Nature Reviews Neurology **11**, 668 (2015); published online 20 November 2015;
doi:10.1038/nrneurol.2015.211;
doi:10.1038/nrneurol.2015.212;
doi:10.1038/nrneurol.2015.213;
doi:10.1038/nrneurol.2015.214

IN BRIEF

TRAUMATIC BRAIN INJURY

High serum caspase-3 level confers poor prognosis after TBI

Nonsurvivors of traumatic brain injury (TBI) are known to exhibit higher levels of caspase-3 in contused brain tissue than do survivors. Now, Lorente *et al.* report that serum caspase-3 levels are also linked to increased mortality. Caspase-3 inhibitors have been shown to reduce apoptosis in rat models of TBI, making suppression of apoptotic activity a potential novel strategy to treat TBI.

Original article Lorente, L. *et al.* Serum caspase-3 levels and mortality are associated in patients with severe traumatic brain injury. *BMC Neurol.* **15**, 28 (2015)

PAIN

Epigenetic regulation could contribute to pain chronification

Downregulation of K^+ channel genes in the injured dorsal root ganglia is one of the hallmarks of neuropathic pain. In a new mouse study, nerve injury increased activity of histone-lysine *N*-methyltransferase EHMT2 (G9a), resulting in epigenetic silencing of genes encoding K^+ channels and transition from acute to chronic neuropathic pain. The findings indicate that G9a is a potential epigenetic target for prevention and treatment of chronic neuropathic pain.

Original article Laumet, G. *et al.* G9a is essential for epigenetic silencing of K^+ channel genes in acute-to-chronic pain transition. *Nat. Neurosci.* doi:10.1038/nn.4165

ALZHEIMER DISEASE

Suppression of stress signalling prevents AD onset in mice

Stress has been implicated as a risk factor for Alzheimer disease (AD), and altered stress signalling has been demonstrated in mouse models of AD, but the mechanisms underlying the involvement of stress in AD pathogenesis remain elusive. Cheng Zhang and colleagues treated female AD model mice with the corticotropin-releasing factor receptor 1 antagonist R121919, and found that inhibition of corticotropin signalling mitigated amyloid-β pathology and prevented onset of cognitive impairment. No signs of toxicity or tolerability issues associated with R121919 administration in mice were observed, but its safety in humans is unknown.

Original article Zhang, C. *et al.* Corticotropin-releasing factor receptor-1 antagonism mitigates beta amyloid pathology and cognitive and synaptic deficits in a mouse model of Alzheimer's disease. *Alzheimers Dement.* doi:10.1016/j.jalz.2015.09.007

DISORDERS OF CONSCIOUSNESS

Functional connectivity could be a prognostic neuroimaging marker for recovery from coma

Spared posteromedial network connectivity could predict favourable outcome in comatose patients, according to a recently published prospective study of 27 patients in coma after severe brain injury (Glasgow Coma Scale score <8), and 14 age-matched healthy participants. Specifically, strong functional connectivity between the posterior cingulate cortex and medial prefrontal cortex was associated with increased likelihood of recovery from coma. The results suggest that the major consciousness deficit in coma could arise from disrupted long-range neuronal communication.

Original article Silva, S. *et al.* Disruption of posteromedial large-scale neural communication predicts recovery from coma. *Neurology* doi:10.1212/WNL.0000000000002196