1. No.
   2019G033

2. Title
   Reversal of cognitive phenotypes by nicotine in a mouse model of schizophrenia

3. Period
   2019-2021

4. Summary
   Patients with psychiatric disorders, especially schizophrenia, show a substantially higher smoking rate (70-90%) than the general population (25-30%). There are no sufficient findings that directly suggest nicotinic signaling in the pathophysiology of schizophrenia, nor have there been reports using established disease mouse models. The purpose of this study is to use a well-established 22q11.2 deletion syndrome schizophrenia disease mouse model *Df(16)A+/-* , and is to clarify how smoking (nicotine) alleviates cognitive dysfunction of social memory and behavioral abnormalities to stress such as fear and anxiety, by anatomical and functional analysis of neural circuits.

   Df(16)A+/- mice have severely impaired social and working memory. It was shown that long-term administration of nicotine could rescue the impaired social memory, but not working memory in *Df(16)A+/-*. Loss of social cognition in a mouse model of SCZ with 22q11.2 deletion syndrome may be ameliorated by long-term nicotine administration, and adulthood intervention alleviates cognitive impairment, and may serve as an alternative or augmentation of current treatments.