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Jun 1, 2020. (PDF), (MP3). ud, water-soluble laquered and the red "M" shining and ready to be used this amazing sound. References Category:1952 births Category:Living people Category:Turkmenistan musicians Category:People from AshgabatPROJECT SUMMARY/ABSTRACT Delineating the mechanisms underlying the development of spinal motor deficits is crucial for the design of effective therapies to enhance functional recovery after spinal cord in jury (SCI). SCI leads to rapid loss of monosynaptic transmission in corticospinal pathways. Despite the extensive research on corticospinal plasticity, the mechanisms underlying the maintenance of synaptic efficacy, in particular, of weakened synapses, remain relatively poorly understood. Using a mouse model of spinal cord hemisection, we propose to test the general hypothesis that inhibition of adenosine A2A receptors (A2ARs) is sufficient to restore synaptic strength and boost functional connectivity in corticospinal pathways after SCI. In aim 1, we will investigate the role of A2ARs in synaptic maintenance after SCI. We will first examine the role of A2ARs in SCI-induced synaptic and motor dysfunction using an adeno-associated virus (AAV) approach to inhibit A2ARs in adult corticospinal neurons in vivo. We will then examine the consequences of inhibiting A2ARs on neural activity and synaptic transmission in cortical and spinal circuits. In aim 2, we will evaluate the role of A2ARs in pathological processes leading to synaptic loss after SCI. We will first study synaptic structural defects, such as increased spine length, in primary motor cortex after SCI using an AAV approach to inhibit A2ARs in the mouse cortex in vivo. We will then evaluate the contributions of axons and dendrites in SCI-induced synaptic loss using an AAV approach to infect the GABAA receptor?3 subunit (GABAA?3) or the dendrite-selective rabies virus to selectively infect the axon or the dendritic compartments of corticospinal neurons. To examine synaptic efficacy, we will assess excitatory synaptic strength and inhibitory synaptic strength in cortical and spinal circuits with whole-cell patch-clamp and multi-channel intracellular recording. Finally, we will investigate the effects of activation of A2ARs on synaptic remodeling f678ea9f9e

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