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IN BRIEF

NEURAL REPAIR AND REHABILITATION

Robot-assisted physiotherapy in cerebral palsy

A brief intervention with robot-assisted therapy could improve upper limb function in children with cerebral palsy (CP), a new study shows. Researchers randomly assigned 16 children with CP to receive three sessions of conventional therapy and two robot-assisted therapy sessions, or five sessions of conventional therapy. In a blinded assessment, the children who had received robot-assisted therapy showed markedly improved smoothness of movement and manual dexterity compared with the children in the conventional therapy group.

Original article Gillaux, M. *et al.* Upper limb robot-assisted therapy in cerebral palsy—a single-blind randomized controlled trial. *Neurorehabil. Neural Repair* doi:10.1177/1545968314541172

STROKE

Altered astrocytic ion channel expression in ischaemia

A recent study has shown that experimentally induced cerebral ischaemia in mice and rats results in increased expression of hyperpolarization-activated cyclic nucleotide-gated (HCN) channels in astrocytes. HCN channel overexpression contributed to the altered membrane properties of reactive astrocytes after ischaemia, and correlated with glial scar formation. The HCN channel, therefore, represents a target for stroke therapy.

Original article Honsa, P. *et al.* Increased expression of hyperpolarization-activated cyclic nucleotide-gated (HCN) channels in reactive astrocytes following ischemia. *Glia* doi:10.1002/glia.22721

HUNTINGTON DISEASE

Transneuronal propagation of mutant huntingtin protein

Mutant huntingtin (mHTT) might spread from neuron to neuron in a prion-like fashion. Pecho-Vrieseling *et al.* discovered that neurons derived from human stem cells could acquire mHTT aggregates and HD-like pathology when cultured with mouse organotypic slices that were genetically engineered to recapitulate HD pathophysiology, including mHTT expression. The results suggest that prion-like propagation of mHTT through synaptic vesicle trafficking might contribute to HD pathophysiology.

Original article Pecho-Vrieseling, E. *et al.* Transneuronal propagation of mutant huntingtin contributes to non-cell autonomous pathology in neurons. *Nat. Neurosci.* doi:10.1038/nn.3761

NEURO-ONCOLOGY

GABAergic excitation drives epileptiform activity in glioma

Seizures are a common comorbidity of gliomas. Johan Pallud and colleagues studied the cellular mechanisms of epileptic activity in the peritumoral surgical margin from 29 patients with gliomas. Altered chloride cotransporter expression perturbed chloride homeostasis and rendered neurons around gliomas susceptible to γ-aminobutyric acidergic (GABAergic) excitation, thereby increasing epileptiform activity. Controlling chloride balance might, therefore, be a potential therapeutic strategy for reducing seizures in patients with gliomas.

Original article Pallud, J. *et al*. Cortical GABAergic excitation contributes to epileptic activities around human glioma. *Sci. Transl. Med.* doi:10.1126/scitranslmed.3008065