IN BRIEF

NEUROLOGICAL DISORDERS

IKK mediates ischemia-induced neuronal death.

Herrmann, O. *et al. Nature Med.* 13 November 2005 (10.1038/nm1323)

The IκB kinase complex IKK is a central component of the signalling cascade that controls nuclear factor-κB-dependent gene transcription. Its function in the brain is largely unknown. Herrmann *et al.* show that IKK is activated in a mouse model of stroke. Constitutive activation of IKK increases the infarct size, whereas interference with IKK2 function in neurons reduces ischaemic brain damage. A small-molecule inhibitor of IKK can mimic this effect, which indicates that this class of IKK inhibitors could provide a new neuroprotective strategy.

CELL BIOLOGY OF THE NEURON

The cell cycle–apoptosis connection revisited in the adult brain.

Bauer, S. & Patterson, P. H. *J. Cell Biol.* **171**, 641–650 (2005)

Adult neurogenesis is studied using thymidine analogues such as bromodeoxyuridine (BrdU) to label DNA synthesis. However, it is not clear whether BrdU also labels DNA synthesis events not directly related to cell proliferation, such as DNA repair and/or abortive re-entry into the cell cycle, which can occur as part of an apoptotic process in postmitotic neurons. Using three well-characterized models of injury-induced neuronal apoptosis, Bauer and Patterson found that BrdU is not significantly incorporated during DNA repair and that labelling is undetectable in dying postmitotic neurons.

GLIA

Involvement of aquaporin-4 in astroglial cell migration and glial scar formation.

Saadoun, S. *et al. J. Cell Sci.* 22 November 2005 (10.1242/jcs.02680)

Aquaporin 4 (AQP4), the main water-selective channel in astroglia throughout the CNS, facilitates water movement into and out of the brain. The authors report a novel role for AQP4 in astroglia migration and glial scar formation. Astroglia cultured from the neocortex of AQP4-knockout mice had normal morphology as well as proliferative and adhesive activity, but their migratory ability was markedly impaired. In AQP4-knockout mice, glial scar formation was significantly attenuated, with reduced migration of reactive astroglia towards the site of injury.

COGNITIVE NEUROSCIENCE

DCDC2 is associated with reading disability and modulates neuronal development in the brain.

Meng, H. *et al. Proc. Natl Acad. Sci. USA* 22 November 2005 (10.1073/pnas.0508591102)

The DYX2 on chromosome 6p22, which contains ~19 genes, is the most replicated reading disability locus. Meng *et al.* have identified a large polymorphic deletion of DCDC2, which lies on this locus, in families with reading disabilities. DCDC2 localizes to regions of the human brain responsible for fluent reading, and interference of DCDC2 expression in rat embryos causes defects in neuronal migration during development. The authors propose that DCDC2 is a candidate gene for reading disabilities.