GLIAL CELLS AND THEIR MOLECULES IN NERVE REGENERATION

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Peripheral nerve injury has many causes ranging from traumatic damage to metabolic disturbances (e.g.in diabetes mellitus). The primary pathology can affect axons (nerve transection) or Schwann cells, and their interdependence means that injury to one will ultimately affect the other. Fortunately, the PNS has a significant capacity for repair, and axons are able to regenerate over long distances and are also capable of remyelination. This repair process leads to appreciable recovery of function after damage, but the clinical outcome after nerve injury often remains poor. Traumatic nerve injury provides a model for the analyses of biological pathways important for nerve degeneration and repair, and the stereotypic events allow the quantitation of degeneration/regeneration. Especially useful is the mouse as a model and including mutant mice in these experiments has shed some light into the functional aspects of several molecules. An interesting concept that emerged over the last decade is that Schwann cells in the distal nerve stump display unique characteristics and were also called “repair cells” so do not simply revert to an earlier developmental stage as previously suggested. Characterizing the properties of these cells and identify molecules expressed in these cells may provide us with useful candidates for enhancing the endogenous regenerative potential and thus improve clinical outcome.