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IN BRIEF

NEURODEGENERATIVE DISEASE

Tau acetylation is an early pathological event in neurodegenerative disease

Soluble tau oligomers have been strongly implicated in the pathogenesis of neurodegenerative diseases such as Alzheimer disease (AD) and frontotemporal dementia (FTD). Research published in *Nature Medicine* now shows that soluble tau is acetylated at Lys174 in the human brain from the early stages of AD, and that inhibition of the acetyltransferase p300 ameliorates hippocampal atrophy and cognitive deficits in a transgenic mouse model of FTD. The authors conclude that Lys174 acetylation is a crucial determinant of tau oligomer accumulation and toxicity.

Original article Min, S.-W. *et al.* Critical role of acetylation in tau-mediated neurodegeneration and cognitive deficits. *Nat. Med.* **21**, 1154–1162 (2015)

DEMYELINATING DISEASE

Neurodegeneration distinguishes MS from NMO

Widespread CNS neurodegeneration is a feature of multiple sclerosis (MS) but not neuromyelitis optica (NMO), according to a new report. In a longitudinal advanced MRI study, Jacqueline Palace and colleagues found inflammatory brain lesions in both MS and NMO, but diffuse neurodegeneration was only detectable in patients with MS. These findings might explain why NMO almost invariably follows a relapsing–remitting course, whereas MS frequently enters a progressive phase.

Original article Matthews, L. *et al.* Imaging surrogates of disease activity in neuromyelitis optica allow distinction from multiple sclerosis. *PLoS ONE* **10**, e0137715 (2015)

TRAUMATIC BRAIN INJURY

Functional connectivity is altered after mild TBI

Resting-state magnetoencephalography (MEG) has uncovered changes in brain functional connectivity after mild traumatic brain injury (mTBI). In *NeuroImage: Clinical*, Dimitriadis *et al.* reported a reduction in the strength of local connections and an increase in the strength of long-range connections in the brains of individuals with mTBI compared with healthy controls. The researchers suggest that MEG-based functional connectivity could provide a diagnostic and therapeutic biomarker for mTBI.

Original article Dimitriadis, S. I. *et al.* Functional connectivity changes detected with magnetoencephalography after mild traumatic brain injury. *Neuroimage Clin.* doi:10.1016/j.nicl.2015.09.011

MOVEMENT DISORDERS

Is essential tremor a neurological channelopathy?

A new study has found that an autosomal dominantly inherited mutation in the *SCN4A* gene is associated with essential tremor and increased susceptibility to epilepsy in a large Spanish family. *SCN4A* encodes the voltage-gated sodium channel Na_v1.4, which was previously thought to be confined to muscle, but the researchers were able to detect *SCN4A* mRNA in neuronal tissue. The results point towards neurological channelopathies as one of the underlying causes of essential tremor.

Original article Bergareche, A. *et al.* *SCN4A* pore mutation pathogenetically contributes to autosomal dominant essential tremor and may increase susceptibility to epilepsy. *Hum. Mol. Genet.* doi:10.1093/hmg/ddv410