

## IN BRIEF

 **NEUROLOGICAL DISORDERS****Telomeres and depression**

Studies of a link between reduced telomere length and depression and/or anxiety disorders have produced conflicting results. A recent large-scale survey of health and nutrition of US civilians found that women (but not men) with anxiety disorders had shorter telomeres than controls. Both men and women with major depression that was severe enough to be prescribed antidepressants were found to have shorter telomeres than those with less severe depression who were not taking antidepressants. These findings link telomere length to anxiety disorders and severity of depression.

**ORIGINAL RESEARCH PAPER** Needham, B. L. *et al.* Depression, anxiety and telomere length in young adults: evidence from the National Health and Nutrition Examination Survey. *Mol. Psychiatry* <http://dx.doi.org/10.1038/mp.2014.89> (2014)

 **GENES AND DISEASE****Schwann cells keep axons healthy**

Charcot-Marie-Tooth disease 1A (CMT1A) is an inherited neuropathy caused by duplication of the gene encoding peripheral myelin protein 22 (PMP22). In this study, *PMP22* duplication in rodents caused defects in Schwann cell differentiation in early postnatal development, which persisted into adulthood. This abnormal Schwann cell phenotype could be rescued by axonal overexpression of neuregulin-1 (NRG1) or by treatment with soluble NRG1 in early postnatal stages. These data suggest that there is a temporal window for correct Schwann cell differentiation that is vital to prevent the neuropathy observed in CMT1A.

**ORIGINAL RESEARCH PAPER** Fledrich, R. *et al.* Soluble neuregulin-1 modulates disease pathogenesis in rodent models of Charcot-Marie-Tooth disease 1A. *Nature Med.* <http://dx.doi.org/10.1038/nm.3664> (2014)

 **CELLULAR NEUROPHYSIOLOGY****Clearing away the debris**

Damaged mitochondria are cleared from neurons by mitophagy. At the soma, this process is mediated by lysosomes and requires two Parkinson's disease-related proteins, parkin and PINK1. However, it remains controversial whether the same mechanisms operate in distal axons where lysosomes are sparse. The authors showed that damage to mitochondria in the distal axons of hippocampal neurons caused recruitment of PINK1 and parkin, which act locally to mediate mitophagy. Knockout of either protein prevented mitophagy in damaged hippocampal axons. These findings implicate the PINK1–parkin pathway in the response of distal axons to mitochondrial damage.

**ORIGINAL RESEARCH PAPER** Ashrafi, G. *et al.* Mitophagy of damaged mitochondria occurs locally in distal neuronal axons and requires PINK1 and Parkin. *J. Cell Biol.* **206**, 655–670 (2014)

 **COGNITIVE NEUROSCIENCE****Telling the truth, honestly...**

Although the prefrontal cortex (PFC) has been implicated in aspects of behaviour relating to honesty, the mechanisms and circuits involved are unknown. The authors tested the responses of people in games in which subjects had to decide between self-interest and honesty. People with lesions to the dorsolateral region of the PFC (DLPFC) had fewer concerns about the honesty of a particular decision than healthy participants. These data suggest that the role of the DLPFC could be to rein in self-interest urges, allowing more honest decisions to be made.

**ORIGINAL RESEARCH PAPER** Zhu, L. *et al.* Damage to dorsolateral prefrontal cortex affects tradeoffs between honesty and self-interest *Nature Neurosci.* <http://dx.doi.org/10.1038/nn.3798> (2014)