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Changes in Inhibitory Synapse Composition and Plasticity along the Progression of Neurodegenerative Diseases

Guest Editor:

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Message from the Guest Editor

Dear Colleagues,

Synaptic dysfunction, including altered synaptic plasticity, seems to be a central pathophysiological process in several neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis, and Huntington's disease. Research in recent years has strongly supported the involvement of GABAergic neurotransmission in both early and late pathogenesis of these groups of diseases. The GABAergic system might undergo dynamic remodeling and play different roles in the pathology at various disease stages. This seems to include stage-dependent alterations in presynaptic and postsynaptic components of inhibitory synapses and particularly scaffolding, anchoring and supporting proteins of GABAA receptors which influence the plasticity of the GABAergic system. The modulation of GABAergic neurotransmission through targeting inhibitory synapse proteins and their interactions (e.g., receptor–scaffold protein interactions) was proposed as a promising pharmacological strategy to normalize synapse dynamics and restore cognitive function.

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Message from the Editor-in-Chief

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