1. **No.**

   FP01912180

2. **Title**

   Role of Nicotinic Receptor in Neuroprotection and Neuroregeneration Mediated by Neuron-glia Interaction

3. **Period**

   2016-2020

4. **Summary**

   As a result of investigating the effect of nicotine receptor (nAChR) stimulation on the activation of microglia *in vivo*, the microglia activation in the brain by Aβ treatment was suppressed by the administration of nicotine. Nicotine also reduced the proliferation of neural stem cells by Aβ. *In vitro* study using primary cultured microglia, nAChR stimulation by donepezil suppressed the activation of microglia accompanied by suppression of inflammatory cytokine release. On the other hand, nAChR stimulation failed to suppress LPS-induced microglial activation in BV2 cells. Nicotine and donepezil increased the production of hydrogen sulfide in cultured cerebral cortical neurons. Hydrogen sulfide produced by 3-MST is involved in the neuroprotective effect of nAChR stimulation. Hydrogen sulfide would exert a neuroprotective effect mediated by the PI3K / Akt pathway.

   In the experiments of tau phosphorylation, nAChR stimulation inhibited tau phosphorylation caused by low temperature treatment in both *in vivo* and *in vitro* experimental systems.