Promoting remyelination as a novel therapy in multiple sclerosis

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Multiple sclerosis (MS) is a chronic inflammatory disease of central nervous system. Myelin destruction due to inflammatory oligodendrocyte cell damage or death in conjunction with axonal degeneration are among the main pathological hallmarks of MS. Current therapies used to treat MS patients generally act by modulation or suppression of the immune system to reduce relapse rates and magnetic resonance imaging lesions. But these medications are not useful during progressive phase of MS when axonal degeneration plays the main role. Recent researches on oligodendroglial precursor cells (OPCs) with the aim of promoting remyelination have shown promising results. It seems that the property to remyelinate, demyelinated axons can protect them from secondary degeneration and cause clinical remission in relapsing-remitting form of disease. Fingolimod, benztropine, BIIB033, rHigM22, GNbAC1, IRX4204 and a few other medications have been efficient in increasing remyelination by promoting OPC differentiation and enhancement of myelin sheath formation in experimental models. Although many more clinical trials are needed to determine their potential capability for future MS therapy. In this review we aimed to investigate the role of promoting remyelination as a novel therapy in MS and to discuss about the efficacy of existing medications with the same mechanism.

Key words: Multiple sclerosis, oligodendrocyte, remyelination, treatment