Neural Plasticity

Hindawi

Special Issue on

Cortical Circuitry and Synaptic Dysfunctions in Alzheimer's Disease and Other Dementias

CALL FOR PAPERS

The observable symptoms of dementia can be considered as the tip of an iceberg, made up of neuropathological alterations which accumulate over time and lead to loss of synaptic connections and dysfunction of cortical circuitry. Specific histological and functional abnormalities can be associated with different degenerative types of dementia. Over recent decades, several studies have attempted to establish the relation between neuropathological findings, such as amyloid and tau protein alterations, and impairment of synaptic transmission. In Alzheimer's disease, attention has long been focused on the central cholinergic deficit and impairment of synaptic plasticity, but recent findings also point towards selective dopaminergic neuron degeneration leading to hippocampal long-term potentiation (LTP) dysfunction as a possible upstream phenomenon. A primary dysfunction of synaptic plasticity, possibly favored by genetic factors, has also been hypothesized as the leading cause of molecular and histopathological modifications.

A better understanding of the above alterations is required for the development of drugs that target specific pathological mechanisms. Moreover, the early detection of pathological changes is crucial for the use of novel disease-modifying treatments that need to be administered early in the disease course. Current biomarkers for the diagnosis of dementias are mainly based on evidence of amyloid deposition and neuronal injury. Increasing evidence also suggests a role for functional neuroimaging and neurophysiological tools (fMRI, EEG, and transcranial magnetic stimulation) in the identification of novel biomarkers of dysfunction of neurotransmission and synaptic plasticity.

This Special Issue aims to collect both original research and review articles addressing the role of cortical circuitry and synaptic dysfunctions in both cognitive impairment and potential biomarkers of Alzheimer's disease and other degenerative forms of dementia. Submissions concerning neural network dysfunction are also welcomed, as are studies in both humans and animal models.

Potential topics include but are not limited to the following:

- Structural and functional alterations of synaptic connections in dementias, investigated in humans and in animal models
- Links between genetic risk factors, neuropathological alterations, and neural plasticity
- Time course of neurophysiological alterations from the subclinical stages to mild cognitive impairment and dementia
- Neuroimaging and neurophysiological biomarkers of altered functional connectivity and synaptic transmission (investigated with MRI, EEG, and transcranial magnetic stimulation)
- Neurogenesis following neuronal damage
- Pharmacological interventions targeting specific neuropathological mechanisms of altered synaptic plasticity
- New experimental approaches to reestablish neural network function and/or synaptic plasticity using both optogenetics and chemogenetics

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/np/ccsd/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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