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Why neuroregeneration in CNS is slow?

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The environment within the CNS, especially following trauma, counteracts the repair of myelin and neurons. Growth factors are not expressed or re-expressed; for instance, the extracellular matrix lacks laminins. Glial scars rapidly form, and the glia actually produce factors that inhibit remyelination and axon repair. For instance, chondroitin sulfate proteoglycans (CSPGs): astrocytes up regulate the production of chondroitin sulfate proteoglycans. Keratan sulfate proteoglycans or keratosulfate (KSPG): reactive astrocytes up regulate the chondroitin sulfate proteoglycans as part of glial scar formation and inhibit neurite outgrowth extension, limiting nerve regeneration. Proteins of oligodendritic or glial debris origin that influence neuroregeneration including NOGO: the protein family Nogo, especially a myelin-associated neurite outgrowth inhibitor (Nogo-A) acts as an inhibitor of remyelination in the CNS; antagonising this inhibitor results in improved remyelination, as it is involved in the RhoA pathway. NI-35 (neurite growth inhibitor) a non-permissive growth factor from myelin, MAG (myelin-associated glycoprotein): acts via the receptors NgR2, GT1b, NgR1, p75, TROY and LINGO1, OMgp (oligodendrocyte myelin glycoprotein), ephrin B3 inhibits remyelination and Sema 4D (semaphorin 4D) inhibits remyelination. Sema 3A (semaphorin 3A) is present in the scar that forms in both the central nervous system and peripheral nerve injuries, and contributes to the outgrowth-inhibitory properties of these scars. The axons themselves also lose the potential for growth with age, due to a decrease in GAP 43 (growth associated protein 43) expression among others.

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