of AFFF once daily via oral gavage for either 1 or 14 days. Plasma, urine, and liver were collected to monitor for PFAS accumulation and excretion. Clinical chemistry, hematology, thyroid hormones, liver histology and gene expression were used to characterize biological response. Among the five PFAS quantitated, 6,2-fluorotelemer sulfonate was generally the most abundant in all five AFFFs. Analytes had different patterns of accumulation and excretion and the quantity of analytes varied by AFFF. AFFF1 tended to have the highest accumulation of analytes. Decreases in serum triglycerides and total thyroxine and triiodothyronine were observed only in animals exposed to AFFF3. Microscopic lesions were not observed in the liver after exposure to any AFFF. Changes in hepatic gene expression of PFAS-associated receptor pathways were modest (~2-fold) but revealed differential expression across AFFF and timepoints. AFFF4 produced statistically significant, dose-dependent increases in expression of constitutive androstane receptor target genes on Day 1 but not on Day 14, suggesting adaptation. In conclusion, PFAS from 14 days of AFFF exposure accumulated in rat liver and plasma and had different accumulation and excretion profiles across AFFFs. While exposure to AFFF3 and AFFF4 produced more biological changes—many consistent with reported effects of PFAS, exposure to AFFF1 resulted in the highest accumulation of PFAS. These data are important for understanding relative biological activity of AFFFs and can guide regulation of AFFF use in the future.

2030 Firefighter Dermal Exposure Assessment with Silicone Samplers

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Epidemiology studies have demonstrated elevated cancer rates for structural firefighters and a high percentage of cardiovascular events during or following firefighting activity. Polycyclic aromatic hydrocarbons (PAHs) are generated during combustion and are recognized as carcinogens and are implicated in cardiovascular disease progression and events. In this intervention study, commercially available firefighter personal protective equipment (PPE) typical of the modern US fire service was compared to an intervention configuration of PPE with a one-piece liner to eliminate interfaces at the waist and neck. Mannequins (n=16) dressed in the PPE ensembles were placed in a Fireground Exposure Simulator for 12 min with a couch as a fuel to mimic chemical exposures during a residential fire. Silicone samplers were placed outside of the PPE in the chamber to measure air concentrations. Silicone samplers were also worn by mannequins under the PPE at the neck, chest, and wrist to passively sample organic chemicals that broke through the PPE during the burn scenarios. Mannequins wearing the two PPE configurations were paired by co-location in the chamber; four total burns were conducted, with two mannequin pairs in each. All silicone samplers were analyzed with gas chromatography, tandem mass spectrometry for 63 different parent and alkylated PAHs. The list of PAH analytes includes and exceeds the EPA's current list of 16 priority PAHs, which is the current standard for evaluating firefighter exposures. 51 of these analytes were detected in at least one sample in the study, 9 of which have not been previously reported in fireground exposure studies. Paired t-tests with a Benjamini-Hochberg correction were used to compare co-located mannequin samples at the neck, chest, and wrist, for sum concentrations of low and high molecular weight PAHs (2-3 or 4-7 rings respectively). There is moderate statistical evidence that low molecular weight sum concentrations at the neck (p=0.001) and chest (p=0.015) and high molecular weight sum concentrations at the chest (p=0.020) are higher under the standard PPE than the intervention PPE after the simulated burns. Furthermore, exposures at the neck were generally greatest, and exposures at the wrist were the lowest for both types of PPE. Based on this study, firefighter dermal protection could be improved with the implementation of a physical barrier at the interfaces of the PPE, such as the one-piece liner tested.

2031 Excretion of Polybrominated Diphenyl Ethers and AhR Activation in Firefighter Breastmilk

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Little is known about the extent and effects of chemical exposure in lactating firefighters. The aims of this study were to identify if exposures during firefighting could lead to an increased concentration of polybrominated diphenyl ethers (PBDEs) excreted in breastmilk and breastmilk aryl hydrocarbon receptor (AhR) activity. Firefighters and non-firefighter controls collected breastmilk samples prior to any firefighting responses (baseline). Firefighters also collected breastmilk samples at 2, 8, 24, 48, and 72 hours after responding to a structural fire. Breastmilk extracts were measured for nine PBDEs. Five PBDEs (Di-15, Tri-28, Tetra-47, Penta-99, and Hexa-153) were above the limit of detection in at least 90% of samples and were summed for analyses. *In vitro* bioassays were conducted to assess the toxicity of breastmilk extracts via an AhR mediated response. Baseline measurements of PBDEs and AhR were compared between firefighters and non-firefighters using t-tests. Separate linear mixed models were used to assess potential changes in the sum of 5 PBDEs and AhR response among firefighters over time. We also considered potential effect modification by role at fire (interior vs exterior). Baseline measurements did not differ between the 21 firefighter and 10 non-firefighter participants. We did not observe that the sum of 5 PBDEs or AhR response among firefighters changed significantly over time after fire exposure; we would have detected a difference between mean response at baseline and mean response at a post-fire time point if going to a fire caused an increase in mean AhR by a factor of 1.4 or the mean of the sum 5 PBDEs by a factor of 1.5. We similarly observed no significant changes over time when we considered potential effect modification by role at fire. Plots of individual firefighters over time for sum 5 PBDEs and AhR demonstrated variation between individuals but no consistent pattern over time. Results from our study demonstrated similar average concentrations of PBDEs excreted in breastmilk in firefighters and non-firefighter controls and no significant increase after exposure to a fire. Similarly, average AhR response did not differ in firefighters and non-firefighter controls and was not significantly affected after fire exposure.

