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IN BRIEF

NEURODEGENERATIVE DISEASE

Actin up

The GGGGCC (G4C2) repeat expansion in the *C9orf72* gene is a common genetic cause of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD), but the mechanism is unknown. Using quantitative proteomics, the authors found that C9orf72 protein interacts with the actin-binding protein cofilin. Cofilin phosphorylation was increased in mouse motor neurons with reduced C9orf72 expression and in ALS patients with the *C9orf72* G4C2 expansion. Moreover, dynamics were found to be disrupted in both cultured mouse motor neurons lacking C9orf72 and in induced pluripotent stem cell-derived motor neurons from people with ALS, suggesting that altered actin dynamics could contribute to the pathology underlying ALS and FTD.

ORIGINAL ARTICLE Sivadasan, R. *et al.* C9ORF72 interaction with cofilin modulates actin dynamics in motor neurons. *Nat. Neurosci.* http://dx.doi.org/10.1038/nn.4407 (2016)

NEURAL DEVELOPMENT

The river runs through it

In humans, some neurons can be detected migrating after birth from the subventricular zone (SVZ) to cortical regions; however, it is not known to what extent these cells mature and become integrated into human forebrain circuits. Here, brain imaging and time-lapse confocal microscopy were used to show that young migrating neurons expressing interneuronal markers travel from the SVZ to various forebrain regions, including the anterior cingulate cortex, where they become integrated into local circuits.

ORIGINAL ARTICLE Paredes, M. F. *et al.* Extensive migration of young neurons into the infant human frontal lobe. *Science* http://dx.doi.org/10.1126/science.aaf7073 (2016)

NEURODEGENERATIVE DISEASE

Resisting the chop

It is currently unclear which forms of the protein tau contribute to Alzheimer disease (AD) pathology, but Δtau314, which is produced following caspase 2-mediated tau cleavage, is elevated in the brains of cognitively impaired mice and humans with AD. Here, *in vitro* expression of a mutant form of tau that was resistant to caspase 2 cleavage reduced tau accumulation in dendritic spines and tau-induced synaptic dysfunction. Moreover, knockdown of caspase 2 expression in the rTg4510 mouse tauopathy model improved long-term memory in mice with existing deficits; thus, caspase 2 inhibition might have therapeutic potential in human tauopathies.

ORIGINAL ARTICLE Zhao, X. *et al.* Caspase-2 cleavage of tau reversibly impairs memory. *Nat. Med.* http://dx.doi.org/10.1038/nm.4199 (2016)

SLEEP AND MEMORY

Rippling memories

Transient, high-frequency hippocampal oscillations called sharp wave–ripples may be involved in the formation of memories that are later consolidated in the neocortex. Oscillations of ripple frequency (~200 Hz) have been reported in the rodent neocortex during sleep but are not well understood. Here, the authors made field-potential recordings from the neocortex of sleeping rats and found that short periods of ripple oscillations, produced by subpopulations of fast-spiking, parvalbumin-containing basket cells, occurred in the troughs of neocortical sleep 'spindles' (periods of low-frequency oscillations) and could play a part in memory processes.

ORIGINAL ARTICLE Averkin, R. G. *et al.* Identified cellular correlates of neocortical ripple and high-gamma oscillations during spindles of natural sleep. *Neuron* http://dx.doi.org/10.1016/j.neuron.2016.09.032 (2016)