Treatment of cigarette smoke condensate accelerates nonalcoholic steatohepatitis *in vitro*

Zixiong Zhou[1], Kyuhong Lee[2] and Bumseok Kim[1]
[1]Chonbuk National University, South Korea
[2]Korea Institute of Toxicology, South Korea

It has been well known that Cigarette Smoke (CS) is a leading cause of various diseases worldwide. Recently, cumulative evidence has suggested that exposure to CS detrimentally a ects the pathogenesis of several chronic liver diseases, including nonalcoholic fatty liver disease (NAFLD). Nonalcoholic steatohepatitis (NASH), more severe stage of NAFLD, is characterized by steatosis, hepatocellular ballooning degeneration and lobular in ammation. Relationship between CS exposure and progression of NASH has not been fully understood. erefore, the purpose of this study was to evaluate the e ects of CS extract (CSE) or CS condensate (CSC) on the , NASH model using mouse primary hepatocytes (HPs) treated with palmitic acid (PA) or PA plus LPS. Increased hepatocellular damage was observed in PA-treated HPs with CSC or CSE treatment, but increased triglyceride level was only observed in PA-treated HPs with high concentration CSC. Also, expression levels of NASH-related genes such as in ammation, oxidative stress and lipogenesis were signi cantly increased by treatment of CS. In order to more clearly demonstrate the e ects of CSE or CSC, we used trans-well co-culture system of HPs and Kup er cells (KCs) under the same condition of above mentioned. e levels of in ammatory cytokines and oxidative stress-related gene were markedly increased in co-cultured KCs with treatment of CSE or CSC. Furthermore, treatment of CSC or CSE signi cantly augmented the expression levels of KC activation markers including CD14 and CD68. Interestingly, each type of CS could not a ect HPs apoptosis when only HPs were cultured; however, CS increased PA-induced HPs apoptosis when HPs were co-cultured with KCs. Overall, our current ndings indicate that , treatment of CSE or CSC di erentially contributes to the severity of NASH by modulating NASH-related hepatocellular lipotoxicity and in ammation. ese e ects might be caused by KCs activation, subsequently inducing HPs apoptosis.

References

1.

Notes: