IN BRIEF

PSYCHIATRIC DISORDERS

Reversal of hippocampal neuronal maturation by serotonergic antidepressants

Kobayashi, K. *et al. Proc. Natl Acad. Sci. USA* **107**, 8434–8439 (2010)

The cellular mechanisms underlying the behavioural effects of serotonergic antidepressants are largely unknown. Here, the authors show that chronic treatment of adult mice with the selective serotonin reuptake inhibitor fluoxetine can reverse the phenotypic maturation of granule cells in the dentate gyrus. Granule cells from treated animals showed lower expression of the mature granule cell marker calbindin, reduced synaptic facilitation and altered membrane properties, indicating reversal of the late step of maturation. These findings uncover a novel mechanism of action for serotonergic antidepressants.

NEURONAL MIGRATION

The disintegrin/metalloproteinase ADAM10 is essential for the establishment of the brain cortex

Jorissen, E. *et al. J. Neurosci.* **30**, 4833–4844 (2010)

ADAM10 is thought to mediate the shedding of several cell surface proteins that are important for brain development, but its precise function remains unknown. In this study, the authors disrupted ADAM10 expression specifically in neural progenitors and found that this caused premature differentiation into postmitotic neurons. This led to aberrant neuronal migration during development and a complete disorganization of cortical layering. These results suggest that ADAM10 has a central role in the organization of the cortical region by regulating the differentiation of neural progenitors during development.

NEUROIMAGING

Noninvasive imaging of endogenous neural stem cell mobilization *in vivo* using positron emission tomography

Rueger, M. A. *et al. J. Neurosci.* **30**, 6454–6460 (2010)

Pharmacological stimulation to increase the endogenous pool of neural stem cells (NSCs) has been proposed as a potential method for repairing damaged neural tissue. In this study, the authors describe how endogenous NSCs can be visualized *in vivo* with positron emission tomography using the radiotracer 3′-deoxy-3′[^{18}F]fluoro-L-thymidine. They were also able to quantify the expansion of NSCs induced by pharmacological stimulation. This non-invasive technique holds great promise for the future development of endogenous NSC-based therapies.

NEUROLOGICAL DISORDERS

L-Histidine decarboxylase and Tourette's syndrome

Ercan-Sencicek, A. G. *et al. N. Engl. J. Med.* 5 May 2010 (doi:10.1056/NEJMoa0907006)

Tourette's syndrome is known to have a strong genetic component, but the identification of candidate genes has proven difficult owing to its complex pattern of inheritance. Here, the authors describe a linkage analysis in a family in which the syndrome is segregated in an autosomal-dominant fashion. They detected a mutation in the gene encoding L-histidine decarboxylase, the rate-limiting enzyme in histamine biosynthesis. These results suggest a disruption in histaminergic neurotransmission as a possible cause of Tourette's syndrome.