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Diesel engine exhaust origin secondary organic aerosol affects olfactory-based spatial learning performance and related gene expressions in preweaning mice

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【Background and Aim】

Exposure to environmental pollutants containing nano-sized particles during the developmental period might represent a major risk factor for children or the next generation's health. Early diagnosis is necessary for proper treatment to prevent disability in later life of the children. A variety of species have the ability to learn from the surrounding cues and use such spatial memory to navigate using landmarks from any position to a specific location. Many tests are well established to examine learning and memory functions in adults, however, there are very limited tests to examine learning and memory functions in neonates. The aims of our present study were to establish a novel olfactory-based spatial learning test and to examine the effects of exposure to nano-sized diesel exhaust-origin secondary organic aerosol (SOA), a model environmental pollutant, on the learning performance in preweaning mice.

【Methods】

Pregnant BALB/c mice [Japan SLC Co. (Tokyo, Japan)] were exposed to clean air, diesel exhaust (DE) or DE-origin SOA (DE-SOA) from gestational day 14 to postnatal day (PND) 10 in exposure chambers. Food (a commercial CE-2 diet, CLEA Japan, Inc., Tokyo, Japan) and water were given *ad libitum*. On PND11, the preweaning mice were examined by the olfactory-based spatial learning test. After completion of the spatial learning test, the hippocampus from each mouse was removed and examined for the expressions of neurological and immunological markers using real-time RT-PCR.

【Results】

In the test phase of the study, the mice exposed to DE or DE-SOA took a longer time to reach the target as compared to the control mice. The expression levels of neurological markers such as the N-methyl-D aspartate (NMDA) receptor subunits NR1 and NR2B, and of immunological markers such as TNF-α, COX2 and Iba1 were significantly increased in the hippocampi of the DE-SOA-exposed preweaning mice as compared to the control mice.

【Conclusion】

Our results indicate that developmental exposure to DE-SOA may affect olfactory-based spatial learning behavior in preweaning mice by modulating NMDA receptor and signaling pathway gene CaMKII and the inflammatory markers in the hippocampus. We suggest that, although the potential toxic substances contained in DE-SOA have not yet been identified, they may reach the brain via olfactory nerve route or systemic circulation and induce spatial learning deficit.