1. **No.**

   FP01805065

2. **Title**

   The role of α7nACh receptor on the host-microbita interactions in colitis

3. **Period**

   2017-2019

4. **Summary**

   The pathogenesis of ulcerative colitis is not fully elucidated, and some cases are resistant to current therapies, including 5-ASA, steroid, immunosuppressant, and molecular targeting drugs. Smoking has been known to reduce the incidence and severity of ulcerative colitis, but therapeutic effect of nicotine, one of ingredient of cigarette, is not proved. In order to establish α7nAChR targeting strategy for ulcerative colitis therapy, we tried to elucidate the role of α7nAChR in murine colitis.

   Expression of multiple nAChRs, including α7nAChR was observed in mouse small intestine and colon. Administration of α7nAChR agonist to colitis mice induced restoration of goblet cells, decrease of lamina propria DCs and macrophages, indicating attenuated colitis. But it did not alter fecal microbial composition in colitic mice. Nicotine treatment to organoid-BMDC coculture attenuated cystic change of organoids, depending on α7nAChR of BMDC. α7nAChR deletion of IL-10KO DCs exacerbated colitis.

   These results suggest α7nAChR signaling on dendritic cells would be an attractive therapeutic target for the treatment of inflammatory bowel disease.