grip strength in the high-dose group. At all doses, nerve excitability data showed an effect on tail mixed nerve action potentials, suggesting alterations to nerve fibers that were consistent with neuronal depolarization (altered Na^+ conduction) and possible K^+ channel activity. In the high-dose group, CNAP recordings showed an increase in amplitude and NCV, possibly due to a loss of small nerve fibers. A reduction in latency was seen in the SEP cerebellum recordings, suggesting faster conduction and/or less inhibitory input. These changes further support the possibility that small afferent nerve fibers were altered. DCB treatment was associated with the development of an additional peak (N1-P1) at the front of the somatosensory cortical response, changes which could be related to effects at the cortical level or possibly a splitting of the afferent volley. Overall, this data indicates that treatment with DCB over this dose range and duration influences peripheral nerve and somatosensory function. Future studies should include histopathology to assess nerve fiber degeneration. *This is an abstract of a proposed presentation and does not necessarily reflect US EPA policy*.

3057 Exploring the Effects of Developmental Exposure to Flame Retardant Mixture and Components on Adult Wistar Rat Behavior

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FireMaster 550 (FM550) is one of the most commonly used fire retardants (FR) on foam-based furniture and baby products. This commercial mixture includes 2 brominated compounds, 2-ethylhexyl-2,3,4,5-tetrabromobenzoate (EH-TBB) mixed with bis (2-ethylhexyl) tetrabromophthalate (BEH-TEBP), and two organophosphates, triphenyl phosphate (TPhP) and assorted isopropyl triphenylphosphate (ITP) isomers, in the ratio: 36% EH-TBB, 14% BEH-TEBP, 18% TPhP and 32% ITPs by weight. As an additive FR, components of FM550 are readily leaching from consumer goods with detectable levels found globally in indoor dust, indoor/outdoor air, aquatic biota and food leading to widespread human exposure. Due to the structural similarities with known neurotoxicants and endocrine-disrupting chemicals (EDCs), including polybrominated diphenyl ethers (PBDE) and organophosphate pesticides, this is of great concern. Previously our lab demonstrated sex specific behavioral outcomes following developmental exposure to FM550 with males displaying increased anxiety and females being hyperactive. We are now exploring the relative contributions of the FM550 components on vulnerable brain and placental endpoints. The present, ongoing, studies focus on adult behaviors. Female Wistar rats were exposed to either vehicle, 1000µg brominated mixture (EH-TBB and BEH-TEBP), 1000µg ITP mixture or 2000µg FM550 mixture once daily from gestation day 0 to postnatal day 21. To monitor growth, all female and male offspring weights are collected regularly. Beginning on PND 65, at least one male and one female per litter are being evaluated using an array of behavior tests to assess repetitive impulsive behaviors, activity levels, social behaviors, anxiety-like behavior and short and long-term memory. Preliminary analysis suggests increased running wheel activity in males and females exposed to FM550 or the ITP mixture. These outcomes are helping to elucidate which FM 550 components contribute to sex-specific effects of exposure.

3058 Addressing the Impact of Polychlorinated Biphenyl Environmental Mixtures on Ryanodine Receptor Activity

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Polychlorinated biphenyls (PCBs) are halogenated aromatic hydrocarbons with 209 congeners. They were commonly used in commercial and industrial products until their ban in 1979 due to the rising concerns over their adverse effect on humans and the environment. Despite the ban, PCB congeners are still found in the air, in environmental water, on sediment, and in organismal and human samples. Some PCB congeners can alter the activity of ryanodine receptors (RyR), a Ca^{2+} channel important for neuronal and muscle cell Ca^{2+} homeostasis. Most studies focus on the effect of individual PCBs on RyR activity, but PCBs are found in mixtures in the environment. The aim of this study is to address the additivity of environmental mixtures of PCBs on RyR activity using radio-ligand binding assays. Currently, the binding assay was validated in a crude protein preparation and has confirmed that the highly active PCB 95 causes a 700% increase in activity at the ryanodine receptor. The development of a sucrose gradient is underway to further purify the crude protein in order to isolate the target ryanodine receptor. Based on previous research, the developed hypothesize predicts that PCBs will act additively towards RyRs, providing more insight into the influence of environmental mixtures on RyR based cellular pathways and organismal physiology. *Acknowledgements: This research was supported by the National Institute of General Medical Sciences of the National Institutes of Health under Award Numbers; UL1GM118979; TL4GM118980; RL5GM118978.*

