

## IN BRIEF

**MEMORY**

Patients with hippocampal amnesia cannot imagine new experiences.

Hassabis, D. *et al.* *PNAS* **104**, 1726–1731 (2007)

Patients with primary damage to the hippocampus have deficits in recollecting past events. Hassabis *et al.* show that four out of five of such patients were also impaired in imagining new experiences based on simple scenarios given to them by the researchers. The imagined experiences were scored for the amount of detail they contained, including spatial coherence. The patients had particular difficulties imagining the environmental setting of the story, indicating that the hippocampus is important in providing a spatial context into which an imagined experience is set.

**GENES AND DISEASE**

The neuronal sortilin-related receptor SORL1 is genetically associated with Alzheimer disease.

Rogaeva, E. *et al.* *Nature Genet.* **39**, 168–177 (2007)

Abnormal processing of amyloid precursor protein (APP), leading to accumulation of the amyloid- $\beta$  peptide (A $\beta$ ), may contribute to the pathology of Alzheimer's disease (AD). The authors showed that inherited variants in SORL1, a member of the vacuolar protein sorting family, which result in reduced levels of this protein, were associated with late-onset AD. Furthermore, SORL1 regulated the sorting of APP into a recycling pathway or into an endocytic pathway that produces A $\beta$ , and reducing SORL1 levels led to increased A $\beta$  production.

**NEUROIMAGING**

Wandering minds: the default network and stimulus-independent thought.

Mason, M. F. *et al.* *Science* **315**, 393–395 (2007)

When carrying out non-demanding or familiar tasks, the mind often tends to wander, but which parts of the brain are responsible for this activity? The authors used functional MRI to compare brain activity during the performance of familiar tasks, which allowed the subjects' minds to wander, with that during novel task performance. They identified an association between activity in a default network of cortical areas and the performance of familiar tasks, which also correlated with the subjects' reported incidence of mind wandering.

**PRIONS**

Targeting cellular prion protein reverses early cognitive deficits and neurophysiological dysfunction in prion-infected mice.

Mallucci, G. R. *et al.* *Neuron* **53**, 325–335 (2007)

In mice, prion infection causes early changes in brain and behaviour that precede the neuron loss and motor symptoms associated with advanced prion disease. Mallucci *et al.* showed that hippocampus-related cognitive and behavioural abnormalities in mice with early prion infection can be reversed by removing the normal prion protein. This shows that behavioural deficits occur before there is neuron loss, and that removing the normal prion protein can reverse these deficits, offering potential for recovery in the early stages of prion infection.

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