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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 injury (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 82138339de

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