expressions of cleaved-caspase 3 and cleaved-caspase 9 at 4 hr were the highest ones. In conclusion, the autophagy was firstly induced, then the apoptosis was mediated via the intrinsic apoptotic pathway, and finally the necrosis was caused in BEAS-2B cells exposed to low concentrations of crotonaldehyde.

2790 Crude Oil Vapor Effects upon Airway Epithelial Ion Transport and Lung Function in the Rat

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Crude oil vapor (COV) is a mixture of hydrocarbon vapors and volatile organic compounds (VOCs). Workers in the oil and gas industry are potentially exposed to COV while conducting routine tasks such as manual sampling, gauging and filling crude oil storage tanks from oil tanker trucks. The effects of COV inhalation exposure on the pulmonary system are unknown. Previously, we found there were no significant changes in pulmonary function or airway epithelial ion transport after acute inhalation exposure to COV (300 ppm total VOCs, 6 h/d, 1 d). In the current study, the effects of a sub-chronic inhalation exposure of COV on lung function and epithelial ion transport were investigated. Rats in whole body chambers were exposed to 300 ppm total VOCs for 28 d. Experimental endpoints were measured at 18 h, 28 and 90 d post-exposure. Total VOCs, benzene, toluene, ethylbenzene, and xylene concentrations were monitored and regulated during exposures to maintain concentration constancy. Transepithelial potential difference (V_t), transepithelial resistance (R_t), and short-circuit current (I_{SC}) were measured in tracheas mounted in Ussing chambers and treated with the ion transport inhibitors amiloride (Na^+ channel blocker; apical), 5-nitro-2-(3-phenylpropylamino)benzoic acid (NPPB; Cl^- channel blocker; apical), and ouabain (Na^+,K^+-pump blocker; basolateral). Compared to air-breathing controls, the I_{SC} response to NPPB was increased significantly at 28 d post-exposure, indicating an increase in Cl^- transport in the airway epithelium. There were no changes in V_t or R_t at 18 h or 28 d post-exposure. Lung resistance (R_L), dynamic compliance (C_{dyn}) and reactivity to inhaled methacholine (MCh) were measured in anesthetized rats. COV significantly increased basal R_L compared to air-breathing controls at 90 d post-exposure. There was no effect of COV on basal C_{dyn} or reactivity to inhaled MCh at any time point. Our results indicate that sub-chronic exposure to COV changes airway ion transport and pulmonary function.

2791 Three Dimensional (3D) Printer Emission-Induced Cell Toxicity

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Three dimensional (3-D) printing with polymer thermoplastics is known to emit millions to billions of ultrafine particles (diameter <100 nm) and volatile organic compounds that are formed by thermal decomposition and/or vaporization and condensation of the filament. There is a paucity of data on potential toxicity of these emissions. As such, we generated emissions from a commercially available 3-D printer while operating with acrylonitrile butadiene styrene (ABS) or polycarbonate (PC) filaments in a 0.5 m^3 environmental chamber. Particles were collected into serum free cell culture media using a liquid impinger sampler and the size and number concentration determined using nanoparticle tracking analysis. The mean sizes of PC and ABS-emitted particles in cell culture media were 160 ± 57 nm and 181 ± 21 nm, respectively. To determine the cytotoxicity of 3-D printer emissions, human small airway epithelial cells (SAEC) were seeded in a 96-well plate at a density of 1.5×10^4 cells per well and treated for 24 h with 1 x 10^8 particles/ml from PC and ABS emissions, followed by the cellular viability assays. The results showed that the exposures of PC and ABS emissions to SAEC significantly decreased the cell viability by 67% and 31%, respectively. Furthermore, it was found that the exposure of the PC emission significantly induced more toxicity than that of the ABS emission. Our preliminary data indicate that the emissions generated by 3-D PC and ABS filaments induce a toxic response in SAEC, and the emission of the PC filaments is significantly more toxic than that of the ABS filaments. Further studies are in progress to evaluate the mechanisms of 3-D printer emission-induced cellular toxicity.

