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# Lamina X of the spinal cord in motor neuron disease

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A number of plastic events were described in the spinal cord in the course of amyotrophic lateral sclerosis (ALS). These consist of various morphological effects, involving neurons, glia, and inflammatory cells, as well. Among plastic changes, an increase in neuronal progenitor cells (NPC) occurs within ependymal cells layer of lamina X. This stem cell-like activity is known to be weak in baseline conditions but it is known to increase significantly during spinal cord disorders, when it preferentially generates glial cells, due to the strong gliogenic effect of the spinal cord "milieu". In the present work, we used immunohistochemistry and electron microscopy to analyze cell number within lamina X at the end stage of disease in the G93A mouse model of ALS in baseline conditions and following chronic lithium administration. These cells were identified by using GFAP, bIII-tubulin, NeuN, and calbindin-D28K immunostaining. In the absence of lithium we observed an increase of lamina X cells in ALS mice with a glial phenotype, while in G93A mice treated with lithium these cells differentiate towards neuronal-like phenotype. These effects of lithium are concomitant with slowed disease progression and are reminiscent of the neurogenetic effects described in the sub-ependymal ventricular zone of the hippocampus. The present data confirm the scarce NPC activity in the intact spinal cord which is enhanced by disease conditions; in the presence of chronic lithium, such increased NPCs differentiate towards a neuron-like rather than a glial phenotype.

Keywords
Stem-like cells, ALS, G93A mouse, lithium, glial and neuronal markers, immunohistochemistry.