**REMYELINATION IN ADULT ANIMAL: NEW APPROACHES FOR MYELIN SHEATH RECOVERY**

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**INTRODUCTION**

Myelin is composed of several layers of lipid-rich membrane that protects the axons enveloping them in both Central Nervous System (CNS) and Peripheral Nervous System (PNS). In CNS, it is produced by oligodendrocytes derived from NG2-glia (polydendrocytes) and its main function is to facilitate the action potentials propagation.

Loss of myelin, called demyelination, produces functional deficits and may be due to several factors including genetic factors, infectious agents, immunoreactivity and trauma. Demyelination causes an imbalance of axons homeostasis and consequently, they are more susceptible to degeneration.

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**OBJECTIVES**

- Identification of factors that trigger or difficult physiological remyelination.
- Identification of elements that cause differentiation from polydendrocytes to oligodendrocytes.
- Promote polydendrocytes differentiation to establish a therapy.

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**REMYELINATION**

Polydendrocytes react to any type of demyelinating lesion by changing their morphology and proliferation rate, they migrate to the injured area to repair damaged axons. However, while remyelination in acute lesions is quite successful, it becomes unsatisfactory in chronic lesions as it happens in demyelinating diseases. There are some hypotheses about this fact:

- Polydendrocytes migrate to injured area, but there are lacks of molecular signals, growth factors or an inhibitory environment around the lesion.
- With chronicity, there is an exhaustion of progenitor cells.
- NG2-glia population is heterogeneous in their differentiation capacity.
- Degeneration of demyelinated neurons.
- Presence of antibodies anti-NG2 that harm or destroy the NG2-glia.

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**CONCLUSIONS**

Polydendrocytes represent a promising therapeutic target because oligodendrocytes derived from them protect axons integrity; however, further studies are needed to know all details about its molecular mechanisms, their recruitment and differentiation.

Numerous extrinsic signals have been identified that can influence polydendrocytes proliferation such as paracrine factors, neurotransmitters, molecules on the cell surface and extracellular matrix and interactions between neurons and NG2-glia.

Replacement of treatments that require cell/genetic manipulation for drug delivery is a very promising therapeutic strategy because it would provide better accessibility, easier and cheaper treatment.