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Author	鈴木, 岳之(Suzuki, Takeshi) 野中, 光(Nonaka, Hikaru) 藤本, 和子(Fujimoto, Kazuko) 川島, 紘一郎(Kawashima, Koichiro)
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Effects of Physostigmine and Some Nitric Oxide–Cyclic GMP–Related Compounds on Muscarinic Receptor–Mediated Autoinhibition of Hippocampal Acetylcholine Release*

Takeshi SUZUKI, Hikaru NONAKA, Kazuko FUJIMOTO and Koichiro KAWASHIMA

鈴木岳之，野中　光，藤本和子，川島紘一郎

We have investigated the effects of (a) the cholinesterase inhibitor physostigmine and (b) drugs that are known to change intracellular cyclic GMP levels on the autoinhibition of acetylcholine release from rat hippocampal slices. Autoinhibition was triggered by submaximal electrical stimulation in both the absence and presence of physostigmine. The results obtained indicate that an unusual increase in the extracellular acetylcholine content, such as that induced by cholinesterase inhibition, is not essential for autoinhibition triggering. Dibutyryl cyclic GMP reduced significantly the stimulation–evoked acetylcholine release in the presence, but not in the absence, of atropine. Neither sodium nitroprusside nor glyceryl trinitrate exerted a dibutyryl cyclic GMP–like effect. N^G–Nitro–L–arginine did not lessen the autoinhibition. These results indicate that an increase in the intracellular cyclic GMP level reduces acetylcholine release, and that the muscarinic receptor stimulation–nitric oxide synthesis–(soluble) guanylyl cyclase activation pathway is not involved in the cholinergic autoinhibition process.

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