IN BRIEF

NEURAL STEM CELLS

Communication via gap junctions underlies early functional and beneficial interactions between grafted neural stem cells and the host

Jäderstad, J. *et al. Proc. Natl Acad. Sci. USA* 10 February 2010 (doi: 10.1037/pnas.0915134107)

How do grafted neural stem cells (NSCs) interact with existing neurons? NSCs transplanted into the cerebellum of mouse models of Purkinje cell death formed functional connexin 43-containing gap junctions with existing neurons. The grafts influenced host network activity, increased host neuron survival and rescued behavioural deficits. These effects could be inhibited by blocking gap junction formation or function, suggesting that NSC–neuron communication through gap junctions has modulatory and protective actions.

CIRCADIAN RHYTHMS

The cerebellum harbors a circadian oscillator involved in food anticipation

Mendoza, J. *et al. J. Neurosci.* **30**, 1894–1904 (2010)

Animals kept on a restricted feeding schedule show food-anticipatory behavioural and physiological rhythms, but the location of a 'food-entrainable' clock has remained elusive. The authors found that the clock gene *Per1* showed circadian expression in cerebellar slices, indicating intrinsic cerebellar circadian rhythmicity. In mice subjected to restricted feeding, cerebellar expression of several clock genes shifted. This effect was absent in mice with Purkinje cell loss or abnormalities, which also showed reduced food-anticipatory activity. These findings suggest that the cerebellum contains a food-entrainable oscillator that involves Purkinje cells.

LEARNING AND MEMORY

Forgetting is regulated through Rac activity in *Drosophila*

Shuai, Y. *et al. Cell* **140**, 579–589 (2010)

To determine the molecular mechanism for forgetting newly formed memories, the authors used fruitflies undergoing olfactory aversive conditioning. Expressing a dominant-negative form of the Rho GTPase *Rac* in mushroom body neurons slowed both passive decay and interference-induced forgetting of early memory. The resulting prolonged memory was not blocked by protein synthesis inhibitors, indicating that it is distinct from long-term memory. Furthermore, RAC activity correlated with memory removal during reversal learning.

APOPTOSIS

Suppression of the intrinsic apoptosis pathway by synaptic activity

Léveillé, F. *et al. J. Neurosci.* **30**, 2623–2635 (2010)

Synaptic activity protects neurons against oxidative stress-induced apoptosis. Here, the authors show in cortical cultures that it also protects against other apoptotic insults by suppressing the expression of the pro-apoptotic gene p53 upregulated modulator of apoptosis (*Puma*) in an NMDA (*N*-methyl-D-aspartate)-dependent manner. Neuronal activity also inhibited apoptotic processes downstream of cytochrome *c* release, by directly suppressing the expression of key components of the apoptotic pathway.