2792 Exposing the Possible Hazards of Plug-In Air Fresheners: Volatile Organic Compounds Emitted into Room Air

D. Vado, and S. Hsieh. *Trinity Washington University, Washington, DC.* Sponsor: E. Weaver

Have you ever purchased plug-in air fresheners to give your home or office a clean smell? If so, have you ever considered what exactly it is that you are inhaling and if it is hazardous? Plug-in air fresheners are household products that release scents into room air. To determine the chemicals emitted by plug-ins, air samples were analyzed with particular attention to possible hazardous air pollutants (HAPs), carcinogens, and chemicals associated with asthma or other adverse health effects. To determine the chemical composition of initial emissions from a new plug-in, the headspace above the heated liquid was analyzed by GCMS (gas chromatography mass spectroscopy). To generate a picture of how plug-ins affects room air over time, samples were also collected in a home setting over a one-week period using solid-phase microextraction (SPME) field samplers. Our headspace analysis was done on three well-known brands (Glade, Airwick, and Febreze) and one generic brand (Great Value). We tested scents that were similar to each other from all four brands. The samples included compounds with scent (D-limonene, benzaldehyde, linalool, 3-carene), but surprisingly the generic brand also contained alkane hydrocarbons, which are not particularly associated with desirable smell. Alkane hydrocarbons are associated with petroleum and gasoline. The brand used for the at home setting was Glade. Not all of the compounds in the Glade headspace were detected in room air by SPME/GCMS. Concentrations of the detected Glade compounds increased over a period of days after starting plug-in use and decreased after removing the plug-in from the home setting. Compounds that were most prevalent in the headspace show a clear trend of both increasing and decreasing in concentration in room air, while others showed no trend.

2793 Volatile Organics Emitted from Little Trees Car Air Fresheners

A. Pino-Delgado, and S. Hsieh. *Trinity Washington University, Washington, DC.* Sponsor: E. Weaver

Air quality in motor vehicles is a function of the outdoor environment and also the use of scented products inside the car cabin. Little Trees air fresheners are commonly-used devices designed to hang in from the rear view mirror. To determine the volatile compounds emitted by these products, three types were analyzed for their emissions by headspace and gas-chromatography mass spectroscopy (GCMS). Several compounds detected are known to react with components of air associated with vehicular emissions and smog. To determine how the compounds are emitted in an actual user environment, air samples were collected by solid-phase microextraction (SPME) from inside a vehicle before and during the use of a Little Trees air freshener. Analytes collected on the SPME fiber were analyzed by GCMS.

2794 Effect of Secondhand Smoke Exposure on the Pathogenesis of Muco-Obstructive Airway Disease

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Poor indoor air quality, particularly secondhand smoke (SHS), has been associated with the augmentation of pre-existing muco-obstructive lung conditions including cystic fibrosis and asthma. Effects of SHS in the initiation and progression of these diseases remain poorly understood. To address this knowledge gap, a murine model of muco-obstructive lung disease [*Scnn1b*-Tg+ (Tg+)] was exposed to either SHS (SHS-Tg+) or HEPA-filtered air (FA-Tg+) from postnatal day (PND) 3-21 and lung phenotypes were examined on PND22. Hallmarks of the Tg+ lung disease, including airway inflammation, marked mucus obstruction, and mucous cell metaplasia (MCM), were evident in FA-Tg+ group. As previously reported, spontaneous bacterial infections were cleared in these mice at PND 22. In contrast, SHS exposures altered all of these responses in SHS-Tg+ group. SHS-Tg+ had diminished phagocytic cell recruitment and suppressed IgA levels in the airspaces that were strongly associated with exaggerated bacterial burdens at PND 22. Further, the SHS-Tg+ mice exhibited reduced mucus obstruction and diminished histological as well as molecular characteristics of MCM. This phenomenon was associated with dramatically down-regulated expression of IL33 mRNA and protein. IL33 is a known stimulator of type 2 innate lymphoid cells (ILC2) that orchestrates adaptive Type 2 (Th2) responses. Our data confirms strong association between down-regulation of IL33 levels, impaired

