fected. However, NLRP3 expression was increased when CORT exposure was extended to 7 days of treatment. Past studies in our lab revealed that PB alone or with CORT pretreatment do not produce neuroinflammation, thus it is not surprising that we found no effect on any of these genes of interest following exposure to PB. Interestingly, expression of serum amyloid a (SAA1), an endogenous ligand of TLR2, was not increased by DFP or CPF, with or without prior CORT exposure. Our data indicate potential involvement of TLR2 signaling in the CORT-priming of the neuroinflammatory response to the AChEIs, DFP and CPF. However, the enhanced expression of inflammatory cytokines in the brain following exposure to CORT and these AChEIs is independent of NLRP3 expression, which appears to be a primary effect of prolonged CORT exposure. By further examining the role of TLR2 in an augmented neuroinflammatory response following the combined CORT and AChEI exposure, we can identify potential targets for the treatment of illness associated with these exposures, such as Gulf War Illness.

1213 Revealing Behavioral Learning Deficit Phenotypes Subsequent to *In Utero* Exposure to Benzo(a)pyrene

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To characterize behavioral deficits in pre-adolescent offspring exposed *in utero* to B(a)P, timed-pregnant Long Evans Hooded rats were treated daily with B(a)P (150, 300, 600 and 1200µg/kg BW) or peanut oil (vehicle) on E14,15,16, and 17. Following birth, but during the pre-weaning period, B(a)P metabolites were examined in plasma and whole brain or cerebral cortex from exposed and control offspring. Tissue concentrations of B(a)P metabolites were 1) dose-dependent and 2) followed a time-dependence for elimination with an approximate 60% reduction by PND5 in the 1200µg/kg BW experimental group. Spatial discrimination-reversal learning was utilized to evaluate potential behavioral neurotoxicity in P40-P60 offspring. Late-adolescent offspring exposed *in utero* to 600 and 1200 µg/kg BW were indistinguishable from their control counterparts in the ability to acquire an original discrimination and reach criterion. However, a dose-dependent effect of *in utero* B(a)P-exposure was evident upon a discrimination reversal as exposed offspring perseverated on the previously correct response. This newly characterized behavioral deficit phenotype for the 1st reversal was not apparent in either the 1) original discrimination or 2) subsequent reversal sessions relative to the respective control offspring. This is the first such documented report of a spatial discrimination reversal deficit after *in utero* exposure to B(a)P. Furthermore, the expression of activity related-cytoskeletal associated protein (Arc), an experience-dependent cortical protein marker known to be up-regulated in response to acquisition of a novel behavior, was greater in B(a)P exposed offspring included in the spatial discrimination cohort versus home cage controls. Collectively, these findings support the hypothesis that *in utero* exposure to B(a)P during critical windows of development representing peak periods of neurogenesis results in behavioral deficits in later life.

1214 Exploration of the Gulf War Illness Phenotype in a Mouse Model Challenged with LPS at Long Term Time Points

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Chronic exposure to the glucocorticoid, corticosterone (CORT), at high physiological stress levels, can prime the CNS to release proinflammatory cytokines following exposure to neurotoxic exposures and systemic inflammation. Such neuroinflammatory events are associated with sickness behavior. Gulf War Illness (GWI) is a multi-symptom disorder characterized by persistent headaches, chronic fatigue, muscle pain, memory loss, confusion, gastrointestinal problems, and rashes, features also characteristic of persistent sickness behavior. Recently we found that, of the multiple exposures soldiers faced in the 1991 Gulf War theatre, two produced exacerbated neuroinflammation in mice. By mimicking the stresses of war with high physiologic exposure to CORT (200 mg/L 0.6% EtOH drinking water) over 7 days followed by sarin gas exposure with surrogate acetylcholinesterase inhibitor, diisopropyl fluorophosphate (DFP; 4 mg/kg, i.p.), we found heightened neuroinflammatory responses without evidence of astrogliosis or neurodegeneration 6 to 72 hours after DFP exposure. While these observations recapitulated the early symptoms of GWI, the essential pathobiology of GWI is the persistence of heightened responses to external stimuli for the past 20+ years. Here we employed episodic exposure of CORT in the drinking water of C57Bl6/J male mice for up to 180 days (CORT drinking water for 4 or 7 days every other week) to emulate episodic stress incurred by ill veterans following their exposures to multiple agents in theater, including sarin, 2 decades ago. Systemic exposure to LPS, at sub- and neuroinflammatory doses, were used to challenge the GWI phenotype (0.5 or 2 mg/kg, s.c., respectively). Our results show that successive waves of CORT provide a priming of the neuroinflammatory response to LPS, and that DFP (single exposure on the last day of the first CORT treatment) can exacerbate this effect. Thus, confirming this mouse model to produce a similar phenotype as that seen in soldiers diagnosed with GWI. Interestingly, we found that while 4 days of CORT in the drinking water is sufficient to produce an exacerbated neuroinflammatory response to LPS, that successive waves of CORT in 7 day, but not 4 day, exposures, every other week for 90 days produces a synergistic priming of the CNS to greatly augment the neuroinflammatory response to LPS. Together, these data suggest that GWI is a chronic, stressor primed, neuroinflammatory condition.

