IN BRIEF

NEURODEVELOPMENTAL DISORDERS

Developmental delays

Mutations in the gene encoding fragile X mental retardation protein (FMRP), an RNA-binding protein expressed during brain development, lead to fragile X syndrome (FXS). Bagni and colleagues show that, in embryonic mice, loss of FMRP delays the migration of new-born neurons to the cortical plate by reducing N-cadherin levels. This delay results in an imbalance in excitatory and inhibitory activity and altered structural connectivity in the postnatal brain. Thus, disrupted FMRP activity in early embryonic brain development may contribute to altered network connectivity in FXS.

ORIGINAL RESEARCH PAPER La Fata, G. *et al.* FMRP regulates multipolar to bipolar transition affecting neuronal migration and cortical circuitry. *Nature Neurosci.* **17**, 1693–1700 (2014)

BRAIN AGEING

Last in, first out?

It has been proposed that the last parts of the brain to mature are the first to degenerate as we age. By analysing structural MRI data from 484 healthy participants aged from 8 to 85 years, Douaud *et al.* now provide direct evidence for this assertion. They reveal a network of brain regions that develop late in adolescence and degenerate first in old age. Furthermore, this network overlaps with those areas that are most vulnerable to changes in Alzheimer's disease or schizophrenia.

ORIGINAL RESEARCH PAPER Douaud, G. *et al.* A common brain network links development, aging, and vulnerability to disease. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1410378111 (2014)

SYNAPTIC PHYSIOLOGY

Human efficiency

In terms of cognitive ability, humans outperform other species, suggesting that our brains possess enhanced information processing capacity. Testa-Silva *et al.* here show that human synapses can indeed relay information with high efficiency. They recorded from pairs of pyramidal neurons in human and mouse cortical slices during the transfer of an action potential train and found that human neurons recovered much faster from use-dependent depression than mouse neurons. Using information theory, they showed that human synapses have a greater dynamic range of responses to changes in presynaptic activity and better temporal resolution than those of mice.

ORIGINAL RESEARCH PAPER Testa-Silva, G. *et al.* High bandwidth synaptic communication and frequency tracking in human neocortex. PLoS Biol. **12**, e1002007 (2014)

NEUROINFLAMMATION

Transport disruption in multiple sclerosis

Impaired axonal transport is implicated in the pathogenesis of many neurodegenerative diseases; but its potential role in the neuroinflammatory disease multiple sclerosis has not been determined. Using *in vivo* two-photon imaging, Sorbara *et al.* found widespread deficits in both anterograde and retrograde transport in acute and chronic mouse models of multiple sclerosis. These defects occurred even in 'normal'-appearing axons, suggesting that axonal transport deficits are an early event in disease pathogenesis. The defects were reversed by treatment with anti-inflammatory drugs or redox scavengers, suggesting that they might be a suitable target for therapeutic strategies.

ORIGINAL RESEARCH PAPER Sorbara, C. D. *et al.* Pervasive axonal transport deficits in multiple sclerosis models. *Neuron* http://dx.doi.org/10.1016/j.neuron.2014.11.006 (2014)