IN BRIEF

SYNAPTOGENESIS

Grb4 and GIT1 transduce ephrinB reverse signals modulating spine morphogenesis and synapse formation.

Sugura, I., Essmann, C. L., Weinges, S. & Acker-Palmer, A. *Nature Neurosci.* **10**, 301–310 (2007)

Eph receptors have a role in both spine formation and long-term plasticity. This study shows that their transmembrane ligands, ephrin Bs, initiate a signalling cascade in their host cell that regulates spine maturation and synaptogenesis in rat hippocampal neurons. Ephrin B1 signalling induces the formation of a complex with the SH2–SH3 adaptor protein GRB4, and the Rac regulating protein GIT1. The authors propose that by recruiting GIT1 to the synapse, ephrin B1 stimulates Rac activity, cytoskeleton remodelling and synapse formation.

EMOTION

Reversal of neurosteroid effects at $\alpha4\beta2\delta$ $GABA_A$ receptors triggers anxiety at puberty.

Shen, H. *et al. Nature Neurosci.* 11 March 2007 (doi:10.1038/nn1868)

The action of the neurosteroid tetrahydropregnanalone (THP) during puberty may explain the mood swings and anxiety experienced by teenagers. THP is produced in response to stress and may reduce anxiety by enhancing the inhibitory effects of $GABA_A$ receptors in the hippocampal CA1 region. Shen *et al.* now show that THP has the opposite effect on $\alpha_4\beta_2\delta$ $GABA_A$ receptors, a subtype that is highly expressed in mice during puberty. Thus, paradoxically, THP increases anxiety after the onset of puberty.

BRAIN EVOLUTION

Brain shape in human microcephalics and *Homo floresiensis*.

Falk, D. *et al. PNAS* **104**, 2513–2518 (2007)

The debate over whether LB1, the first specimen of the proposed new hominid species *Homo floresiensis*, was actually a microcephalic human remains unresolved, partly because there are few definitive criteria to classify a brain as microcephalic. The authors used three-dimensional computed tomographic reconstructions of the internal braincase based on the skulls of 9 microcephalics and 10 normal humans to identify two variables that can distinguish the groups with 100% success. The same criteria classified LB1 in the normal human group, providing further evidence that LB1 was not microcephalic.

NEUROLOGICAL DISORDERS

Reversal of neurological defects in a mouse model of Rett syndrome.

Guy, J. *et al. Science* **315**, 1143–1147 (2007)

Mutations in *MECP2* are the cause of the neuronal abnormalities underlying Rett syndrome. However, the affected neurons do not die, raising the question of whether replacement of normal *MECP2* activity might restore function to these cells. The authors used a transgenic mouse model in which *Mecp2* was silenced, but could be reactivated by tamoxifen treatment. Reactivation of *Mecp2* in mature mice with advanced symptoms reversed their neurological phenotype, suggesting that the neurolocial defects observed in humans with Rett syndrome and related disorders might not be irreversible.

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