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IN BRIEF

TRANSPLANTATION

Bridging the gap

In this study, Lu *et al.* investigated the ability of neural stem cells (NSCs) to repair the spinal cord following injury. Embryonic rat NSCs or human stem cell lines were embedded into fibrin matrices containing various growth factors and were grafted at spinal lesion sites in adult rats. After 7 weeks, around one-third of the grafted cells had differentiated into neurons, many of which extended axons that formed functional synapses with host neurons. Furthermore, axon outgrowth was found to be dependent on mammalian target of rapamycin signalling. Importantly, electrophysiological analysis suggested synaptic transmission and functional recovery of spinal cord relay circuits.

ORIGINAL RESEARCH PAPER Lu, P. *et al.* Long-distance growth and connectivity of neural stem cells after severe spinal cord injury. *Cell* **150**, 1264–1273 (2012)

STEM CELLS

Prolactin unlocks stem cell latency

Activation of endogenous neurogenesis has therapeutic potential for neurodegenerative conditions. Walker *et al.* found that neurospheres of latent precursor cells derived from adult mouse hippocampus increased in number by 50% following exposure to the peptide hormone prolactin (PRL). Conversely, PRL-null mice had fewer hippocampal-derived neurospheres and showed learning and memory deficits, suggesting a link between the influence of PRL on cell proliferation and behaviour.

ORIGINAL RESEARCH PAPER Walker, T. L. *et al.* Prolactin stimulates precursor cells in the adult mouse hippocampus. *PLoS ONE* **7**, e44371 (2012)

SYNAPTIC PHYSIOLOGY

Meeting point for autism and fragile X syndrome

The changes in synaptic physiology that accompany fragile X syndrome (FXS) and autism are poorly understood, but Baudouin *et al.* provide evidence that the synaptic protein neuroligin 3 (NLGN3) might be involved in both. Non-syndromic autism (in which autism is the primary disorder) can be associated with defects in NLGN3. The authors showed that cerebellar Purkinje cells of NLGN-null mice exhibited a loss of long-term depression mediated by metabotrophic glutamate receptors. This type of plasticity is also associated with FXS and suggests that NLGN3-related synaptic mechanisms may be involved in both FXS and autism.

ORIGINAL RESEARCH PAPER Baudouin, S. J. *et al.* Shared synaptic pathophysiology in syndromic and nonsyndromic rodent models of autism. *Science* 13 Sep 2012 (doi:10.1126/science.1224159)

ION CHANNELS

No REST for NMDA receptors

During development, NMDA receptors consist primarily of NR2B, but a few weeks after birth there is a switch to NR2A-containing receptors. The trigger for this switch is unknown, but Rodenas-Ruano *et al.* show that at developing rat hippocampal synapses, NMDA receptor genes are subject to transcriptional control by repressor element 1 silencing transcription factor (REST). Analysis of dentate granule cells revealed that REST becomes activated between postnatal day 15 (p15) and p60 and acts to repress *GRIN2B* (the gene that encodes NR2B). Interestingly, maternal deprivation reduced REST activation and prevented the switch in NMDA receptor phenotype, suggesting an involvement in experience-dependent synaptic plasticity.

ORIGINAL RESEARCH PAPER Rodenas-Ruano, A. *et al.* REST-dependent epigenetic remodeling promotes the developmental switch in synaptic NMDA receptors. *Nature Neurosci.* **15**, 1382–1390 (2012)