IN BRIEF

CEREBRAL CORTEX

Childhood abuse alters cortical fields

A long-term consequence of childhood sexual abuse can be the development of sexual dysfunction in adulthood, although the underlying mechanisms are unknown. Using MRI, the authors found that individuals who had suffered childhood sexual abuse display cortical thinning in regions of the somatosensory cortex that represent the genitals. Emotional abuse correlated with thinning of cortical areas involved in self-awareness and evaluation. Thus, experience-dependent developmental neuroplasticity as a result of aversive childhood events may alter cortical structure in a regionally specific manner.

ORIGINAL RESEARCH PAPER Heim, C. M. *et al.* Decreased cortical representation of genital somatosensory field after childhood sexual abuse. *Am. J. Psychiatry* **170**, 616–623 (2013)

NEURAL DEVELOPMENT

Compartmentalized calcium triggers pruning

During development, the refinement of neural circuitry involves the removal of specific dendritic branches, but how this selective elimination is mediated is unclear. The authors monitored changes in levels of intracellular calcium before and during dendritic pruning in *Drosophila melanogaster* sensory neurons. They found that localized changes in excitability in specific dendritic branches result in the generation of compartmentalized calcium transients that trigger pruning of those branches through mechanisms that involve activation of the calcium-activated protease calpain.

ORIGINAL RESEARCH PAPER Kanamori, T. *et al.* Compartmentalized calcium transients trigger dendrite pruning in *Drosophila* sensory neurons. *Science* 30 May 2013 (doi:10.1126/science.1234879)

NEURODEGENERATIVE DISEASE

TDP43 mutation in astrocytes is neurotoxic

Mutations in TAR DNA-binding protein 43 (TDP43) are associated with amyotrophic lateral sclerosis; however, the precise mechanisms of TDP43-mediated pathogenesis are unclear. The authors show that the specific expression of mutant TDP43 in spinal astrocytes is sufficient to cause progressive motor neuron degeneration, muscular atrophy and paralysis in rats, through mechanisms involving downregulation of 'neuroprotective' genes such as those encoding glutamate transporters and upregulation of 'neurotoxic' genes such as lipocalin 2 (*Lcn2*).

ORIGINAL RESEARCH PAPER Tong, J. *et al.* Expression of ALS-linked TDP-43 mutant in astrocytes causes non-cell-autonomous motor neuron death in rats. *EMBO J.* 28 May 2013 (doi:10.1038/emboj.2013.122)

SENSORY SYSTEMS

Investigating itch circuitry

Itch-inducing stimuli are detected by somatosensory neurons expressing the ion channel transient receptor potential V1 (TRPV1); however, the circuitry that mediates itch signalling has not yet been elucidated. Here, the authors showed that a subset of TRPV1 neurons express natriuretic polypeptide B (NPPB) and that loss of NPPB, or of its spinal receptor, specifically abolishes responses to itch-inducing agents in mice. They found that NPPB acts as a specific itch-related neurotransmitter to relay information from peripheral itch-sensitive neurons to the spinal cord.

ORIGINAL RESEARCH PAPER Mishra, S. K. & Hoon, M. A. The cells and circuitry for itch responses in mice. *Science* **340**, 968–971 (2013)