

The Impact of Diesel Exhaust Particles on Tight Junction Proteins on Nose and Lung in a Mouse Model

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Abstract : Background: Diesel exhaust particles (DEPs) lead to trigger airway hyperresponsiveness (AHR) and airway dysfunction or inflammation in respiratory systems. Whether tight junction protein changes can contribute to development or exacerbations of airway diseases remain to be clarified. Objective: The aim of this study was to observe the effect of DEP on tight junction proteins in one airway both nose and lung in a mouse model. Methods: Mice were treated with saline (Sham) and exposed to 100 $\mu\text{g}/\text{m}^3$ DEPs 1 hour a day for 5 days a week for 4 weeks and 8 weeks in a closed-system chamber attached to a ultrasonic nebulizer. Airway hyperresponsiveness (AHR) was measured and bronchoalveolar lavage (BAL) fluid, nasal lavage (NAL) fluid, lung and nasal tissue was collected. The effects of DEP on tight junction proteins were estimated using western blot, immunohistochemical in lung and nasal tissue. Results: Airway hyperresponsiveness and number of inflammatory cells were higher in DEP exposure group than in control group, and were higher in 4 and 8 weeks model than in control group. The expression of tight junction proteins CLND4, -5, and -17 in both lung and nasal tissue were significantly increased in DEP exposure group than in the control group. Conclusion: These results suggesting that CLDN4, -5 and -17 may be involved in the airway both nose and lung, suggesting that air pollutants cause to disruption of epithelial and endothelial cell barriers. Acknowledgment: This research was supported by Korea Ministry of Environment (MOE) as 'The Environmental Health Action Program' (2016001360009) and Soonchunhyang University Research Fund.

Keywords : diesel exhaust particles, air pollutant, tight junction, Claudin, Airway inflammation

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