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Titel des Beitrags: Accelerated remyelination during inflammatory demyelination prevents axonal loss and improves functional recovery.

Abstract: Demyelination in MS disrupts nerve signals and contributes to axon degeneration. While remyelination promises to restore lost function, it remains unclear whether remyelination will prevent axonal loss. Inflammatory demyelination is accompanied by significant neuronal loss in the experimental autoimmune encephalomyelitis (EAE) mouse model and evidence for remyelination in this model is complicated by ongoing inflammation, degeneration and possible remyelination. Demonstrating the functional significance of remyelination necessitates selectively altering the timing of remyelination relative to inflammation and degeneration. We demonstrate accelerated remyelination after EAE induction by direct lineage analysis and hypothesize that newly formed myelin remains stable at the height of inflammation due in part to the absence of MOG expression in immature myelin. Oligodendroglial-specific genetic ablation of the M1 muscarinic receptor, a potent negative regulator of oligodendrocyte differentiation and myelination, results in accelerated remyelination, preventing axonal loss.
and improving functional recovery. Together our findings demonstrate that accelerated remyelination supports axonal integrity and neuronal function after inflammatory demyelination.