



Nicotinic Acetylcholine Receptors: Novel Targets for Neurological Diseases

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Message from the Guest Editor

Nicotinic acetylcholine receptors (AChR) play pivotal roles in the etiology of neurological disorders. The most well-known example is the AChR at the skeletal muscle neuromuscular junction, in which auto-antibodies against AChR subunits are a major underlying cause of Myasthenia Gravis.

The AChR subunits expressed at synaptic and non-synaptic sites throughout the body are also important therapeutic targets for several neurological disorders. AChR make good therapeutic targets for neurological diseases because they are often modulatory and regulate the primary neurotransmitters glutamate or dopamine. In other instances, AChR gene duplication or deletion is causative in neurological disease. *CHRNAT*, for example, is an important gene in the 15q13.3 microdeletion syndrome. The $\alpha 9$ subunit is unique in that it is not expressed in normal brain, but may be expressed during migration and/or proliferation in glioblastoma.

In this topic section we will focus on novel therapies that target AChR, particularly in neurological disorders that are currently resistant to treatment.





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