SPINAL CORD INJURY INDUCES CHANGES IN ION CHANNELS OF RETICULOSPINAL NEURONS IN LARVAL LAMPREY

Mykola Kovalenko

Dr. Andrew McClellan, Thesis Supervisor

ABSTRACT

After spinal cord injury in larval lamprey, RS neurons regenerate their axons (Davis and McClellan, 1994a,b), restore synaptic contacts and most of the original locomotor functions (McClellan, 1998). It was previously demonstrated that calcium influx in RS neurons in culture results in inhibition of neurite outgrowth (Ryan et al., 2007). There is a hypothesis that injured RS neurons may undergo a number of changes in their electrical properties to maintain relatively low intracellular calcium concentrations and to promote axonal regeneration (Ryan et al., 2007; McClellan et al., 2008).

For uninjured RS neurons, blocking calcium channels with various calcium channel blockers significantly reduced or eliminated the sAHP and produced changes in firing patterns in response to applied depolarizing current pulses that mimicked some of the effects of SCI. Furthermore, blocking SKKCa channels with apamin significantly reduced the sAHP compared to control values. However, blocking calcium and SKKCa channels in injured RS neurons did not have an apparent effect on sAHP and firing properties of the neurons. In addition, computer modeling shown that the AHP and firing properties of axotomized RS neurons could be obtained by substantially reducing calcium and SKKCa channel conductances compared to those in computer model of control RS neurons. This work provides additional information for understanding changes in ion channel expression in RS neurons that might be important to promote axonal regeneration in higher vertebrates, including humans.