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**Title:** The effects of diesel exhausted particles on the oxidative systems in smoking-induced emphysema model

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**Body:** Introduction: Diesel exhausted particles (DEP) is one of major constituent in air pollution which has been known as an important cause of acute exacerbation of COPD. The role of oxidative stress is unknown in acute exacerbation of COPD. We hypothesized the responses of DEP are different between in emphysema and non-emphysema murine model in terms of oxidative systems. Methods: Smoking-induced emphysema models were exposed to cigarettes for 6 months. Suspended DEP were given intratracheally after 0, 2, 4, 6 days after last expose to smoking. The mice were sacrificed 3 days after last DEP instillation. Bronchoalveolar lavage (BAL), mean liner intercept (MLI), ELISA for TNF- $\alpha$  and IL-8, Western blot for PCNA, HNE, Nrf-2, and Tunnel assay were performed. Results: MLI significantly more increased in emphysema group compared to those in control group ( $36.7 \pm 3.5$  vs  $26.5 \pm 6.5$   $\mu$ m, respectively) and significantly distended alveoli were observed in emphysema group. Total cell count and percentages of lymphocyte and neutrophil in BAL fluid significantly increased by DEP treatment. The amounts of IL-8 and TNF- $\alpha$  in BAL fluid were not different by treatment of DEP. The expressions of PCNA were increased and expressions of Nrf-2 were depressed by treatment of DEP in both emphysema and control group. The expression of PCNA and Nrf-2 were more increased by treatment of DEP in emphysema group compared to that in control group. The expressions of HNE were depressed by treatment of DEP in both emphysema and control group. Apoptosis were significantly increased in emphysema group by treatment of DEP. Conclusion: The oxidative systems are more exaggerated by DEP in emphysema model.