

printers include thermoplastics, metals, nanomaterials, polymers, and volatile and semi-volatile organic chemicals. The printing process may take several hours, and during this time period, the base materials are subjected to high heat (e.g., 220°C), resulting in a range of chemical by-products and particulates that may be released into indoor environments. Given these unknowns, scientists have begun to conduct studies to characterize and quantify these releases and their specific composition, particle size, and residence time in the indoor environment and produce data that can ultimately be incorporated into robust exposure assessments. *In vitro* and *in vivo* studies of the toxicity of the base materials and reactive by-products and particulates. This session will include leading researchers who are conducting critical investigations into the release of compounds of concern from 3D printers and their toxicity potential. This Workshop session will provide an overview of additive manufacturing/3D printers and some key factors associated with these devices that may impact release of and exposure to chemicals of concern. Included in this analysis is the impact of device- and feedstock-related factors that influence emissions, providing important insights on exposure potential, efficacy of control technologies, and design of experimental toxicology studies. The session will describe the impact of ABS thermoplastic emissions on the pulmonary system of rats and their implications for human health, in addition to the release of volatile organic compounds (VOCs) as well as particles and the need to mitigate exposures to these compounds.

### **1054 Inhalation Toxicity of Acrylonitrile Butadiene Styrene (ABS) 3D Printer Emissions in Rats**

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Fused filament fabrication 3D printing with acrylonitrile butadiene styrene (ABS) filament emits billions of particles and numerous volatile organic compounds (VOCs). This study sought to investigate the toxicity of ABS emissions from a 3D printer both *in vivo* and *in vitro*. For the *in vivo* studies, Sprague Dawley rats were exposed to real-time ABS printing emissions or air (control) for 4 h/day, 4 days/week for 1, 4, 8, 15, and 30 days. The average aerosolized particle concentration was  $0.24 \pm 0.09$  mg/m<sup>3</sup>, and the average median particle electric mobility diameter was 85 nm with an average geometric standard deviation of 1.6. Benzene was the predominant VOC released during printing. At 24 h after the last exposure, rats were assessed for pulmonary injury, inflammation, and oxidative stress as well as systemic and other organ toxicity. Results showed that among the measured cytokines in bronchoalveolar lavage, only IL-10 and IFN- $\gamma$  at day 1 and 4, and IL-13 at day 30, of the exposure were increased when compared with the air-control. Moreover, neither pulmonary oxidative stress responses nor histopathological changes of the lungs were found among the exposed rats. There were no significant differences in serum cytokines levels or hematological indices, except for an increase in platelets and monocytes at day 15. Several serum biomarkers involved in liver damage were significantly higher at day 1 of the exposure. For the *in vitro* study, both particles and VOCs were collected into serum-free cell culture medium using an impinger sampler inside a chamber while printing for 1.5 h, followed by characterization of the physicochemical properties, as well as assessment of cytotoxicity, oxidative stress, and cytokine production in human small airway epithelial cells (SAEC). Results showed that particle numbers and VOC concentrations varied between print runs. Based on mixed model regression analyses, at 24 h post-exposure, ABS emissions induced significant dose-dependent cytotoxicity, oxidative stress, and production of proinflammatory cytokines in SAEC. In conclusion, our *in vitro* studies indicated that the emissions from ABS 3D printing induced toxicological effects, which were not substantiated by the *in vivo* studies with the current low exposure concentrations. Thus, more *in vivo* studies with higher dose-response are needed to verify the *in vitro* findings.

### **1055 3D Printer Emission Inhalation Impairs Systemic Microvascular Function**

T. Nurkiewicz. *West Virginia University, Morgantown, WV.*

We have previously reported that inhalation exposure to a variety of xenobiotic particles attenuates arteriolar dilation in the periphery and initiates an inflammatory response characterized by increased leukocyte trafficking. Three-dimensional printing has become routine in industrial, occupational, and domestic environments. Significant amounts of respirable emissions (3DPE) result from this process. We hypothesized that because both particles and gases are generated by this process, that after 3DPE inhalation exposure, the resultant systemic microvascular impacts would be more robust. Sprague Dawley rats were exposed to acrylonitrile butadiene styrene (ABS) 3DPE via whole body inhalation. The exposure parameters were: [3DPE] = 250  $\mu$ g/m<sup>3</sup>; 4 hr/exposure; 1-30 days of exposure. The 3DPE mass median aerodynamic diameter was ~45 nm. This produced five calculated target 3DPE total lung burden groups: 2, 8, 16, 30, and 60  $\mu$ g. Sham-control rats were exposed to

