In utero and early life exposure to diesel exhaust-derived secondary organic aerosol on neurobehavior, neurological and immunological biomarkers in male rats

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In utero and early life exposure to diesel exhaust-derived secondary organic aerosol on neurobehavior, neurological and immunological biomarkers in male rats

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[Background and Aim]
Epidemiological and experimental studies have indicated that associations exist between exposure to air pollutants during the brain developmental period and occurrence of neurodevelopmental and neurodegenerative disorders. In the present study, we aimed to examine the effects of exposure to diesel exhaust (DE)-derived secondary organic aerosol (DE-SOA) during gestational and lactational periods on neurobehaviors and gene expression of neurological and immunological biomarkers in male rats.
[Methods]
Sprague-Dawley pregnant rats were exposed to clean air (control), DE (101 mg/m3) and DE-SOA (118 mg/m3) in the exposure chamber from gestational day 14 to postnatal day 21 with their pups. At the age of 10-13 weeks, neurobehaviors were examined by three-chambered social behavior test, social dominance tube test and marble burying test. After completion of behavioral tests, the prefrontal cortex from each rat was collected to investigate neurological markers such as brain-derived neurotrophic factor (BDNF) and immunological markers such as mast cells and microglia, proinflammatory cytokines (interleukin (IL) 1・), cyclooxygenase (COX) 2, oxidative stress marker heme oxygenase 1 (HO-1) and apoptosis markers (Bax and Bcl2) using immunohistochemical analysis and real-time RT-PCR method.
[Results]
Poor sociability and social novelty preference, socially dominant behavior, and increased repetitive behavior were observed in DE-SOA-exposed male rats. Messenger RNA expression level of BDNF was decreased whereas IL-β, COX2 and HO-1 were increased in the prefrontal cortex of male rats exposed to DE-SOA. The expression of mast cells and microglia marker ionized calcium-binding adapter molecule (Iba)1 were increased in the prefrontal cortex of male rats exposed to DE-SOA. In addition, mRNA expression level of Bax/Bcl2 ratio was significantly increased in male rats exposed to DE-SOA.
[Discussion and Conclusion]
We suggest that developmental exposure to DE-SOA may cause neuroinflammation and apoptosis in the immature rat brain and these effects may persist in later life. Our results indicate that in utero and early life exposure to DE-SOA may induce neurobehavioral abnormalities via activation of brain immune cells and apoptosis in the prefrontal cortex of male rats.