

Selective plasticity in subpopulations of mouse lamina I GABAergic neurons after chronic spinal injury.

K.J. Dougherty, S. Hochman
Emory University, Dept. of Physiology, Atlanta, GA.

Lamina I is involved in thermal and nociceptive signaling and contains both projection cells and local interneurons that differ in morphology, response modality, and firing characteristics. We examined the cellular properties of spinal GFP expressing GABAergic neurons in normal and spinal cord injured (SCI) mice. GAD67-GFP transgenic mice were used in all studies. Whole-cell patch recordings were obtained from visually-identified GFP⁺ neurons in lamina I of lumbar cord sections from normal and SCI mice (P13-16). SCI mice had a thoracic segment (T8-11) removed 7 days prior to experiments. Following SCI, there were no changes in membrane properties in cells that responded to current injection with a single spike. In contrast, cells displaying tonic/initial burst firing had increased peak inward and steady-state outward currents, increased spike heights, and decreased resistance and time constant values. Moreover, higher firing frequencies were more prevalent after SCI. Spinal cord injury also increased the incidence of cells exhibiting spontaneous plateau potentials. However, voltage ramp protocols failed to activate persistent inward currents that may account for the observed plateaus. Since persistent inward currents emerged following K⁺ channel block, the currents responsible for the observed plateaus are likely dendritic in origin. Both persistent Na⁺ and L-type Ca²⁺ channels contributed to these currents, as demonstrated pharmacologically. In conclusion, there are significant changes in the membrane properties of tonic/initial burst, but not single spike, GABAergic neurons in lamina I following SCI. Persistent inward currents can be activated in GABAergic neurons and changes in these currents may contribute to the increase in excitability and plateaus after SCI.

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