1. No.
   2020G026

2. Title
   Signaling Pathway for Cigarette Smoking-induced Neuroinflammation Caused by Dysregulated Glycolipids Metabolism in Neuron/Glial Cells

3. Period
   2020-2022

4. Summary
   In the present study, we explored whether smoking, especially main constituents of smoking, nicotine (Nic) affects normal neuronal and glial cell biology and functions. For these purposes, we examined the effects on cell morphology and intracellular signaling pathway such as autophagy and nicotinic acethylcholine receptor-mediated pathway in KT-5 astroglial cells and Trk-neutrophin receptor-mediated intracellular signaling pathway in PCT neuronal cells. The data strongly suggest that Nic has neuroprotective effects on neuronal cells through the augmentation of NGF-induced Trk autophosphorylation responses. Nic also has profound impacts on lipid metabolism, especially neutral glycolipids in KT-5 astroglial cells. Moreover, Nic induces intracellular ROS production and its main site for the ROS production seemed to be through mitochondria, because mitochondria-targeted antioxidant, Mito-TEMPO completely inhibited Nic-induced ROS production in KT-5 cells. Therefore, Nic might affect normal mitochondrial function. Nic also elicited profound effects on sphingolipids and glycolipids metabolism. These effects might be dependent on the treating time with Nic. In summary, Nic provoked various influences on the intracellular signaling machinery and lipids metabolism as well.