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Cannabinoid CB1 receptors mediate neurokinin A-induced synaptic plasticity in the spinal locomotor network

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The spinal network underlying locomotion is composed of excitatory and inhibitory interneurons. Once locomotion is initiated, several modulatory systems including the neurokinin system are activated. Based on the implication of the endocannabinoid signaling in the spinal locomotor circuitry, we aimed to investigate whether this ubiquitous signaling system is involved in the modulation of locomotor frequency by neurokinin A. In male Wistar rats, the facilitatory effects of the neurokinin A (2 μM, 15 min) on the burst frequency and the amplitude of the contralateral compound glycinergic inhibitory postsynaptic potential (IPSP) were evaluated before and after the administration of 3-7 μM of the cannabinoid CB_1 and CB_2 receptor antagonists, AM251 and SR144528. In order to evaluate whether the endocannabinoid system is implicated in neurokinin A-mediated modulation of reciprocal inhibition, the commissural axons were stimulated, while recording intracellularly from motoneurons. Based on the results, treatment with 7 μM AM251, but not SR144528, prevented the facilitatory effects of neurokinin A on the frequency of burst and its inhibitory effects on IPSP amplitude. It is concluded that the effects of neurokinin A on the frequency of locomotor burst and IPSPs is mediated, at least in part, by CB_1 receptors.

Keywords: Cannabinoid CB1 receptors, neurokinin A, synaptic plasticity, AM251, SR144528, rat

Biography

Parichehr Hassanzadeh has received her Ph.D. in Pharmacology in 2006 and completed her postdoctoral studies in 2012 at Shahid Beheshti University of Medical Sciences, Tehran, Iran. Currently, she is research advisor in Nanomedicine and Tissue Engineering Research Center at the above-mentioned university. She has published about 20 research articles and 4 books either in Farsi or English (The World of Neuroscience) and is the member of the International Association for the Study of Pain.

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