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Metabotropic GABAB receptors mediate GABA inhibition of acetylcholine release in the rat neuromuscular junction

Malomouzh A., Petrov K., Nurullin L., Nikolsky E.
Kazan Federal University, 420008, Kremlevskaya 18, Kazan, Russia

Abstract

© 2015 International Society for Neurochemistry. Gamma-aminobutyric acid (GABA) is an amino acid which acts as a neurotransmitter in the central nervous system. Here, we studied the effects of GABA on non-quantal, spontaneous, and evoked quantal acetylcholine (ACh) release from motor nerve endings. We found that while the application of 10 μM of GABA had no effect on spontaneous quantal ACh release, as detected by the frequency of miniature endplate potentials, GABA reduced the non-quantal ACh release by 57%, as determined by the H-effect value. Finally, the evoked quantal ACh release, estimated by calculating the quantal content of full-sized endplate potentials (EPPs), was reduced by 34%. GABA's inhibitory effect remained unchanged after pre-incubation with picrotoxin, an ionotropic GABAA receptor blocker, but was attenuated following application of the GABAB receptor blocker CGP 55845, which itself had no effect on ACh release. An inhibitor of phospholipase C, U73122, completely prevented the GABA-induced decrease in ACh release. Immunofluorescence demonstrated the presence of both subunits of the GABAB receptor (GABABR1 and GABABR2) in the neuromuscular junction. These findings suggest that metabotropic GABAB receptors are expressed in the mammalian neuromuscular synapse and their activation results in a phospholipase C-mediated reduction in the intensity of non-quantal and evoked quantal ACh release.

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Keywords

GABA receptors, phospholipase C, quantal and non-quantal acetylcholine release