IN BRIEF

NEURODEGENERATIVE DISEASE

Inhibition of amyloid-β (Aβ) peptide-binding alcohol dehydrogenase–Aβ interaction reduces Aβ accumulation and improves mitochondrial function in a mouse model of Alzheimer's disease

Yao, J. *et al. J. Neurosci.* **31**, 2313–2320 (2011)

Amyloid-β-mediated cell stress can be potentiated through the interaction of this peptide with amyloid-β peptide-binding alcohol dehydrogenase (ABAD), which is expressed in neuronal mitochondria. In this study, targeted delivery of ABAD decoy peptides to mitochondria in a transgenic mouse model of Alzheimer's disease inhibited amyloid-β–ABAD complex formation, increased mitochondrial function and improved spatial memory. Thus, such complexes might be targets for future drug development in Alzheimer's disease.

METABOLISM

Neuropeptide exocytosis involving synaptotagmin-4 and oxytocin in hypothalamic programming of body weight and energy balance

Zhang, G. *et al. Neuron* **69**, 523–535 (2011)

Whether dysregulation of synaptic neuropeptide exocytosis from hypothalamic neurons leads to obesity is unknown. Here, mice fed a high-fat diet became obese and had an increase in binding between synaptotagmin 4 and synaptic vesicles in hypothalamic oxytocin-positive neurons. Obesity was also associated with a decrease in oxytocin release from such cells. These data support central roles for synaptotagmin 4 and hypothalamic oxytocin exocytosis in the regulation of body weight.

LEARNING AND MEMORY

Astrocyte–neuron lactate transport is required for long-term memory formation

Suzuki, A. *et al. Cell* **144**, 810–823 (2011)

Unlike neurons, astrocytes can store glycogen, which can be broken down into lactate, a potential source of fuel for both cell types. In a study involving rats, Suzuki *et al.* showed that long-term memory formation was associated with a rise in extracellular lactate in the hippocampus, and that inhibition of glycogen breakdown impaired long-term potentiation. Disruption of lactate transport out of astrocytes or into neurons led to amnesia, further supporting a role for astrocyte-derived lactate in the formation of long-term memory.

STEM CELLS

Fat cells reactivate quiescent neuroblasts via TOR and glial insulin relays in *Drosophila*

Sousa-Nunes, R., Yee, L. L. & Gould, A. P. *Nature* 23 Feb 2011 (doi:10.1038/nature09867)

Various progenitor and stem cells can enter transient periods of mitotic quiescence, but the signals underlying transition to and from these dormant phases are unclear. Here, reactivation of neural progenitor cells from *Drosophila melanogaster* was dependent on amino acid-induced signals that originate in fat body cells and activate target of rapamycin signalling. Moreover, insulin-like peptides produced by glia were crucial in relaying this reactivation signal. Thus, stem cell behaviour might be influenced by nutrients as well as other cell types.