1. No.
   2022Y007

2. Title
   The Effect of Polycyclic Aromatic Hydrocarbons from Cigarette Smoke on the Progression of NASH

3. Period
   2022-2024

4. Summary
   Preliminary studies suggest that "the suppression of NASH fibrosis by hepatic AhR" is mediated by induction of expression of FSP27β through directly AhR activation by dietary or endogenous ligands. On the other hand, low concentrations of BaP were shown to suppress the development of NASH without mediating hepatocyte AhR. The molecular mechanism by which low concentration of BaP exposure suppressed NASH fibrosis were involved in the bone marrow-derived macrophage infiltration and increased Tregs. On the other hand, there was little effect of low levels of BaP on fat denaturation and lipid metabolism. However, this is unlikely to be related to macrophage AhR activation by dietary and endogenous AhR ligands. Macrophage AhR activation by low concentrations of BaP is currently being analyzed. Although the epidemiological study shows that tobacco smoke exacerbates NAFLD/NASH, this effect was not observed in mice, suggesting that the AhR sensitivity and activation potential by PAHs and tobacco sensitivity are likely to differ according to race, sex, and age, *etc*.