radial glia. Far from acting as mere scaffolds, these cells exhibit neuronal-activity-dependent Ca^{2+} transients and undergo rapid structural remodelling in response to *N*-methyl-D-aspartate receptor (NMDAR) activation in the tectum. Whether this responsiveness contributes to experience-dependent topographic map formation and plasticity remains to be determined.