Multiple sclerosis (MS) is a demyelinating disease of the central nervous system (CNS), characterized by chronic inflammation and neurodegeneration. Myelin is an insulating and protective layer that is wrapped around axons by oligodendrocytes that enables fast signal transmission between neurons. In MS demyelination is induced by an unknown trigger, resulting in the appearance of lesions. Changes in the lesion environment prevent efficient remyelination to proceed. This leads to a multitude of cognitive and physical symptoms. Fibronectin accumulates within lesions, and negatively affects remyelination. Previously, we demonstrated that a small molecule (GD1a) overcomes the remyelination-inhibiting effect of fibronectin. Treating progressive MS is difficult because of the existence of a tight layer of cells, the blood brain barrier (BBB) that separate the brain from the periphery. In this thesis we aimed to investigate modes of transporting GD1a from the periphery to the brain, while retaining its therapeutic effectiveness. GD1a-loaded and brain-targeted nanoparticles were efficiently transported across an in vitro BBB, but could not significantly improve myelin membrane formation, as GD1a likely exerts its restorative effect at the cell surface. We elucidated two interaction partners via which GD1a induces myelin membrane formation in the presence of fibronectin. As our findings also hint to potential inherent differences between an MS-BBB and their sibling-BBB in response to inflammation, inclusion of MS-derived cells is crucial to determine the success of brain delivery of remyelination therapeutics.