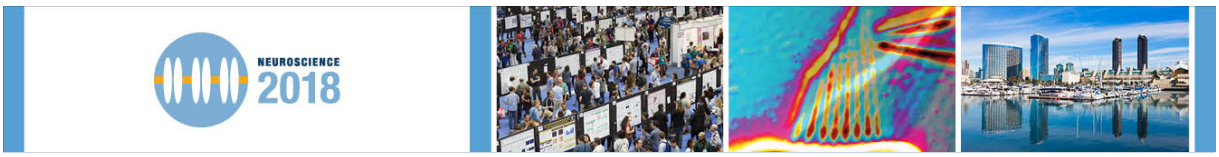

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474.13 / S18 - Glutamatergic mechanisms in depressive responses to prenatal exposure to traffic-related air pollution

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Tue, Nov. 6, 2018, 8:00 AM - 9:00 AM

Grant Support
AG040683

Grant Support
AG051521

Grant Support
ES023780

Authors
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Disclosures
R.G. Johnson: None. **A. Haghani:** None. **V. Coussa:** None. **F. Shirmohammadi:** None. **C. Sioutas:** None. **C.E. Finch:** None. **T.E. Morgan:** None.

Abstract
Prenatal exposure to nano-sized particulate matter (nPM), a subset of traffic-related air pollution particulate matter$_{0.2}$ (TRAP-PM$_{0.2}$), causes depressive-like symptoms in rodents (Davis et al 2013; Woodward et al 2017). Based on the collection methods, the nPM used in those studies were devoid of water-insoluble organic materials, such as polyaromatic hydrocarbons, which are known to cause deleterious neurodevelopmental and behavioral effects in children. Here, we used a novel method of collecting TRAP-PM$_{0.2}$ in a slurry (sPM) which retains the water-insoluble organics for mouse exposure. Pregnant mice were exposed to sPM at 340 ug/m^3 or filtered air for five hours a day for three days a week from conception to birth. At sixteen weeks of age mice underwent the forced swim behavioral test to screen for depressive-like symptoms. Male mice exposed to sPM showed a 22% increase in total time immobile and a 16% decrease in latency to first time immobile, compared to filtered air exposed males. Supporting the nPM study (Davis et al 2013), exposed female mice did not show depressive-like symptoms. The forced swim test was repeated 8 days later, after the mice were injected (i.p.) with the NMDA antagonist, MK-801, 0.06mg/kg, or saline. MK-801 reduced the total time immobile of sPM exposed mice back to control levels. We are further exploring the role of glutamatergic pathways by measuring glutamatergic receptor mRNA and protein levels. These results suggest that the glutamatergic pathway may play a critical role in the mechanism by which prenatal exposure to TRAP-PM$_{0.2}$ causes neurodevelopmental and behavioral effects.

Abstract Citation