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IN BRIEF

REWARD

Loving food too much

Overconsumption of so-called highly palatable (calorie-dense) foods is a contributing factor in obesity. Little is known, however, about the role of the reward system in this overconsumption. In this study, female participants learned to associate a cue with a food reward or a financial reward. When compared with controls, obese participants were found to have impairments in reward-based associative learning related to food but not to financial rewards, suggesting that altered food-based learning might play a part in obesity.

ORIGINAL RESEARCH PAPER Zhang, Z. *et al.* Impaired associative learning with food rewards in obese women. *Curr. Biol.* http://dx.doi.org/10.1016/j.cub.2014.05.075 (2014)

SYNAPTIC PLASTICITY

Motility movers and shakers

Astrocytic perisynaptic processes (PAPs), which envelope excitatory synapses, respond to long-term potentiation (LTP) induction by modifying their morphology, but the underlying mechanisms are unclear. Here, the morphological changes in PAPs were shown to depend on increases in intracellular calcium levels and activation of astrocytic metabotropic glutamate receptors. Furthermore, whisker stimulation increased PAP motility in the somatosensory cortex, suggesting that signalling between neurons and astrocytes regulates PAP structural plasticity.

ORIGINAL RESEARCH PAPER Bernardinelli, Y. *et al.* Activity-dependent structural plasticity of perisynaptic astrocytic domains promotes excitatory synapse stability. *Curr. Biol.* http://dx.doi.org/10.1016/j.cub.2014.06.025 (2014)

NEURODEVELOPMENTAL DISORDERS

Brain–gut connection in autism?

In a new study, resequencing the genomes of several thousands of children with autism spectrum disorders (ASDs) or developmental delay revealed a cohort of children with a diagnosis of ASD that carried mutations in chromodomain helicase DNA binding-protein 8 (*CHD8*). *CHD8* mutations were also associated with an increased likelihood of certain other phenotypic markers of ASD, including increased head size and gastrointestinal (GI) dysfunction. Modelling Chd8 dysfunction in zebrafish produced both increased head size and GI motility problems. Thus, *CHD8* mutations could result in a genetically based subtype of ASD in which both the brain and gut are affected.

ORIGINAL RESEARCH PAPER Bernier, R. *et al.* Disruptive *CHD8* mutations define a subtype of autism early in development. *Cell* **158**, 263–276 (2014)

NEURODEGENERATIVE DISEASE

Faulty splicing in Huntington's disease

Huntington's disease (HD) is caused by an expanded CAG repeat in huntingtin (*HTT*) and is characterized by tau deposition. Tau can be alternatively spliced to produce isoforms containing either three or four microtubule-binding repeats, and CAG repeats have been shown to induce aberrant alternative splicing in favour of the four-repeat isoform, which is sufficient to induce neurodegeneration. The authors found that human HD brains had increased levels of the four-repeat tau isoform as well as higher overall levels of tau. These findings suggest that dysregulated alternative splicing plays an important part in HD pathology.

ORIGINAL RESEARCH PAPER Fernández-Nogales, M. *et al.* Huntington's disease is a four-repeat tauopathy with tau nuclear rods. *Nature Med.* http://dx.doi.org/10.1038/nm.3617 (2014)

CORRECTION

Brain–gut connection in autism?

Sian Lewis

Nature Reviews Neuroscience http://dx.doi.org/10.1038/nrn3806 (2014)

The ministrapline used for this In Brief article should have been 'Neurodevelopmental disorders'. This has been corrected in the online version.