3059 Whole Genome Transcriptome Analysis in a Genetic Model of Gulf War Illness

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Gulf War illness (GWI) affected up to 30% of the nearly one million personnel sent to the Persian Gulf in 1991. The probable cause was exposure to organophosphorus compounds coupled with high circulating glucocorticoids as would be expected in a combat theater. Previously, we developed a mouse model consisting of 7 days of exposure to corticosterone in the drinking water followed by injection with diisopropylflurophosphate (DFP, surrogate for sarin) and assessment of pro-inflammatory cytokines in frontal cortex and hippocampus 6h after DFP treatment. In order to assess genetic-based individual differences in susceptibility to developing GWI, we applied the model to male and female C57BL/6J and DBA/2J mice. Consequently we observed wide-genetic differences in pro-inflammatory gene expression by qPCR in the prefrontal cortex. We then subjected the prefrontal cortex to genome-wide transcriptome response by RNA-seq, comparing the combined corticosterone-DFP treatment. Gene ontology analysis showed altered immune function and apoptosis as the top systems affected. We also verified a previous nomination of Spondin 1 and ILb1 as candidate genes underlying individual differences in susceptibility. *This research was supported in part by CDMRP grant W81XWH-17-1-047.*

3060 Assessing the Differential Sensitivities of a Microelectrode Array Assay and a Neurite Outgrowth Assay for Detecting CNS Liabilities Using a Set of Neurotoxic and Seizurogenic Compounds

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Domoic acid is a neurotoxin first associated with the poisoning of 107 individuals and three subsequent deaths on Prince Edward Island in 1987 after these individuals consumed mussels containing this toxin. Due to its neurologic adverse effects, including memory loss, the illness was called amnesic shellfish poisoning. The toxin has since been attributed to the diatom, *Nitzschia pungens* f. *multiseries*, which was ingested by the mussels during normal filter feeding. In our lab, we use domoic acid as a tool compound when testing neurons on a microelectrode array (MEA) platform (Axion Biosystem's Maestro). Domoic acid completely eliminates all spontaneous spike activity when treating neurons at concentrations > 1 µM. When tested in a neurite outgrowth assay, using a high content imager (ArrayScan VTi), domoic acid does not affect cell health or neurite outgrowth when tested up to 10 µM for 72 hours. We tested various neurotoxins and seizurogenic compounds on the MEA platform and in the neurite outgrowth assay to the compare results and assay sensitivities. The antipsychotic haloperidol demonstrated seizurogenic liabilities at 3.16 µM on the MEA but had an IC50 of > 30 µM in an HCS assay. $GABA_A$ antagonist, picrotoxin, has a seizurogenic MEA profile as low as 0.2 µM but does effect cell health and neurite outgrowth at concentrations up to 10 µM. Chlorpromazine, on the other hand, has a seizurogenic profile and effects cell health and neurite outgrowth at similar concentrations (3 µM). With the results from domoic acid, haloperidol, picrotoxin, chlorpromazine and 6 additional controls compounds, we have determined that the MEA platform is more sensitive to detecting electrophysiological CNS liabilities than the neurite outgrowth assay. Alternatively, compounds which affect neurons by non-electrophysiological cytotoxic effects can be detected more sensitively than the acute MEA assay. Therefore, an effective overall strategy would be to test compounds for safety in both assays.

SOT 2020

59th Annual Meeting & ToxExpo

Anaheim, California • March 15–19

The Toxicologist

Supplement to *Toxicological Sciences*

Toxicological Sciences

ISSN 1096-6080
Volume 174, Issue 1
March 2020

The Official Journal of the Society of Toxicology

OXFORD UNIVERSITY PRESS

SOT | Society of Toxicology

www.academic.oup.com/toxsci

Publication Date: February 21, 2020

Preface

This issue is devoted to the abstracts of the presentations for the Continuing Education courses and Scientific Sessions of the 59th Annual Meeting of the Society of Toxicology, held at the Anaheim Convention Center, Anaheim, California, March 15–19, 2020.

An alphabetical Author Index, cross-referencing the corresponding abstract number(s), begins on page 542.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 580.

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To cite a 2020 SOT Annual Meeting abstract, please format as follows: *The Toxicologist*, Supplement to *Toxicological Sciences*, 174 (1), abstract #__, 2020, Title, First Author.

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