

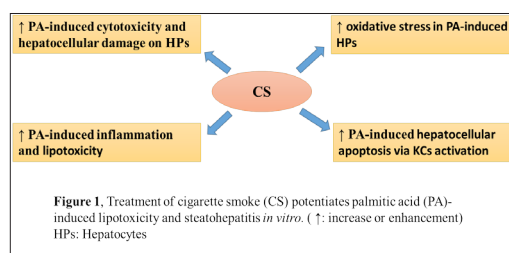
14th World Congress on

TOXICOLOGY AND PHARMACOLOGY

March 12-14, 2018 Singapore

Treatment of cigarette smoke condensate accelerates nonalcoholic steatohepatitis *in vitro*Zixiong Zhou¹, Kyuhong Lee² and Bumseok Kim¹¹Chonbuk National University, South Korea²Korea 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 affects 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 inflammation. Relationship between CS exposure and progression of NASH has not been fully understood. Therefore, the purpose of this study was to evaluate the effects of CS extract (CSE) or CS condensate (CSC) on the *in vitro* 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 inflammation, oxidative stress and lipogenesis were significantly increased by treatment of CS. In order to more clearly demonstrate the effects of CSE or CSC, we used trans-well co-culture system of HPs and Kupffer cells (KCs) under the same condition of above mentioned. The levels of inflammatory 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 significantly augmented the expression levels of KC activation markers including CD14 and CD68. Interestingly, each type of CS could not affect HPs apoptosis when only HPs were cultured; however, CS increased PA-induced HPs apoptosis when HPs were co-cultured with KCs. Overall, our current findings indicate that *in vitro* treatment of CSE or CSC differentially contributes to the severity of NASH by modulating NASH-related hepatocellular lipotoxicity and inflammation. These effects might be caused by KCs activation, subsequently inducing HPs apoptosis.



References

1. Park S, et al. (2016) Mainstream cigarette smoke accelerates the progression of nonalcoholic steatohepatitis by modulating Kupffer cell-mediated hepatocellular apoptosis in adolescent mice. *Toxicology Letters*; 256: 53-63.
2. Bataller R (2006) Time to ban smoking in patients with chronic liver diseases. *Hepatology*; 44(6): 1394-1396.
3. Hezode C, et al. (2005) Daily cannabis smoking as a risk factor for progression of fibrosis in chronic hepatitis C. *Hepatology*; 42(1): 63-71.
4. Zein C O (2010) Clearing the smoke in chronic liver diseases. *Hepatology*; 51(5): 1487-1490.
5. Sanyal A J (2005) Mechanisms of Disease: pathogenesis of nonalcoholic fatty liver disease. *Nature Clinical Practice Gastroenterology & Hepatology*; 2(1): 46-53.

Biography

Zixiong Zhou has received his BS degree in 2014 from Southwest Minzu University of China and obtained MS degree from Chonbuk National University, South Korea in 2017. Currently he is pursuing his Doctoral degree at Chonbuk National University. He is focusing on various research topics including effects of cigarette smoke components to liver disease and Acetaminophen induced hepato-toxicology.

Notes:

zzxaiqj@gmail.com