1215 Comparison of Nerve Excitability, Nerve Conduction Velocity, and Behavioral Tests for Acrylamide-Induced Peripheral Neuropathy

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Nerve excitability (NE) testing is a sensitive method to test for peripheral neurotoxicity in humans, and may be more sensitive than compound nerve action potential (CNAP) or nerve conduction velocity (NCV). We used acrylamide to compare the NE and CNAP/NCV methods. Behavioral tests known to be sensitive to acrylamide-induced neurotoxicity were included. Male Long-Evans rats aged 10-11 weeks were given acrylamide in their drinking water at 0, 1.5, 2.5 or 3.0 mM for 6 weeks. These concentrations resulted in an average exposure of 0, 10.3, 14.7, and 14.4 mg/kg/day of acrylamide. NE testing occurred during the 5th week, and CNAP/NCV testing was during the 6th week of treatment. Only 5 animals in the 3.0 mM group were tested due to pronounced neuromuscular and respiratory toxicity, and were not included in statistical analysis. Behavioral testing occurred weekly during treatment, prior to electrophysiology testing, and included open-field evaluations and measures of forelimb/hindlimb grip strength and landing foot splay. Both 1.5 and 2.5 mM treatments altered measures of neuromuscular and sensory function, including increased splay, lower hindlimb grip strength and rearing, and impaired gait and righting reflex. These changes worsened over the 6 weeks of exposure. NE tests indicated decreased nerve excitability, possibly coupled with altered K+ channel activation in sciatic motor nerves and tail mixed nerves. The NE changes were observed at the 2.5 mM treatment concentration. Changes in tail motor nerve NE and CNAPs/NCV in tail mixed nerves were not significant. Results indicate that for acrylamide-induced peripheral neurotoxicity, NE testing detected changes in physiological function, which differed between types of nerves, and was more sensitive than traditional CNAP/NCV methods. Behavioral changes were consistent with previous studies. This is an abstract of a proposed presentation and does not necessarily reflect EPA policy.

1216 Exposure to Fine Particulate Matter Increases Glutamate Uptake in Bergmann Glia Cells

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Exposure to urban airborne particulate matter (PM) is associated with adverse health effects, and outcomes in the central nervous system have become a relevant public health issue. Recent studies have linked exposure to fine particulate matter to dysfunction of the glutamatergic neurotransmission, given the fact that its components can reach the brain and instigate damage locally or systemically. Glutamate, the main excitatory amino acid transmitter triggers a wide variety of signal transduction cascades that regulate protein synthesis at the transcriptional and translational levels. Activity dependent differential gene expression has been attributed to the activation of both membrane glutamate receptors and transporters. The bulk of glutamate uptake takes place in glia cells. Within the cerebellum, Bergmann glia cells (BGC) are responsible for most of the glutamate uptake activity through the Na+ dependent glutamate/aspartate transporter (GLAST/EAAT-1). Taking into consideration the functional role of Bergmann glia, in terms of the recycling of glutamate, the supply to neurons of lactate, and the prevention of neurotoxic insults, we decided to investigate if air pollution fine particulate matter (PM2.5) exposure affects glia cells that surround glutamater-

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Preface

This issue is devoted to the abstracts of the presentations for the Continuing Education courses and scientific sessions of the 55th Annual Meeting of the Society of Toxicology, held at the New Orleans Ernest N. Morial Convention Center, March 13–17, 2016.

An alphabetical Author Index, cross referencing the corresponding abstract number(s), begins on page 603.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 629.

The abstracts are reproduced as accepted by the Scientific Program Committee of the Society of Toxicology and appear in numerical sequence. Author names which are underlined in the author block indicate the author is a member of the Society of Toxicology. For example, J. Smith.

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