LEARNING AND MEMORY

Neurotrypsin down memory lane

Mental retardation in humans is caused by various factors, including mutations in single genes. Many of these genes have orthologues in the fruit fly *Drosophila melanogaster*, and Didelot *et al.* have shown that one of these is crucial for the formation of long-term memory.

Tequila (TEQ) is a serine protease in the fly, and is the orthologue of human neurotrypsin. Mutations in neurotrypsin can lead to 'non-syndromic' mental retardation (where there are no apparent defects in neural development and no other symptoms). The authors therefore investigated whether TEQ is involved in long-term memory in the fly.

The type of memory they tested is called 'odour avoidance'. Flies are repeatedly exposed to two odours. One odour is always accompanied by an electric shock, so that the flies learn to avoid it. Repeated training sessions with rests in between them lead to long-term memory formation, which requires protein synthesis, whereas continuous 'massed training' leads to the formation of a type of memory that does not require new proteins to be made.

Flies carrying a mutation in the *teq* gene that reduces TEQ expression showed a reduced ability to form long-term memories, although their performance after massed training and on short-term memory tests was normal. This pattern of memory defect also occurred in flies in which RNA interference was used to suppress *teq* expression specifically in the mushroom bodies, which are crucial for olfactory learning and memory.

But these results do not tell us whether the effects of the TEQ mutation result from defects in the process of memory formation in the adult or from abnormal development of the neural systems that are required for olfactory long-term memory. To address this question, the authors used an inducible system to turn *teq* expression on or off in the mushroom bodies at specific times. When *teq* expression was suppressed in the mushroom bodies of adult flies, they developed a strong defect in long-term memory, which was restored when *teq* expression was allowed to resume.

These data, together with evidence that *teq* mRNA is upregulated in the mushroom bodies after long-term memory training, support the idea that TEQ has a crucial role in the formation of long-term memories in the fly. It is possible that neurotrypsin has a similar function in humans, but more work will be needed to clarify just what that function is.

Rachel Jones

ORIGINAL RESEARCH PAPER Didelot, G. *et al.* Tequila, a Neurotrypsin ortholog, regulates long-term memory formation in *Drosophila*. *Science* **313**, 851–853 (2006)

IN BRIEF

NEUROGENETICS

A new chromosome 17q21.31 microdeletion syndrome associated with a common inversion polymorphism.

Koolen, D. A. *et al. Nature Genet.* **38**, 999–1001 (2006)

Microdeletion encompassing *MAPT* at chromosome 17q21.3 is associated with developmental delay and learning disability.

Shaw-Smith, C. *et al. Nature Genet.* **38**, 1032–1037 (2006)

Discovery of previously unidentified genomic disorders from the duplication architecture of the human genome.

Sharp, A. J. *et al. Nature Genet.* **38**, 1038–1042 (2006)

Advances in the detection of submicroscopic chromosome deletions are helping to uncover genomic disorders that cause mental retardation. Three studies have identified a microdeletion syndrome affecting chromosome 17q21.3, estimated to account for up to 1% of mental retardation cases. The deletion was associated with an inversion polymorphism found in 20% of Europeans and gave rise to a clinically recognizable phenotype. The deletion encompassed several genes, including microtubule-associated protein tau, which has been linked to neural development and neurodegeneration.

SYNAPTIC PLASTICITY

Cooperative astrocyte and dendritic spine dynamics at hippocampal excitatory synapses.

Haber, M., Zhou, L. & Murai, K. K. *J. Neurosci.* **26**, 8881–8891 (2006)

Although the role of morphological rearrangements in dendritic spines in synaptic plasticity is well appreciated, less is known about glial motility at synapses, despite evidence that glia can modulate synaptic function. To enable the visualization of synaptic interactions in live cells, Haber *et al.* infected hippocampal slices with viral vectors to mediate the expression of two different fluorescent proteins in astrocytes and neurons. Time-lapse imaging revealed that astrocytes exhibit dynamic morphological plasticity, rapidly extending and retracting processes in a manner that was often coordinated with changes in dendritic spines. Astrocyte plasticity might therefore make an important contribution to the function of synapses, by regulating communication between neurons and glia.

BEHAVIOURAL NEUROSCIENCE

A role for the macaque anterior cingulate gyrus in social valuation.

Rudebeck, P. H. *et al. Science* **313**, 1310–1312 (2006)

In humans, damage to the frontal lobe can lead to impairments in social interaction, and in extreme cases, sociopathy, but the exact region that is responsible for these deficits has been unclear. New work in macaques shows that damage to the anterior cingulate cortex disrupts normal patterns of social interest in other macaques. By contrast, the orbitofrontal cortex did not seem to be necessary for this type of social functioning, but was instead associated with fear responses. It is therefore likely that the anterior cingulate cortex contributes, along with other brain regions, to normal social behaviour.