3AD.5

Chronic Exposure to Real-time Traffic Related Air Pollution Increases Neuroinflammation and Exacerbates Plaque Burden in TgF344-AD Rats. Kelley Patten, Anthony Valenzuela, Ameer Taha, Keith Bein, ANTHONY S. WEXLER, Pamela Lein, *University of California, Davis*

Introduction. Epidemiological studies have linked traffic-related air pollution (TRAP) to increased risk of Alzheimer's disease (AD). However, this association has yet to be confirmed in a preclinical model. Moreover, the mechanism(s) by which TRAP influences AD are unclear.

Methods. To address these issues, we exposed male and female TgF344-AD rats and congenic controls to real-time TRAP or filtered air (FA) over the course of 15 months, using a mobile exposure facility that samples air from a highway tunnel in the Bay Area of California. Rats were exposed to TRAP or FA from postnatal day 28 to 15 months of age. At 3, 6, 10, and 15 months of age, brain samples were collected, and analyzed for plaque burden, bioactive lipids, microgliosis, astrogliosis, and cytokine protein levels.

Conclusions. Chronic TRAP exposure increased plaque burden in AD transgenic rats at 6 months. In addition, we found that TRAP exposure increased pro-inflammatory cytokines as early as 3 months of age, and modulated levels of both pro- and anti-inflammatory cytokines at later time points. Finally, both microgliosis and astrogliosis were increased by TRAP exposure. These data suggest that TRAP may exacerbate AD-relevant phenotypes, and that these results may be mediated through neuroinflammation.

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3AD.6

Temporal Changes in the Per Unit Mass Toxicity of Ambient PM2.5 in New York State. PHILIP K. HOPKE, Daniel Croft, Wangjian Zhang, Shao Lin, Mauro Masiol, Stefania Squizzato, Sally Thurston, Edwin van Wijngaaten, Mark Utell, David Q. Rich, *University of Rochester Medical Center*

From 2005 to 2016, there have been dramatic decreases in PM2.5 and the primary pollutant gases across New York State as a result of regulatory actions and changing economic conditions. The major PM constituents that declined include sulfate, nitrate, elemental carbon (EC), and primary organic carbon (POC). However, ozone and secondary organic aerosol (SOA) have increased in many locations. Source apportionment allows identification of the trends in source-specific PM. Secondary inorganic aerosol types have decreased, but spark-ignition vehicular contributions have increased. Rates of cardiopulmonary and respiratory infectious hospitalizations and emergency department (ED) visits in Buffalo, Rochester, Albany, and New York City over this period have also declined. However, an examination of these associations in 3 time intervals, 2005-07, 2008-13, and 2014-16, showed that for some health outcomes, the excess rates of hospitalization and/or ED visits per unit mass of PM2.5 have increased. When assessing associations between these hospitalization and ED visits and source-specific PM2.5, the apparent change in PM2.5 toxicity is associated with the changing relative proportions of the various source contributions over these time intervals. These results will be presented, and their implications for future regulatory actions to further improve public health, will be discussed.