Public Abstract
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Title:SPINAL CORD INJURY INDUCES CHANGES IN ION CHANNELS OF RETICULOSPINAL NEURONS IN LARVAL LAMPREY

Spinal cord injury (SCI) induces significant changes in the components of action potential (AP) in identified reticulospinal (RS) neurons in larval lamprey. The fast AHP (fAHP) was significantly larger and the slow AHP (sAHP) was absent or significantly reduced compared to uninjured neurons. Moreover, as a result of SCI and in response to a depolarizing current pulse firing properties of the neurons also undergo a profound change from a smooth train of APs to a single short burst of APs or multiple short bursts.
For uninjured RS neurons, blocking calcium channels with nickel and cadmium, or cobalt, or omega-conotoxin MVIIC 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. The sAHP also was significantly reduced or eliminated by blocking only N-type calcium channels with omega-conotoxin GVIA. Moreover, blocking SKKCa channels with apamin significantly reduced the sAHP compared to control values. In addition, computer modeling shown that the AP and firing patterns of axotomized RS neurons could be obtained by substantially reducing SKKCa and HVA calcium channels conductances compared to those in computer models of uninjured neurons. However, blocking calcium and SKKCa channels in injured RS neurons did not have an apparent effect on the components of AP and firing properties of the neurons.
The sAHP is produced by calcium entering RS neurons during the action potential. Our laboratory has shown that low intracellular calcium concentration is necessary for axonal growth and calcium influx in RS neurons in culture results in inhibition of neurite outgrowth (Ryan et al., 2007). Therefore, injured RS neurons may undergo a number of changes in their electrophysiological properties to maintain relatively low intracellular calcium concentrations and to promote axonal regeneration. Thus, it is very likely that following SCI axotomized RS neurons down-regulate calcium channels to maintain intracellular calcium concentration in a range that is optimal for axonal regeneration. 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.