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Update on treatment and care in Huntington's disease: European Huntington's disease network perspective

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Huntington's disease (HD) is lethal, hereditary neurodegenerative disorder. The gene and the mutation causing this monogenetic disorder was identified more than 20 years agobut disease modifying therapies for HD have not been yet founded. Several preclinical research and large cohort studies like REGISTRY, COHORT or ongoing Enroll-HD significantly supported understanding of HD and its progression as well as the care of HD patientsbut the identification of targets for therapeutic interventions and development new chemical entities and advanced therapies incorporating application of DNA or RNA molecules as therapeutic agents are still ongoing. Disease modifying therapies like huntingtin lowering strategies and improving huntingtin clearance promoted by posttranslational HTT modifications are most important directions of future therapies development. Current symptomatic treatment, palliative surgical interventions, physiotherapy and care are available in highly developed countries for HD patients. It is important to deliver these methods to countries where these are still limited for HD and develop symptomatic treatment options as they are still only available for HD patients. High number of upcoming clinical studies/trials in HD and improvement in symptomatic interventions are reasons for hope for HD patients and their families.

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Basal ganglia oscillations in Parkinson´s disease: From rodents to humans

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Experimental works on animal models of Parkinson´s Disease (PD) and clinical studies have identified changes in the physiology of striatal neurons that occur as a result of dopamine depletion. Many of the neuronal cell types in the striatum are affected. The medium spiny projection neurons (MSNs) that give rise to the direct and indirect pathways of the basal ganglia, striatal interneurons such as the tonically active neurons (TANs, largely corresponding the cholinergic interneurons) and the fast-spiking parvalbumin-containing interneurons (FSIs) whose synaptic pattern is changed drastically after dopamine removal.These neuronal changes have consequences on the overall neuronal activity and changes in long field potentials(LFPs) activity have been identified across the cortico-basal ganglia circuits both clinically and experimentally. Landmark features in PD are the alterations in the brain oscillations increased beta-band activity and diminished gamma-band activity have been described in association with this pathology and these changes may be critical to both motor and cognitive deficits that occur in Parkinsonian patients. Dopamine loss in PD occurs first in the dorsolateral striatum (DLS) before other striatal and extrastriatal regions are gradually affected. The DLS is implicated in the acquisition and performance of habits and procedural behaviors and dopamine may be required for these learning and motor related functions of the DLS. I will review here my studies in which the consequences of dopamine depletion and L-DOPA therapy on task-related striatal network activity have been identified. Both the firing activity of the different sub-classes of striatal neurons and oscillatory field activitywere affected in a very specific manner in an animal model of early PD during the performance and acquisition of a procedural task. Moreover, I will address the similarities and differences in brain oscillations and a common key feature that may be behind the next future therapeutic target in PD research.

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