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# **IN BRIEF**

## **⇒** SLEEP

## Asleep but aware

Although we cannot interact with the environment while asleep, our brains continue to process incoming stimuli. Here, individuals classified spoken words into categories (such as animals or objects) during the transition to sleep. The authors found that the neural activity patterns associated with this task — which represented selection of and preparation for a motor response — continued when asleep individuals were exposed to the words. Thus, goal-directed processing of sensory stimuli can continue to the point of response preparation during sleep.

**ORIGINAL RESEARCH PAPER** Kouider, S. et al. Inducing task-relevant responses to speech in the sleeping brain. *Curr. Biol.* <a href="http://dx.doi.org/10.1016/j.cub.2014.08.016">http://dx.doi.org/10.1016/j.cub.2014.08.016</a> (2014)

### **DEPRESSION**

#### Mood food

Low levels of dietary n-3 polyunsaturated fatty acids (PUFAs) are linked to anxiety and depression; however, the underlying mechanisms are unknown. Larrieu  $et \, al.$  showed that mice that were fed a diet deficient in n-3 PUFAs exhibited behavioural changes and neuronal atrophy patterns that resemble those of mice exposed to social defeat stress, a model of depression. These effects were linked to hypothalamic–pituitary–adrenal (HPA) axis hyperactivity and were reversed by n-3 PUFA supplementation. Thus, dietary n-3 PUFAs may be important in maintaining HPA axis function and preventing emotional impairment.

**ORIGINAL RESEARCH PAPER** Larrieu, T. et al. Nutritional omega-3 modulates neuronal morphology in the prefrontal cortex along with depression-related behaviour through corticosterone secretion. *Transl. Psychiatry* 4, e437 (2014)

#### LEARNING AND MEMORY

# **Epigenetic exchange mechanisms**

Epigenetic mechanisms, including histone modification and DNA methylation, are known to regulate memory formation. Zovkic *et al.* now reveal that histone variant exchange — another form of epigenetic regulation in which variant histones are incorporated into nucleosomes — has a role in memory consolidation. They found that fear conditioning induces changes in the levels of H2A.Z (a variant of histone H2A) in nucleosomes of memory-related genes in mice. Furthermore, H2A.Z depletion in the hippocampus and cortex enhanced early and late stages of memory consolidation, respectively.

**ORIGINAL RESEARCH PAPER** Zovkic, I. B. et al. Histone H2A.Z subunit exchange controls consolidation of recent and remote memory. *Nature* <a href="http://dx.doi.org/10.1038/nature13707">http://dx.doi.org/10.1038/nature13707</a> (2014)

# ■ NEURODEGENERATION

#### Active astrocytes drive inflammation

Astrocyte activation has been linked to neurodegeneration in conditions such as multiple sclerosis (MS); however, the molecular mechanisms involved are unclear. The authors found that the enzyme that synthesizes lactosylceramide (LacCer) is upregulated in astrocytes in experimental autoimmune encephalomyelitis (a mouse model of MS) and in clinical MS lesions. LacCer was found to promote astrocyte activation and to control the transcription of genes related to neuroinflammation and neurodegeneration. Thus, upregulation of LacCer production in astrocytes may contribute to disease pathogenesis in inflammatory CNS conditions.

ORIGINAL RESEARCH PAPER Mayo, L. et al. Regulation of astrocyte activation by glycolipids drives chronic CNS inflammation. *Nature Med.* http://dx.doi.org/10.1038/nm.3681 (2014)