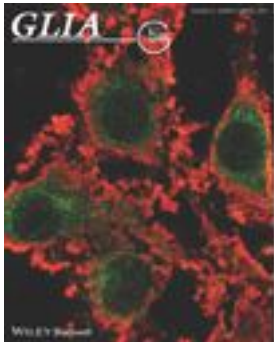


Work in [Prof Carmen Melendez's](#) lab has recently been published in the journal GLIA

[Glia.](#) 2014 Jan 28. doi: 10.1002/glia.22627. [Epub ahead of print]



Accelerated repair of demyelinated CNS lesions in the absence of non-muscle myosin IIB.

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Abstract

The oligodendrocyte (OL), the myelinating cell of the central nervous system, undergoes dramatic changes in the organization of its cytoskeleton as it differentiates from a precursor (oligodendrocyte precursor cells) to a myelin-forming cell. These changes include an increase in its branching cell processes, a phenomenon necessary for OL to myelinate multiple axon segments. We have previously shown that levels and activity of non-muscle myosin II (NMII), a regulator of cytoskeletal contractility, decrease as a function of differentiation and that inhibition

of NMII increases branching and myelination of OL in coculture with neurons. We have also found that mixed glial cell cultures derived from NMIIIB knockout mice display an increase in mature myelin basic protein-expressing OL compared with wild-type cultures. We have now extended our studies to investigate the role of NMIIIB ablation on myelin repair following focal demyelination by lysolecithin. To this end, we generated an oligodendrocyte-specific inducible knockout model using a Plp-driven promoter in combination with a temporally activated CRE-ER fusion protein. Our data indicate that conditional ablation of NMII in adult mouse brain, expedites lesion resolution and remyelination by Plp+ oligodendrocyte-lineage cells when compared with that observed in control brains. Taken together, these data validate the function of NMII as that of a negative regulator of OL myelination in vivo and provide a novel target for promoting myelin repair in conditions such as multiple sclerosis. GLIA 2014.

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