2032 Evaluating Post-Fire Skin Decontamination as a Method for Reducing Firefighters' Exposures to Carcinogens and Mutagens

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Firefighters experience elevated risks of cancer. Firefighters' exposures during fire suppression to combustion emissions, including polycyclic aromatic hydrocarbons (PAHs), are a concern due to their carcinogenic and mutagenic properties. To reduce PAH exposures, post-exposure cleaning of firefighters' skin (i.e., dermal decontamination) is often conducted. However, dermal decontamination has been implemented without any scientific evidence of efficacy. To assess the ability of dermal decontamination to reduce firefighters' exposures to PAHs, we conducted an intervention study at a live fire training centre. After participating in a live fire training exercise, 88 firefighters were randomly assigned and equally divided to one of three intervention groups (i.e., one of two commercially available skin cleaning wipes or soap & water) or the control group (i.e., no dermal decontamination). We measured concentrations of PAHs in personal air during the fire and on firefighters' skin three times (pre- and post-fire, and after the intervention). We also measured pre- and post-fire concentrations of urinary PAH metabolites and mutagenicity. The mean of PAHs in air samples during the fire was 759 μg/m^3. Fire suppression resulted in significant elevation of total PAHs and high molecular weight PAHs on firefighters' skin (1.3- and 2.2-fold, respectively, $p<0.01$). Urinary PAH metabolites increased significant 1.7 - 2.2-fold (depending on the metabolite, $p<0.001$). Urinary mutagenicity did not differ significantly. Soap & water was the only intervention that removed a significant amount of total PAHs from the skin (0.72 ng/cm^2 pre-intervention vs. 0.38 ng/cm^2 post-intervention, $p<0.01$). However, fold changes in urinary PAH metabolites (i.e., pre- versus post-exposure levels) were not affected by dermal decontamination. These data suggest that despite on-site attempts to remove PAHs from firefighters' skin, the intervention does not reduce the internal dose of PAHs. Future work should investigate the effectiveness of post-fire decontamination for other compounds of concern found in combustion emissions. More research is needed to determine effective ways to reduce firefighters' exposures.

2033 Role of Lipid Metabolism in Particulate Matter–Induced Lung Inflammation and Injury

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Particulate matter (PM) is a criteria air pollutant shown to increase morbidity and mortality from chronic lung diseases. Fine PM ($PM_{2.5}$) induces lung injury and inflammation, in part through activation of alveolar macrophages, as well as the production of pro-inflammatory lipid mediators such as prostaglandins and leukotrienes. Recent studies have identified a novel class of lipid mediators, termed specialized pro-resolving mediators (SPMs) that resolve inflammation following injury. However, it is currently unknown if $PM_{2.5}$ alters SPM production. From this, we hypothesize that $PM_{2.5}$ induced lung inflammation/injury is due to an imbalance in pro-inflammatory lipid mediators and SPM production.To test our hypothesis, C57BL/6J male mice were exposed to either filtered air (FA) or concentrated $PM_{2.5}$ (mean daily $PM_{2.5}$ exposure = 78.0 ± 11.1 μg/m^3) 6 hours/day for 3 weeks. 24 hours post final exposure, mice were euthanized and bronchoalveolar lavage (BAL) fluid and lung tissue were collected to assess inflammation/injury and lipidomics. Air space macrophages were isolated from BAL for qPCR to determine the macrophage specific lipid metabolism response.$PM_{2.5}$ exposure induced lung injury/inflammation as demonstrated by an increased BAL neutrophilia and total BAL protein. Lung pathology following $PM_{2.5}$ exposure revealed an increase in epithelial hyperplasia and recruited immune cells. Lung tissue lipidomics indicated no differences in SPM production between $PM_{2.5}$ and FA, whereas pro-inflammatory lipid mediators 9,10-DiHOME and 12,13-DiHOME were increased following

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The Toxicologist

Supplement to *Toxicological Sciences*

SOT | Society of Toxicology

Toxicological Sciences
The Official Journal of the Society of Toxicology
www.academic.oup.com/toxsci

OXFORD UNIVERSITY PRESS

ISSN 1096-6080 Volume 186, Issue S1 March 2022

Publication Date: March 23, 2022