triceps. One week following implantation, each animal was fitted with a backpack carrying an external transceiver coil and a battery-powered data acquisition system, and the dogs were allowed to freely walk down a hallway. EMG recorded from each animal as it walked down a hallway had very low noise and, in conjunction with recorded video, clearly indicated swing/stance phases of gait.

Disclosures: **D. McDonnall:** A. Employment/Salary (full or part-time):; Ripple LLC. **C. Smith:** A. Employment/Salary (full or part-time):; Ripple LLC. **D. Merrill:** A. Employment/Salary (full or part-time):; Ripple LLC. **S. Guillory:** A. Employment/Salary (full or part-time):; Ripple LLC. **S. Hiatt:** A. Employment/Salary (full or part-time):; Ripple LLC.

Poster

523. Stress: Factors Affecting Sensitivity, Protection, and Recovery

Location: Hall A

Time: Tuesday, October 20, 2015, 8:00 AM - 12:00 PM

Program#/Poster#: 523.01/V36

Topic: E.05. Stress and the Brain

Support: CDMRP Grant W81XWH-09-2-0098

Intramural Funds from Centers for Disease Control

Title: Corticosterone primes the neuroinflammatory responses to Gulf War Illness associated exposures: Effects of irreversible vs. reversible acetylcholinesterase inhibitors

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Abstract: Following the 1991 Persian Gulf War, as many as 250,000 soldiers returned with symptoms of Gulf War Illness (GWI), a complex disorder with characteristics similar to neuroinflammation-driven “sickness behavior.” These troops were exposed to a variety of cholinesterase inhibitors during deployment, including exposures to irreversible organophosphate (OP) acetylcholinesterase inhibitors (AChEI), in the form of the insecticide [chlorpyrifos (CPO)], or to warfare nerve agent, sarin. The reversible AChEI, pyridostigmine bromide (PB), was self-administered by soldiers as prophylactic treatment for potential nerve agent exposures. Alongside exposure to these AChEIs in the Gulf War were physiological stressors (e.g., high temperatures, physical exercise, or physical threat). Here, we examined effects of exposure to 3 OPs: CPO, DFP (as sarin surrogate), and PB in mice. These AChEIs

were given alone and with pretreatment with the stress hormone corticosterone (CORT) to mimic high levels of physiological stress. We assessed effects of these treatments on brain regional neuroinflammation by qPCR and neuroinflammation-associated microglial/astroglial activation by levels of phosphorylation of STAT3(tyr705). Adult male C57BL/6J mice were exposed to CORT (400mg/L in 1.2% EtOH), or water for 4 days. On the 5th day, mice were exposed to a single i.p. dose of CPO (8.0mg/kg), DFP (4.0mg/kg), or PB (3.0mg/kg). qPCR at 6 hours post-exposure revealed that acute CPO and DFP treatment alone produced neuroinflammation in the cortex and hippocampus that was enhanced by CORT pretreatment. Furthermore, CORT + CPO or DFP exposure enhanced activation of STAT3 in both brain regions. In contrast, acute exposure to PB alone or with CORT pretreatment did not produce significant increases in neuroinflammation or STAT3 activation. Exposure to the irreversible (CPO, DFP) or reversible (PB) AChEI with or without CORT pretreatment did not result in astrogliosis (increases in GFAP) in either the cortex or hippocampus, findings suggestive of an initial lack of neurodegeneration. Overall, exposure to irreversible AChEIs, CPO and DFP, produce neuroinflammatory effects in the cortex and hippocampus that are augmented by CORT pretreatment, an effect not seen with exposure to the reversible AChEI, PB. These effects may be a result of selective CORT priming of the JAK2/STAT3 pathway for specific GWI-related compounds - an effect that does not induce early signs of neurodegeneration. Thus, this study reveals the potential for selective pathway activation by irreversible AChEIs, a stress-hormone primed neuroinflammation in the absence of neurodegeneration that results in symptoms of GWI.

Disclosures: **A.R. Locker:** None. **K.A. Kelly:** None. **L.T. Michalovicz:** None. **D.B. Miller:** None. **J.P. O'Callaghan:** None.

Poster

523. Stress: Factors Affecting Sensitivity, Protection, and Recovery

Location: Hall A

Time: Tuesday, October 20, 2015, 8:00 AM - 12:00 PM

Program#/Poster#: 523.02/V37

Topic: E.05. Stress and the Brain

Support: Center Grant: P60-AA011605

Title: Acute stress activates neurons and microglia across multiple brain regions: impact of adolescent intermittent ethanol treatment

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