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Title: Diesel exhaust particle induce epithelial-to-mesenchymal transition by oxidative stress in human bronchial epithelial cell

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Body: [Background] Epithelial-mesenchymal transition (EMT) process is recapitulated in adults during wound healing, tissue regeneration, and organ fibrosis and cancer progression. Diesel exhaust particle (DEP) is the major components of PM2.5, and much attention has focused on PM2.5 in relation to many pulmonary diseases. Studies of molecular mechanisms have focused on the role of reactive oxygen species (ROS) generated directly and indirectly by exposure to DEP. [Objective]The current study was designed to explore the role of DEP whether or not induce EMT process in airway epithelial cells by oxidative stress. [Methods] We used human bronchial epithelial cell line BET-1A. In the first experiment, DEP (Standard Reference Material 2975) was treated culture cells with various concentrations for 24h. In the second experiment, different concentrations of N-acetylcysteine (NAC) were pretreated culture cells for 24h, and then DEP was treated with 25μg/ml. After DEP exposure 24h, the cells were harvested to extract RNA and protein. mRNA expression of antioxidant enzymes were determined by real-time RT-PCR, and protein expression of E-cadherin and N-cadherin were assayed by western blotting. [Results]In the human bronchial epithelial cell line BET-1, E-cadherin expression was down-regulated and N-cadherin expression was up-regulated by DEP exposure. HO-1, NQO-1, Nrf2 mRNA expression were up-regulated by DEP. The changes of expressions of E-cadherin and N-cadherin by DEP exposure were blocked by NAC pretreatment. [Conclusion]Our results suggest that DEP might be involved with induction of EMT process in human bronchial epithelial cells by oxidative stress.