**Evidence of spinal cord plasticity induced by peripheral nerve grafting in rats**

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**Background**

Following severe spinal cord injury (SCI), functional deficits are typically permanent. These effects cannot solely be attributed to the reduced intrinsic growth capacity of CN axons, as partial functional restoration can be achieved. In particular, surviving neurons are able to regrow their fibers and re-establish functional contacts. Endogenous neurobiological processes, such as neurotrophicity, neuroprotection, neuroplasticity and neurogenesis, are central to protection and recovery in SCI and represent the background of endogenous defence activities (EDA). Brunelli et al (2005) demonstrated that supraspinal motor neurons can target skeletal muscles via peripheral nerve graft (PNG) causing neuromuscular junction switches from cholinergic to glutamatergic neurotransmission. Knowledge regarding the restorative effects of PNG implantation has been used as the basis for experimental procedures to direct the bridging of the SC. However, these procedures have produced contradictory results, and a complete understanding of CNS plasticity after SCI and PNG remains elusive. Our study has proposed investigating the capacity of various CNS neurons to regenerate PNG, motor nerves, and to restore the functional contacts.

**Results**

After laminectomy, a segment of sural autologous nerve graft was implanted in T11-T13 segments of SC, and sutured to the severed obliquus abdominis internus nerve in 30 adult female Sprague-Dawley rats. After 3 months, 7 rats were positive for muscle reinnervation using electromyography. Histology showed axonal degeneration and regeneration in SC, and regeneration of the nerve. The axonal growths around implanted nerve inside SC are favored by Schwann cell proliferation. Neurons that have regenerated PNG were confirmed by Fast Blue. Regularly, these neurons were located in lamina 9, but also in other laminae of SC, and were not detected in the brain. In addition to cholinergic motor neurons, nerve regeneration with other types of spinal neurons (sensitive, interneurons) was proved by immunolocalization of vesicular glutamate transporter 2 in some axons of regenerated motor nerves and by immunodetection of both types of neurotransmission, cholinergic and glutamatergic, in the reinnervated muscle.

**Conclusions**

Our study demonstrates the ability of lower motor neurons and other types of spinal neurons to produce an axonal growth to regenerate peripheral nerve graft. Moreover, the upper motor neurons were not demonstrated to have this capacity. These findings do not contradict the *Brunelli’s Paradigm*, but sustain the hypothesis that PNG's regeneration can be supported by different types of spinal neurons (motor, sensitive, and interneurons) and the coexistence of the two neurotransmitter types should not be surprising. Also, our results support the idea of neuronal plasticity in both the brain and the SC by endogenous defence activities following PNG in SC.