IN BRIEF

NEUROGENETICS

MED23 mutation links intellectual disability to dysregulation of immediate early gene expression

Hashimoto, S. *et al. Science* **333**, 1161–1163 (2011)

MED23 protein is a component of the mediator complex and is involved in immediate early gene (IEG) transcription that occurs in response to serum mitogen signals. The authors identified a human *MED23* mutation that was associated with non-syndromic intellectual disability. Skin fibroblasts from patients with this mutation showed specific abnormalities in IEG transcription following serum exposure. Other neurological conditions that are associated with gene mutations in mediator subunits or mediator-interacting proteins exhibited similar deficits in transcription, suggesting that abnormal IEG expression might be a feature of mediator-linked intellectual disability.

GLIA

Individual axons regulate the myelinating potential of single oligodendrocytes in vivo

Almeida, R. G. *et al. Development* 31 Aug 2011 (doi:10.1242/dev.071001)

Almeida *et al.* used zebrafish to examine the role of axons in regulating myelination. These animals have two so-called Mauthner neurons, which have large-calibre axons, and the authors found that oligodendrocytes that myelinated such axons did not typically associate with other axons. In zebrafish that were engineered to have artificially high numbers of Mauthner neurons, however, many oligodendrocytes myelinated more than one Mauthner axon, indicating that axons regulate the myelinating potential of oligodendrocytes.

EMOTION

Glutamatergic and dopaminergic neurons mediate anxiogenic and anxiolytic effects of CRHR1

Refojo, D. *et al. Science* 1 Sep 2011 (doi:10.1126/science.1202107)

Corticotropin-releasing hormone receptor 1 (CRHR1) has been implicated in emotional disorders. To examine CRHR1's role in modulating anxiety in mice, the authors determined the expression pattern of CRHR1 in brain and assessed the behavioural effects of deleting *CRHR1* in subsets of neurons. Deletion of *CRHR1* in forebrain glutamatergic neurons led to a decrease in anxiety-like behaviour in mice, whereas deletion of this gene in midbrain dopaminergic neurons increased such behaviour. Thus, an imbalance in these neural systems may contribute to emotional disorders.

PSYCHIATRIC DISORDERS

The neural consequences of combat stress: long-term follow-up

van Wingen, G. A. *et al. Mol. Psychiatry* 30 Aug 2011 (doi:10.1038/mp.2011.110)

Previously, the authors showed that after returning from duty, troops who had experienced combat-induced stress had an increase in amygdala reactivity, and soldiers who reported a rise in perceived threat during deployment had an increase in coupling between the amygdala and the dorsal anterior cingulate cortex (dACC). In this long-term follow up, van Wingen *et al.* show that amygdala reactivity returns to pre-deployment levels ~1.5 years after having experienced combat. However, the altered amygdala–dACC coupling persists, indicating that severe stress can have long-term effects on the brain.