J Alzheimers Dis. 2017 Apr 28. doi: 10.3233/JAD-170036. [Epub ahead of print]

Remyelination: A Potential Therapeutic Strategy for Alzheimer's Disease?

Sun J, Zhou H, Bai F, Zhang Z, Ren Q.

Abstract

Myelin is a lipid-rich multilamellar membrane that wraps around long segments of neuronal axons and it increases the conduction of action potentials, transports the necessary trophic support to the neuronal axons, and reduces the energy consumed by the neuronal axons. Together with axons, myelin is a prerequisite for the higher functions of the central nervous system and complex forms of network integration. Myelin impairments have been suggested to lead to neuronal dysfunction and cognitive decline. Accumulating evidence, including brain imaging and postmortem and genetic association studies, has implicated myelin impairments in **Alzheimer's disease** (AD). Increasing data link myelin impairments with amyloid-β (Aβ) plaques and tau hyperphosphorylation, which are both present in patients with AD. Moreover, aging and apolipoprotein E (ApoE) may be involved in the myelin impairments observed in patients with AD. Decreased neuronal activity, increased Aβ levels, and inflammation further damage myelin in patients with AD. Furthermore, treatments that promote myelination contribute to the recovery of neuronal function and improve cognition. Therefore, strategies targeting myelin impairment may provide therapeutic opportunities for patients with AD.

KEYWORDS: Aging; **Alzheimer**'s **disease**; ApoE4; cognition; **demyelination**; remyelination

PMID: 28453483 DOI: 10.3233/JAD-170036