IN BRIEF

GLUCOSE HOMEOSTASIS

Serotonin 2C receptor agonists improve type 2 diabetes via melanocortin-4 receptor signaling pathways

Zhou, L. *et al. Cell Metab.* **6**, 398–405 (2007)

Glucose homeostasis is in part regulated by central brain pathways, suggesting that these could be targeted to treat type 2 diabetes. Here the authors showed that serotonin 2C receptor agonists can improve glucose homeostasis in mouse models of obesity and in patients with type 2 diabetes, and that these effects are mediated by a pathway that involves the activation of melanocortin 4 receptors in the hypothalamus.

GENETICS

Mutations in ionotropic AMPA receptor 3 alter channel properties and are associated with moderate cognitive impairment in humans

Wu, Y. *et al. Proc. Natl Acad. Sci. USA* **104**, 18163–18168 (2007)

A new study has added another example to the list of genetic mutations that are associated with cognitive impairments. The authors examined males with X-linked mental retardation, and identified one individual with a deletion of *GRIA3*, the gene that encodes a subunit of AMPA receptors, and another with four missense mutations in *GRIA3*. Further work revealed that the mutations resulted in misfolding of the protein and altered ion-channel properties.

SYNAPTIC PLASTICITY

Kalirin-7 controls activity-dependent structural and functional plasticity of dendritic spines

Xie, Z. *et al. Neuron* **56**, 640–656 (2007)

The importance of changes in the shape and size of dendritic spines for synaptic plasticity has become apparent; however, it was unknown how *N*-methyl-D-aspartate receptor (NMDAR) activity regulates actin dynamics to induce structural plasticity. Here the authors showed that, in cultured pyramidal neurons, activated NMDARs stimulate calcium/calmodulin-dependent protein kinase II, which phosphorylates the guanine exchange factor kalirin 7. They showed that kalirin 7 expression and localization in spines is required for spine maintenance and NMDAR-mediated changes in synaptic transmission. Kalirin 7's effects include the activation of Rac1, a key regulator of actin and glutamate-receptor clustering.

NEURODEGENERATIVE DISORDERS

Alzheimer's disease peptide epitope vaccine reduces insoluble but not soluble/oligomeric Aβ species in amyloid precursor protein transgenic mice

Petrushina, I. *et al. J. Neurosci.* **27**, 12721–12731 (2007)

The first clinical trial of an anti-Aβ vaccine for Alzheimer's disease was halted when patients developed meningoencephalitis, thought to be due to an autoimmune response. Here the authors describe the effects of an epitope vaccine containing two copies of $A\beta_{1-11}$ fused to a non-self T cell epitope in APP Tg2576 mice. The vaccine stimulated the generation of antibodies against $A\beta_{1-11}$, without stimulating autoreactive T cells. High antibody titres were associated with reduced amyloid plaques but no adverse events and no changes in soluble Aβ species, suggesting that this might be a safer immunotherapeutic approach.