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 PM2.5(mean daily PM2.5 exposure =78.0 ±11.1 µg/m3) 6 hours/day for 3 or 6 weeks. 24 hours post final exposure, mice were euthanized and bronchoalveolar lavage (BAL) fluid and lung tissue were collected to assess lung inflammation/injury and lipid metabolism via lipidomics. Air space macrophages were isolated from BAL for qPCR to determine the macrophage specific lipid metabolism response.

PM2.5 exposure induced lung injury/inflammation as demonstrated by an increased BAL neutrophilia and total BAL protein following 3 weeks of exposure. However, this increase in lung injury/inflammation was not noted after 6 weeks of PM2.5 exposure. Lung tissue lipidomics indicated no differences in SPM production between PM2.5 and FA at 3-or 6-weeks post exposure, whereas pro-inflammatory lipid mediators 9,10-DiHOME and 12,13-DiHOME were increased following 3 weeks of PM2.5 exposure. Additionally, airspace macrophages had a significant increase in expression of the lipid metabolizing enzyme, ALOX5 following 3 weeks of PM2.5 exposure and was not seen post 6 weeks of exposure.

Taken together, subchronic PM2.5 exposure induces lung injury/inflammation and the pulmonary production of 9,10-and 12,13-DiHOME production. Additionally, preliminary data indicate a macrophage specific change in lipid metabolism following PM2.5 exposure, which will be pursued in future studies. All experiments were performed in accordance with the Animal Welfare Act and the U.S. Public Health Service Policy on Humane Care and Use of Laboratory Animals

Characterization of different aerosols using a novel thermal spray coating and inhalation exposure system

A Afshari, W McKinney, JL Cumpston, HD Leonard, JB Cumpston, M Jackson, S Friend, V Kodali, T Meighan, A Erdely, JM Antonini.
National Institute for Occupational Safety and Health, Morgantown, WV.

Thermal spray coating (TSC) involves spraying a metal coating product that is melted by extremely high temperatures and then applied under air pressure onto a surface. Large amounts of a complex metal aerosol comprised of Fe, Cr, Ni, and Zn are formed during the process, presenting a potentially serious risk to the operator. Information about the health effects associated with exposure to aerosols generated during TSC is lacking. Even less is known about the chemical and physical properties of these aerosols. The goal was to construct and characterize an automated TSC aerosol generator and animal inhalation exposure system that would simulate workplace exposures. An electric arc wire-TSC aerosol generator and exposure system was designed and separated into two areas: (A) an enclosed room with a closed spray booth where the spray coating occurred; (B) an exposure chamber with different particle characterization devices. The physicochemical properties of aerosols generated during electric arc wire-TSC using five different consumable wires were examined. The metal composition of each was determined by ICP-AES, including two stainless-steel wires [PMET720 (82% Fe, 13% Cr); PMET731(66% Fe, 26% Cr)], two Ni-based wires [PMET876 (55% Ni, 17% Cr); PMET885 (97% Ni)], and one Zn-based wire [PMET540 (99% Zn)]. The generated particles, regardless of composition, were poorly soluble, complex metal oxides and mostly arranged as chain-like agglomerates of primary particles in the ultrafine range (<100 nm). The MMAD as determined by a MOUDI was similar when comparing the generated particles from the different wires and ranged from 290-378 nm. To allow for continuous, sequential coating within the spray booth during a 2-hr testing period, a motor rotated the metal feedstock pipe to be coated in a circular and up-and-down direction. The spray gun was controlled by computer software and fired at programmed 1-sec intervals for up to a total of 12 sprays during the testing period. A targeted exposure chamber concentration of 25 mg/m3 was achieved and maintained during the 4-hr period, along with minimal fluctuations in relative humidity, temperature, and CO2 levels. This exposure system will generate continuous, controlled metal spray coating aerosols for future animal exposure studies.

In vivo Lung Toxicity associated with Boron Nitride Nanotubes with Different Purities

J.R. Roberts[1], X. Xin[1], K.A. Roach[1], M.B. Jakubinek[2], K.S. Kim[2], L. Bowers[1], M. Wolfarth[1], S.S. Leonard[1], D.W. Porter[1], S. Friend[1], A. Erdely[1], V. Kodali[1]
[1]NIOSH, Morgantown WV; [2]National Research Council Canada, Ottawa ON

Boron nitride nanotubes (BNNTs) are a high aspect ratio nanomaterial composed of hexagonal B-N in a multi-walled tube formation. Their material properties make them advantageous in applications in composites, energy storage, radiation and thermal shielding, as well as biomedical applications. The goal of this study was to assess the lung toxicity BNNTs with various purities in vivo. The National Research Council, Canada, provided three BNNT samples: a low purity as-produced sample with ~50% tubes (AP-BNNT), a purified sample with elemental boron removed (BR-BNNT), and a highly purified washed/filtered BR-BNNT sample (W2-BNNT). Hexagonal boron nitride (h-BN, <100 nm in diameter) was used as a control material for synthesis by-products. Samples were characterized for material properties including purity and size in powder form and in dispersion medium (DM, vehicle control). Purity (tube concentration) varied with W2> BR >AP with tube dimensions of geometric mean length of 1.68 µm ± geometric standard deviation (GSD) of 1.9 and a geometric mean diameter ± GSD of 0.017 µm ± 1.329 in powder form. Agglomerate sizes in DM ranged from ~285-350 nm for BNNT and h-BN. Male C57BL/6 mice were exposed by oropharyngeal aspiration to 4 or 40 µg of sample/mouse in DM or DM alone. Mice were euthanized at 4 h, 1 d, 7 d, 1 mo, and 3 mo post-exposure, lung lavage was performed to evaluate lung injury and inflammation on one group of animals. Lungs from a second group were collected for histopathology. Lavage parameters of lung injury and inflammation were significantly increased by the high dose of BNNTs at the early time points with W2>BR>AP and persisted in the W2 group up to 1 m post-exposure. h-BN produced low to no indicators of toxicity in lavage parameters measured. In lung tissue, inflammation and microgranuloma formation were noted with h-BN and BNNT exposures; however, severity was minimal to mild for all groups. Incidence and severity were greatest in the W2 group and began to resolve in all groups by 3 mo post-exposure. Recovery was fastest in the h-BN group. The results indicated that the tested BNNT samples induced acute toxicity and inflammation only at high concentration and the effects were more pronounced with increasing purity. The reference material used to represent one of the by-products of synthesis, h-BN, showed little toxicity relative to BNNT, suggesting effects where present may be due to BNNTs.

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