1. No.
   2020G028

2. Title
   Elucidation of the Mechanism of Nicotinic Actions Induced in Prenatal Stress Model Animals with Schizophrenia and Autism-like Findings

3. Period
   2020-2022

4. Summary
   In 2020, we will create a "two-hit model" that applies restraint stress to the maternal body of heterozygous fetuses lacking the GABA synthase gene Gad1. An *in vitro* physiological functional analysis of cortical pyramidal cells was performed, and the results indicated that glutamate was not involved in increasing the excitability of GABA cells, suggesting that ACh-ergic inputs may be involved. In 2021, we will conduct gene expression analysis of model mouse fetal brain, immunohistochemical analysis of model mouse adult brain, and *in vitro* physiological function analysis of model mouse cerebral cortical GABA cells. We conclude that a shift in ACh cell differentiation, rather than a developmental shift (GABA cell depletion and ACh cell increase), results in increased ACh axonal projection to the mPFC. In addition, the enhancement of GABA synaptic currents in pyramidal cells is not due to increased glutamatergic excitatory input to GABA cells, but due to increased ACh input, resulting in increased excitability of GABA cells. showed the possibility that in 2022, we identified ACh receptors that change the membrane properties of model mouse cerebral cortex GABA cells.