Nature Reviews Neurology | Published online 16 Mar 2018

IN BRIEF

SPINAL CORD INJURY

Human neural stem cells elicit regeneration after spinal cord injury in monkeys

Restoration of function after spinal cord injury in humans remains an elusive goal despite the extensive progress that has been made in rodent models. Now, investigators have successfully grafted neural progenitor cells derived from human spinal cord into a primate model of spinal cord injury. The grafted cells survived in the rhesus monkeys and expressed neuronal and glial markers. Strikingly, the cells extended thousands of long axons that bridged a cervical spinal cord lesion and formed synapses with host motor neurons. Furthermore, axons from monkey neurons also regenerated into the graft. After several months of growth, the grafts elicited a partial restoration of limb function in the animals. The findings show that stem cell engraftment might be a viable strategy for spinal cord regeneration in humans.

ORIGINAL ARTICLE Rosenzweig, E. S. et al. Restorative effects of human neural stem cell grafts on the primate spinal cord. *Nat. Med.* https://doi.org/10.1038/nm.4502 (2018)

PARKINSON DISEASE

Truncated α-synuclein causes mitochondrial toxicity

New research has identified a shortened variant of α-synuclein that binds to mitochondria in neurons and induces their destruction. Investigators found that this conformationally distinct form of α-synuclein, termed pα-syn*, is present in cultured primary neurons treated with preformed fibrils of α-synuclein, in Parkinson disease (PD) model mice and in patients with PD. The study showed that the presence of pα-syn* results from failure by the cell to break down fibrillar aggregates of α-synuclein in the lysosome. After release from the lysosome, pα-syn* binds to mitochondria, inducing depolarization of the mitochondrial membrane, mitochondrial fragmentation and mitophagy. This neurotoxic species could represent a crucial player in PD pathogenesis and could be a target of future treatments for patients with the condition.

ORIGINAL ARTICLE Grassi, D. et al. Identification of a highly neurotoxic α-synuclein species inducing mitochondrial damage and mitophagy in Parkinson's disease. *Proc. Natl Acad. Sci. USA* https://doi.org/10.1073/pnas.1713849115 (2018)

PRION DISEASE

Dramatic increases in blood levels of tau and neurofilament light in patients with prion disease

Current biomarkers of neuronal damage in sporadic Creutzfeldt–Jakob disease (sCJD) require analysis of cerebrospinal fluid, which involves an invasive lumbar puncture procedure. However, a study has now shown that levels of tau and neurofilament light (NfL) are raised substantially in the serum of patients with sCJD. Investigators used an ultrasensitive single molecule array assay to monitor very low concentrations of these molecules in the blood. The team found that serum levels of NfL and tau enabled discrimination between healthy individuals and patients with sCJD with high sensitivity and specificity. Longitudinal analysis showed that levels of both proteins increased in patients with sCJD over time. Furthermore, serum levels of tau correlated positively with the rate of disease progression. These findings could facilitate early identification of patients with sCJD and provide useful outcome measures in future therapeutic trials.

ORIGINAL ARTICLE Thompson, A. G. et al. Neurofilament light chain and tau concentrations are markedly increased in the serum of patients with sporadic Creutzfeldt–Jakob disease, and tau correlates with rate of disease progression. *J. Neurol. Neurosurg. Psychiatry* https://doi.org/10.1136/jnnp-2017-317793 (2018)