IN BRIEF

NEUROLOGICAL DISORDERS

Reduced social interaction and ultrasonic communication in a mouse model of monogenic heritable autism

Jamain, S. *et al. Proc. Natl Acad. Sci. USA* **105**, 1710–1715 (2008)

Mutations in the gene encoding neuroligin 4 (*NLGN4*) have been associated with autism spectrum conditions (ASCs). Here, researchers showed that mice with a mutation in *Nlgn4* have altered social behaviour and memory and reduced vocalizations — deficits that mimic some aspects of the human conditions. This mouse might be a useful model with which to enhance our understanding of the contribution of synapse dysfunction to ASCs.

PAIN

Beyond feeling: chronic pain hurts the brain, disrupting the default-mode network dynamics

Baliki, M. N. *et al. J. Neurosci.* **28**, 1398–1403 (2008)

The default-mode network consists of brain regions that are more active at rest than during task performance. In this study, patients suffering from chronic back pain showed reduced de-activation in several areas of the default-mode network during a visual-attention task, although the patients' task performance did not differ from that of control participants. The altered activity during non-pain-related information processing suggests that disruption of this network might underlie the behavioural and cognitive impairments that are associated with chronic back pain.

SLEEP

Modulation of $GABA_A$ receptor desensitization uncouples sleep onset and maintenance in *Drosophila*

Agosto, J. *et al. Nature Neurosci.* 27 Jan 2008 (doi:10.1038/nn2046)

$GABA_A$-receptor agonists increase sleep in insomnia patients, but it is unclear how the $GABA_A$ receptor regulates sleep. Here, fruit flies with a mutant form of the receptor (Rdl^{A302S}) showed greatly reduced sleep latency. Carbamazepine (CBZ) affected both latency and sleep-episode duration in wild-type flies, but in Rdl^{A302S} flies it only reduced sleep-episode duration. Moreover, CBZ increased wild-type $GABA_A$-receptor desensitization, but not that of Rdl^{A302S}, which had a decreased rate of desensitization. These findings indicate that onset and maintenance of sleep are uncoupled, and that sleep initiation is controlled by fast-desensitizing $GABA_A$ receptors.

LEARNING AND MEMORY

Synaptic protein degradation underlies destabilization of retrieved fear memory

Lee, S.-H. *et al. Science* 7 February 2008 (doi: 10.1126/science.1150541)

Memory reconsolidation is thought to require protein synthesis; the authors of this study showed that protein degradation by the ubiquitin–proteasome system is also involved. Infusion of the proteasome inhibitor βlac in the CA1 of mice immediately after retrieval of a conditioned fear memory did not affect a second retrieval 24 hours later, but did prevent the retrieval-impairing effect of a protein-synthesis blocker. Moreover, βlac infusion in the CA1 prevented fear extinction. These findings suggest that protein degradation might destabilize pre-existing memories, allowing them to be updated and reconsolidated by protein synthesis.