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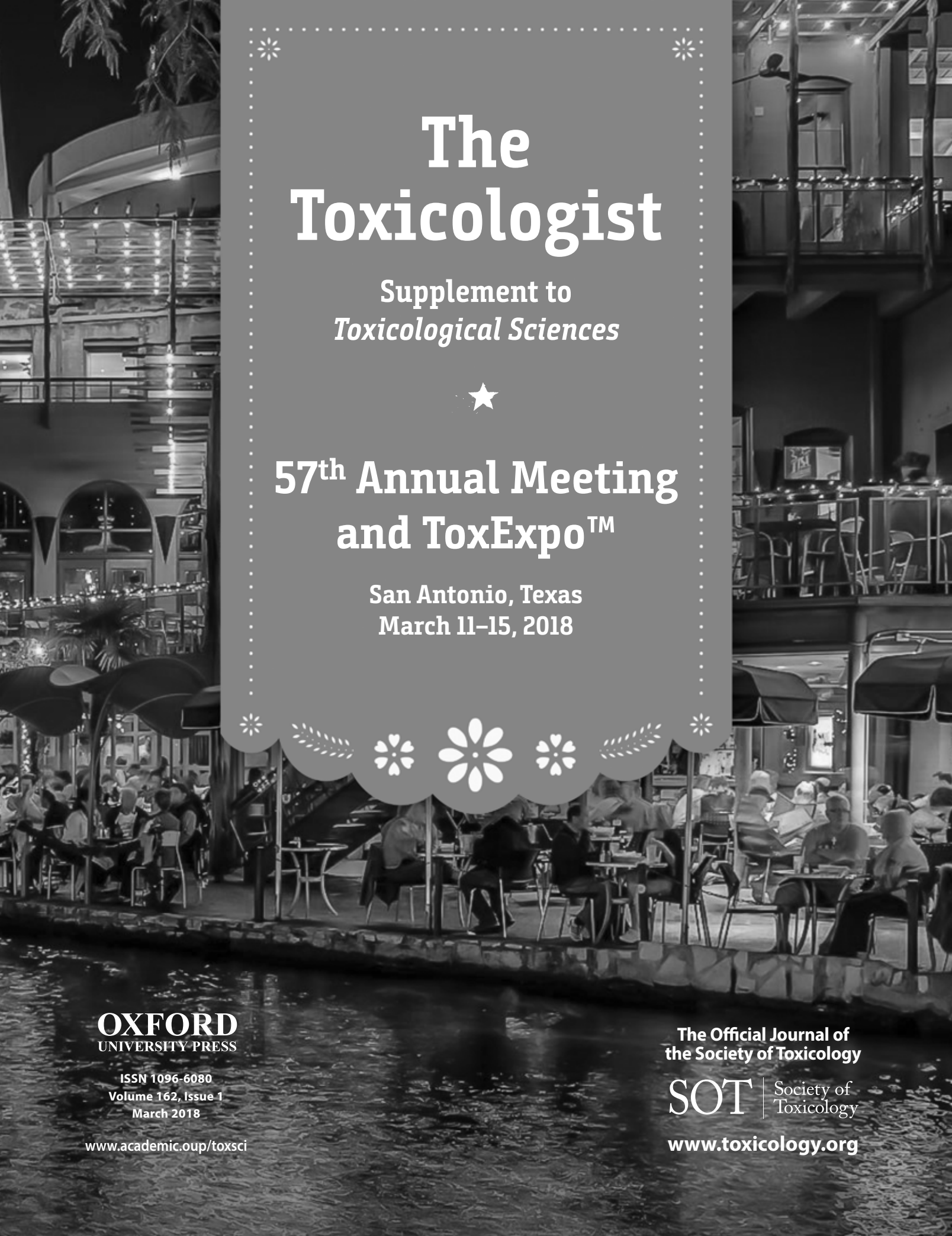

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