filtered air only. Twenty-four hours after the last exposure, rats were anesthetized, and the mesentery was harvested for microvessel isolation. Second- and third-order arterioles were dissected and mounted on glass pipettes, perfused, and pressurized to assess endothelium-dependent (acetylcholine, ACh) and -independent (S-Nitroso-N-acetyl-DL-penicillamine, SNAP) vasoreactivity. Compared with sham-controls, 3DPE inhalation significantly attenuated arteriolar responsiveness to both ACh and SNAP. This was evident in a dose-response manner, and responsiveness was maximally inhibited at the 16  $\mu$ g/rat lung burden. This 3DPE burden is ~50% less than that for a comparable effect after nano-titanium dioxide inhalation exposures. These results collectively support the notion that 3DPE inhalation exposures cause systemic microvascular dysfunction.

### **1056 Release of Aerosols from Fuse-Deposition Modeling 3D Printers and Associated Human Exposure**

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Owing to a range of factors such as advancement in technology and increased affordability, 3D printers are gaining significant attention and popularization as a consumer product. Although there are many different techniques under the 3D printing definition, fuse-deposition modeling (FDM) consists of running a polymeric filament through a heated nozzle that moves to create the layers of a three-dimensional object. The high temperature of this nozzle causes gaseous and particulate air pollutants to be emitted in indoor environments, with compositions and concentrations that can vary significantly depending on the type of filament, nozzle temperature, printer type, and indoor environment characteristics, such as room size and ventilation rate. Another sparsely explored topic is the fundamental aerosol dynamics underlying particulate matter formation and growth during 3D printer operation, which is limited by instrumentation capability to measure the lower end of the particle size distribution. The objective of this work was to characterize aerosol emissions from the operation of a fuse-deposition modeling 3D printer. We characterized particle size distributions, optically absorbing particulate matter (PM), and total non-methane hydrocarbon emissions from a 3D printer in a chamber using a 1 nm SMPS, five wavelength aethalometer, and a flame-ionization detector, respectively. Effects of filament type on PM characteristics, emission rates, and emission factors were established. Our previous work showed that average aerosol emission rates ranged from ~108 to ~1,011 particles min<sup>-1</sup>, and rates varied over the course of a print job. Acrylonitrile-butadiene-styrene (ABS) filaments generated the largest number of aerosols, and wood-infused polylactic acid (PLA) filaments generated the smallest amount. Emission factors ranged from  $6 \times 10^8$  to  $6 \times 10^{11}$  per gram of printed part, depending on the type of filament used. Ongoing work includes the application of a numerical model for particle formation (via nucleation) and growth to the experimental data for the estimation of the physical parameters of the semi-volatile emissions from different types of filaments. Lastly, model results will be used to estimate PM levels and resultant exposure when the same 3D printer is used in built environments with varying physical and ventilation characteristics.

### **1057 Factors Influencing Emissions from 3D Printers**

A. Stefaniak. *NIOSH, Morgantown, WV.* Sponsor: Y. Qian

Additive manufacturing (AM), more commonly referred to as 3D printing, refers to several types of technologies that build physical objects layer by layer from a computer file. While some AM technologies remain limited to industrial settings, others, such as fused filament fabrication (FFF) and vat polymerization (VP), are relatively inexpensive and becoming more commonplace in homes, schools, libraries, and other nonindustrial settings. During operation, FFF and VP 3D printers emit particles and organic vapors. On a number basis, particles are dominated by ultrafine sizes (<100 nm) which can deposit in the lung alveoli. Organic vapors include compounds with known inhalation and dermal effects, including immune sensitization and toxicity. Both *in vitro* and *in vivo* data indicate that emissions may cause adverse health effects, including cytotoxicity and cardiovascular effects. Emissions are influenced by device-related (technology type, design, printing parameters) and feedstock-related (physical form, chemistry) factors. This presentation will summarize results from several years (and ongoing) of research on 3D printer emissions. Systematic investigation of device- and feedstock-related factors that influence emissions provides important insights on exposure potential, efficacy of control technologies, and design of experimental toxicology studies.



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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 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.

The abstracts are reproduced as accepted by the Scientific Program Committee of the Society of Toxicology and appear in numerical sequence. If a number is missing in the numerical sequence, the abstract assigned to the missing number was withdrawn by the author(s). Author names that are underlined in the author block indicate that the author is a member of the Society of Toxicology. For example, J. Smith. SOT members may sponsor abstracts that do not include an author with SOT membership. Authors who are members of designated organizations could serve as the sponsor of the abstract if an SOT member was not a co-author; these types of sponsorships are displayed with an organization name after the sponsor name (e.g., Sponsor: A. Smith, EUROTOX).

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