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IN BRIEF

STROKE

The location of pontine infarction affects progression of motor deficits

Acute pontine infarction is commonly associated with progressive motor deficits (PMD) and functional disability. In a recent study, Semi *et al.* identified 190 patients with acute ischaemic stroke in the pons, 49 of whom had PMD. Clinical, laboratory and imaging data revealed involvement of the lower pons as an independent risk factor for worsening of motor symptoms, suggesting infarct topography could be used in prognostication for this type of stroke.

Original article Semi, O. *et al.* Topographic location of acute pontine infarction is associated with the development of progressive motor deficits. *Stroke* **43**, 708–713 (2012)

NEURODEGENERATIVE DISEASE

Repeat expansions in *NIPA1* confer susceptibility to amyotrophic lateral sclerosis

Mutations in the gene *NIPA1* cause hereditary spastic paraplegia, and deletions of the gene have been linked to development of amyotrophic lateral sclerosis (ALS). Sequencing of the gene in 2,292 patients with ALS and 2,777 controls revealed two gene variants that are specific to patients with ALS and might negatively affect the function of the encoded protein. Moreover, long tracts of polyalanine repeat expansions in *NIPA1* were associated with reduced survival and lower age at onset of symptoms.

Original article Blauw, H. M. *et al. NIPA1* polyalanine repeat expansions are associated with amyotrophic lateral sclerosis. *Hum. Mol. Genet.* doi:10.1093/hmg/dds064

ALZHEIMER DISEASE

Epigenetic modifications could promote cognitive decline in Alzheimer disease

New evidence suggests upregulation of histone deactylase 2 (HDAC2) plays a central part in cognitive decline in Alzheimer disease (AD) through epigenetic modifications. Compared with controls, HDAC2 levels were significantly increased in hippocampal neurons of AD mice, and transcription of genes implicated in learning and memory was reduced. Knockdown of HDAC2 with short-hairpin RNA improved performance in learning and memory tasks. Moreover, in postmortem samples from patients with AD, HDAC2 levels were elevated in hippocampal area CA1 and the entorhinal cortex.

Original article Gräff, J. *et al.* An epigenetic blockade of cognitive functions in the neurodegenerating brain. *Nature* **483**, 222–226 (2012)

STROKE

A role for proton channel Hv1 in ischaemic brain damage

Generation of reactive oxygen species (ROS) by NADPH oxidase (NOX) contributes to brain damage during cerebral ischaemia. Researchers have now shown that proton flux through the voltage-gated proton channel Hv1 expressed on microglia supports NOX activity in mice. In a stroke model, trangenic mice lacking Hv1 showed lower infarct volumes and improved neurological scores compared with wild-type mice, together with reduced markers of cell death in the peri-infarct zone and lower ROS production.

Original article Wu, L.-J. *et al.* The voltage-gated proton channel Hv1 enhances brain damage from ischemic stroke. *Nat. Neurosci.* doi:10.1038/nn.3059