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Targeting remyelination treatment for multiple sclerosis

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Abstract

Since disability in multiple sclerosis (MS) is a product of neurodegeneration and deficient remyelination, the ability to enhance neuroregeneration and myelin regeneration in MS is an enticing goal for MS drug development. In particular, remyelination treatments could promote return of neurological function and also prevent further axonal loss and neurodegeneration in MS due to trophic effects of myelin. The study of remyelination has advanced dramatically in the last several years such that a number of pathways inhibiting remyelination have been discovered, including those involving LINGO-1, Notch-1, hyaluronan, RXR receptor, and wnt/ß-catenin. Other approaches such as high throughput drug screening for remyelination drugs have caught fire, with identification of dozens of known drugs with oligodendrocyte maturation stimulatory effects. Several drugs identified through screens and other mechanisms are in the process of being further evaluated for remyelination in MS and MS models. We discuss the potential molecular targets and the