1. No.
   2022G016

2. Title
   Role of Galectin-3 in Smoking-Induced Ferroptosis; a Comparative Study of Combustion Cigarette and Heated Tobacco Product

3. Period
   2022-2025

4. Summary
   This study revealed the following things: 1) the expression of genes involved in ferroptosis is enhanced in the lungs of rats exposed to combustible cigarettes; however, the effect of heat-non-burn cigarette smoking is limited. 2) galectin-3, a galactose-recognizing lectin, significantly increased in airway epithelial cells and alveolar macrophages after combustible cigarette exposure, suggesting its involvement in the oxidative stress following tobacco smoke exposure. On the other hand, 3) analysis using human alveolar basal epithelial adenocarcinoma A549 cells showed that galectin-3 is not involved in cell death induced by ferroptosis. Furthermore, 4) administration of IKE resulted in enhanced expression of galectin-3 in Kupffer cells in the liver. In addition, 5) galectin-3-deficient mice had low dROM values, an indicator of oxidative stress, suggesting that galectin-3 may function as a factor that exacerbates oxidative stress induced by tobacco smoke exposure and ferroptosis induction. We plan to conduct analyses using tobacco smoke extracts and *in vivo* experiments on galectin-3-deficient mice, to reveal the role of galectin-3 in oxidative stress responses such as ferroptosis caused by tobacco smoke exposure with special reference to the differences in biological effects between combustible cigarettes and heat-non-burn cigarettes.