1. No.
   2020G020

2. Title
   Elucidation of the Molecular Mechanism by which Smoking Relieves Intestinal Inflammation

3. Period
   2020-2022

4. Summary
   Epidemiological studies of ulcerative colitis and smoking habits have reported that nicotine may enhance the intestinal barrier. In previous studies, nicotine has been reported to improve or impair tight junction barrier function. In the present study, we found that when nicotine was added at various concentrations to CaCO-2 (gastrointestinal tract-derived epithelial cells) and EpH4 (mammary gland-derived epithelial cells), the barrier function was enhanced in CaCO-2 cells and reduced in EpH4 cells. The different effects of nicotine on tight junctions observed in these cells may be due to differences in the isoforms of claudin expressed in these cells. To analyze the differential effects of nicotine on the various claudin isoforms, we established an epithelial cell line in which expression of all claudin isoforms was knocked out (claudin-null cells). Then, we established claudin-null cells expressing each single claudin isoform to comprehensively elucidate the effects of nicotine treatment on localization of each claudin isoforms and to elucidate the molecular mechanisms by which nicotine affects the epithelial barrier. We would like to elucidate the molecular mechanisms by which nicotine alters the epithelial barrier.