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IN BRIEF

EPIGENETICS

Timing female puberty

Neuroendocrine changes initiate female reproductive function but little is known about the mechanisms that determine the onset of puberty. The authors identify an epigenetic mechanism of transcriptional repression that regulates the timing of female puberty in rats. They found that increased methylation of the promoter region of two genes encoding Polycomb group family members reduced their expression at the onset of puberty, allowing the expression of *Kiss1*, which controls the release of gonadotropin-releasing hormone from neurosecretory neurons in the hypothalamus.

ORIGINAL RESEARCH PAPER Lomniczi, A. *et al.* Epigenetic control of female puberty. *Nature Neurosci.* 27 Jan 2013 (doi:10.1038/nn.3319)

NEURODEVELOPMENTAL DISORDERS

Lovastatin as fragile X therapy

Fragile X syndrome (FXS) is caused by loss of the fragile X mental retardation 1 gene product, a repressor of mRNA translation. It is thought that excessive protein synthesis downstream of metabotropic glutamate receptor 5 activation leads to many of the neuropsychiatric symptoms of FXS. The authors show that lovastatin, a widely prescribed cholesterol-lowering drug, can normalize protein synthesis in the hippocampus of a mouse model of FXS and prevent epileptogenesis. Although the effects of the drug on the neurocognitive phenotypes of these mice remain to be investigated, lovastatin could be a promising therapy for FXS.

ORIGINAL RESEARCH PAPER Osterweil, E. K. *et al.* Lovastatin corrects excess protein synthesis and prevents epileptogenesis in a mouse model of fragile X syndrome. *Neuron* **77**, 243–250 (2013)

SENSORY SYSTEMS

Stroke-sensitive neurons uncovered

Sensory neurons that respond to pleasant stimulation of the skin are not as well characterized as those that respond to noxious stimulation. Using calcium imaging in live mice, the authors of this study show that neurons in the skin that express the G protein-coupled receptor MRGPRB4 respond to pleasant stroking but not to pinching or poking stimuli. Pharmacological activation of these neurons promoted a preference for the location in which the activation occurred, suggesting that these stimuli have a positive affective valence. Further characterization of this population of neurons may shed light on the neural circuitry and signalling pathways associated with pleasure.

ORIGINAL RESEARCH PAPER Vrontou, S. *et al.* Genetic identification of C fibres that detect massage-like stroking of hairy skin *in vivo*. *Nature* **493**, 669–673 (2013)

PLASTICITY

The power of silence

Previous studies have shown that blocking neural activity with tetrodotoxin (TTX) leads to an increase in synaptic strength. The authors further examined the effects of TTX treatment on Schaffer collateral–CA1 synapses in cultured hippocampal slices and found that chronic activity blockade leads to the formation of new, silent synapses (lacking AMPA receptors). Furthermore, induction of long-term potentiation activated these silent synapses by inducing AMPA receptor insertion. Thus, neural networks can compensate for the lack of synaptic activity by increasing the synaptic strength of existing synapses and by promoting the emergence of new synapses.

ORIGINAL RESEARCH PAPER Arendt, K. L. Sart, F. & Chen, L. Chronic inactivation of a neural circuit enhances LTP by inducing silent synapse formation. *J. Neurosci.* **33**, 2087–2096 (2013)