1. **No.**

   FP01903066

2. **Title**

   Evaluation of Role of microRNA Affected by Nicotine in the Kidney and Vessel

3. **Period**

   2017-2020

4. **Summary**

When cultured podocytes were co-administered with TGF-β1 and nicotine, miR-26a and miR-155 were decreased compared to controls. miR-155 was further decreased compared to TGF-β stimulation alone, and TGF-β1 and COL1A1 mRNAs were decreased. In cultured HUVECs, miR-26a and miR-155 were not changed by the same stimuli. To explain the changes in miRNAs and mRNAs in cultured podocytes, we focused on SMAD2 and SMAD3, which are downstream of TGF-β. Nicotine decreased phosphorylated SMAD3 and tended to decrease phosphorylated SMAD2 under TGF-β1 stimulation compared to controls. miR-155 mimic decreased phosphorylated SMAD2 and increased COL1A1 mRNA expression under nicotine condition, suggesting that miR-155 increases COL1A1 expression by repressing SMAD2, since miR-155 targets SMAD2. COL1A1 was not altered in miR-155 inhibitor. When heart failure mice were administered with nicotine, miR-26a and miR-155 in the whole kidney were examined, but no change was observed, and no difference in COL1A1 gene expression was observed. These results suggest that in cultured podocytes, nicotine may decrease miR-155 under TGF-β stimulation and decrease COL1A1 gene expression by increasing its target, SMAD2.