1. No.
   2022Y011

2. Title
   Therapeutic Strategies for Multiple Sclerosis by Targeting Nicotinic Acetylcholine Receptor in Oligodendrocyte Progenitor Cells

3. Period
   2022-2024

4. Summary
   This study aimed to elucidate the function of nAChRs expressed in OPCs and establish therapeutic strategies for multiple sclerosis by targeting nAChRs in OPCs. Nicotine was shown to promote OPC proliferation and differentiation into OLGs using primary cultured OPCs. It was demonstrated that nicotine-induced differentiation of OPCs into OLGs is mediated through nAChRs expressed in OPCs, particularly via the α7 nAChR subunit. Additionally, α4β2 nAChR inhibited the differentiation of OPCs into astrocytes. In an *in vivo* experiment using a CPZ-induced demyelination model mouse, nicotine administration via drinking water restored coordination deficits and increased OLG expression, suggesting that nicotine promotes remyelination *in vivo*. Furthermore, nicotine administration suppressed microglial and astrocyte activation during the early stages of demyelination, indicating that nicotine promotes remyelination by acting on OPCs and modulating other glial cells. The findings of this study contribute to developing a novel therapeutic approach for multiple sclerosis by targeting nAChRs in OPCs to enhance remyelination.