

## **Chronic Inhalation of Pod-based e-cigarette Aerosols on Inflammatory Biomarkers in the Central Nervous and Peripheral Systems**

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### **ABSTRACT**

Electronic cigarettes (e-cigs) use has been dramatically increased recently, especially among youths. In this study, we evaluated the effect of one- and three-months continuous exposure to e-cig vapor (JUUL pods), containing high nicotine concentration, on the expression of glutamate receptors and transporters and mainly the associated neuroinflammatory markers in drug reward brain regions such as nucleus accumbens (NAc) core (NAc-core), NAc shell (NAc-shell) and hippocampus (HIP) in female C57BL/6 mice. We revealed that three months' exposure to mint or mango flavored-JUUL (containing 5% nicotine, 59 mg/ml) induced upregulation in metabotropic glutamate receptor 1 (mGluR1) and postsynaptic density protein 95 (phosphorylated and total PSD95) expression, and downregulation of mGluR5 and glutamate transporter 1 (GLT-1) in the NAc-shell. Three months' exposure to JUUL induced upregulation of mGluR5 and GLT-1 expression in the HIP. We further revealed that mice exposed to JUUL Mango and JUUL Mint for one month had significantly increased TNF- $\alpha$  expression in the NAc-core and NAc-shell, with elevation in IL-1 $\beta$  as well. NAc-shell also showed significantly increased levels of IL-6 and HMGB-1 for both flavors. There were significantly increased levels of TNF- $\alpha$  in the NAc-core and NAc-shell for both Mint- and Mango-exposed JUUL mice for three months. NAc-shell also showed significantly increased levels of IL-1 $\beta$ , IL-6, HMGB-1, and RAGE after three months of JUUL exposure, while the hippocampus showed significantly decreased levels of HMGB-1 for both flavors. These findings demonstrated that exposure to e-cig vapor containing high nicotine concentrations induced differential effects on glutamatergic system as well as induction of proinflammatory markers in the brain.