1. No.
   2021G016

2. Title
   The effect of a novel receptor involved in immune regulation on nicotine and inflammatory response of pulmonary endothelial cells

3. Period
   2021-2023

4. Summary
   In this study, we show that exposure of lung endothelial cells to cigarette smoke extracts (CSE) reduces the mRNA and protein expression of Gpr116 as well as Gpr116-mediated calcium signaling. We also show that Gpr116 is likely to be essential for mechano-sensing and signal transduction of endothelial cells. CSE-induced reduced function of Gpr116 may disturb the homeostasis of endothelial cell functions, leading to inflammatory response. In addition, we show that downregulation of Gpr116 results in increased expression of P-selectin protein, a regulator of endothelial immune response, in lung endothelial cells. These results indicate that Gpr116 plays an important role in the regulation of immune homeostasis of lung endothelial cells, and will provide a better understanding of the role of lung endothelial cells in development and exacerbation of COPD. Further studies should be required to elucidate the effects of Gpr116-mediated mechano-sensing and signaling on the regulation of homeostasis of endothelial cell functions and immune response.