

## IN BRIEF

**NEURODEGENERATIVE DISEASE****Restoring balance**

Intracellular accumulation of the microtubule-associated protein tau is a hallmark of Alzheimer disease. Alternative splicing of the gene encoding tau can generate tau isoforms with three repeat domains (3R) or four repeat domains (4R), and it has been hypothesized that an excess of 4R tau is toxic. Schoch *et al.* used antisense oligonucleotides to switch the predominant tau splice isoform from 3R to 4R in mice. This resulted in an increase in pentylenetetrazole-induced seizure severity, impaired nesting activity, and increased tau phosphorylation and aggregation, thus demonstrating pathological effects of elevated levels of the 4R isoform.

**ORIGINAL ARTICLE** Schoch, K. M. *et al.* Increased 4R-tau induces pathological changes in a human-tau mouse model. *Neuron* <http://dx.doi.org/10.1016/j.neuron.2016.04.042> (2016)

**CELL BIOLOGY OF THE NEURON****Life in the slow lane**

Dynein is a molecular motor that is involved in retrograde axonal transport, but the mechanism by which it reaches distal axonal locations is incompletely understood. Here, the authors used live cell imaging techniques in the mouse brain and found that dynein undergoes anterograde transport as a complex with kinesin 1 that is attached to microtubules. However, the complex was unstable, and transient periods of dynein transport were interspersed with periods in which dynein disassembled from the transport complex and microtubules.

**ORIGINAL ARTICLE** Twelvetrees, A. E. *et al.* The dynamic localization of cytoplasmic dynein in neurons is driven by kinesin-1. *Neuron* <http://dx.doi.org/10.1016/j.neuron.2016.04.046> (2016)

**SLEEP AND MEMORY****Sleeping through the performance**

The consolidation of memory traces during sleep for their long-term storage in the cortex has not been demonstrated directly. Maingret *et al.* trained rats either in a spatial memory task that required consolidation or a task that did not. They found that hippocampo-cortical oscillatory coupling during sleep increased only following training in the task that required memory consolidation. Moreover, artificially boosting this temporal coordination during sleep increased the responsiveness of prefrontal neurons to the task and improved next-day performance in the task, suggesting that hippocampo-cortical communication during sleep is important for memory consolidation.

**ORIGINAL ARTICLE** Maingret, N. *et al.* Hippocampo-cortical coupling mediates memory consolidation during sleep. *Nat. Neurosci.* <http://dx.doi.org/10.1038/nn.4304> (2016)

**NEUROPHYSIOLOGY****Going with the flow**

The spatial scale over which changes in neural activity influence local blood flow remains under debate. Here, blood vessel diameter and adjacent neuronal spiking activity in the cat visual cortex were monitored simultaneously during exposure of the animal to a range of stimulus orientations. Parenchymal vessel responses showed the same orientation selectivity as the surrounding neural tissue, but vascular dilation also occurred when there was little adjacent neural activity. This suggests that vessel responses do not precisely reflect the activity of adjacent neurons and operate over a wider spatial scale.

**ORIGINAL ARTICLE** O'Herron, P. *et al.* Neural correlates of single-vessel haemodynamic responses in vivo. *Nature* <http://dx.doi.org/10.1038/nature17965> (2016)