Neural Plasticity

Hindawi

Special Issue on

Glutamate Receptors in Alzheimer's Disease: Mechanisms and Therapies

CALL FOR PAPERS

Alzheimer's disease (AD) manifests as a progressive loss in memory and cognition, commonly associated with elevated levels of amyloid-beta peptide (Aβ) and hyperphosphorylated tau in the brain. There is currently no cure for AD as its causes remain poorly understood. Accumulating evidence suggests that synaptic dysfunction is a major contributor early in disease pathogenesis prior to neuronal loss. Glutamatergic neurotransmission is particularly vulnerable to the neurotoxic effect of Aβ and hyperphosphorylated tau. Indeed, these toxic species act in synergy and severely disrupt excitatory synaptic transmission, synaptic plasticity, and network activity, mainly due to the loss of AMPA receptors, as well as NMDA receptors.

Evidence also suggests that Aβ-induced synaptic loss and cell death are mediated by overstimulation of NMDA receptors, which could be ameliorated by NMDAR antagonist, such as memantine, an FDA-approved medication for AD. More recently, the identification of mGluR5 as a coreceptor for Aβ and a potential role for metabotropic-like function of NMDA receptors have provided novel insights into the molecular basis of AD. Therefore, understanding the underlying molecular mechanisms and complex signalling pathways by which these toxic species exert strong detrimental effects on synaptic function may yield novel strategies for the development of meaningful therapeutic agents. We invite authors to submit original research and review articles that explore aspects of glutamate receptor function in AD models, both in vitro and in vivo.

Potential topics include, but are not limited to:

- Effects of Aβ oligomers and tau hyperphosphorylation on glutamate receptor function and their underlying mechanisms of action
- Mechanisms of long-term plasticity in Alzheimer's disease
- Mechanisms of homeostatic synaptic plasticity in Alzheimer's disease
- Mechanisms of network dysfunction in Alzheimer's disease
- Mechanisms of excitotoxicity in Alzheimer's disease
- Modeling Alzheimer's disease in vitro and in vivo (IPSC, CRISPR/Cas9, and transgenic technologies)
- Glutamate receptors as potential therapeutic targets for Alzheimer's disease

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/np/grad/.

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