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Neuregulin1/ErbB expression regulation in the injured rat peripheral nerve during degeneration and regeneration

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(Article begins on next page)

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Presentation Abstract

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Abstract:	Following injury to peripheral nerves, Schwann cells dedifferentiate, proliferate, migrate and, when axon regeneration occurs, redifferentiate into a myelinating phenotype. Schwann cells promote regeneration through the creation of a permissive environment for axon regrowth and the production of neurotrophic factors. Neuregulin1 (NRG1) is a polymorphic factor involved in the myelination and remyelination processes. Through three injury models, the expression of different NRG1 isoforms and of their ErbB receptors was investigated in the distal portion of the rat median nerve under degenerating conditions (unrepaired cut nerve) or under regenerating conditions after a mild (nerve crush) and more severe (end-to-end repair of cut nerve) injury. Following peripheral nerve injury, distinct and consecutive phases occur: nerve degeneration, axonal regrowth, nerve regeneration and maturation. A detailed mRNA and protein expression analysis of the NRG1/ErbB system was carried out, focusing the attention not only on soluble and transmembrane NRG1 isoforms, but also on alpha and beta as well as type a, b, and c isoforms. Expression specificity during the distinct and consecutive phases occurring after nerve injury and regeneration or the progress in nerve degeneration was observed. Nerve ultrastructure changes were evaluated by electron microscopy and related to the results of mRNA and protein expression analyses. At the

mRNA level, soluble NRG1 isoforms alpha and beta, as well as type a and b, are strongly up-regulated early, during nerve degeneration and the early phases of axonal regrowth, but their expression does not seem to be differentially regulated under regeneration and degeneration conditions. ErbB receptors are strongly regulated in the different phases, but at the protein level ErbBs are similarly regulated in the different injury models. On the contrary, we observed that transmembrane NRG1 isoforms are differentially regulated, at the protein level, under degeneration and regeneration conditions, thus suggesting that their expression could be a good marker to follow and monitor the regeneration process. This accurate regulation suggests that each member of the NRG1/ErbB system plays a specific role following nerve injury, that could be clinically exploited to promote nerve regeneration. This project has received funding from the European Union's Seventh Programme for research, technological development and demonstration.

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NERVE INJURY

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