obstructive pulmonary disease (COPD), and long-term health impacts from COVID-19 than the national average. Poor air quality can exacerbate these conditions and lead to premature mortality. There are few studies on air quality in the American South, with even less in MS. Interest in fine particulate matter ($PM_{2.5}$), a complex mixture of air pollution, has grown due to associations with systemic health effects due to the size fraction. While size is one risk factor of $PM_{2.5}$ exposure, allowing for deep penetration into the lungs, composition is also health relevant. An underlying mechanism for these adverse health effects is the ability for $PM_{2.5}$ to induce oxidative stress. Federal regulations are in place to monitor $PM_{2.5}$ concentrations throughout the U.S. with routine filter-based sampling. However, after concentrations are determined, the filters are not commonly used for additional composition analysis; such as, measuring health relevant constituents including black carbon and polycyclic aromatic hydrocarbons (PAHs). **Methods:** $PM_{2.5}$ was collected onto filters by the Mississippi Department of Environmental Quality (MDEQ) at two locations, Hinds Community College (CC) and NCORE, just three miles apart in Jackson, MS. Sampling was performed every three days for the entirety of 2016 and $PM_{2.5}$ concentrations were recorded gravimetrically by the MDEQ. Black carbon concentrations were analyzed using a SootScan transmissometer at 800 nm. Filters were then extracted in a 1:1 ratio of DCM:MeOH with an aliquot of the extractant used for oxidative potential analysis via the Dithiothreitol (DTT) Assay. For chemical analysis, 16 parent PAHs were quantified in the remaining sample aliquots and controls. Statistical analysis was performed using SigmaPlot. **Results:** Initial analysis of summer samples (n = 40) revealed significant differences in $PM_{2.5}$ concentrations between the sampling locations ($p \leq 0.001$), with NCORE (18.50 µg /m^3 ± 8.09) trending higher than Hinds CC (8.73 µg /m^3 ± 3.14). Additionally, significant differences were observed between the sites in the summer for oxidative potential ($p \leq 0.001$), with NCORE (0.385 pmol/min/m^3 ± 0.162) again trending higher than Hinds CC (0.110 pmol/min/m^3 ± 0.095). However, there was not a significant difference between black carbon concentrations ($p = 0.182$) between NCORE (1.50 µg /m^3 ± 0.561) and Hinds CC (1.24 µg /m^3 ± 0.616), suggesting that the difference in oxidative potential between sites is impacted by factors other than black carbon. Analysis of additional seasons and PAHs is underway. We anticipate that there may be a significant difference in PAH composition between sampling locations as suggested by the difference in oxidative potential. **Conclusions:** For summer $PM_{2.5}$ in Jackson, MS, we observed significant differences between sampling sites in the same metro area for $PM_{2.5}$ concentration and oxidative potential, but not for black carbon concentrations. Additionally, we observed that if the summer $PM_{2.5}$ averages persisted throughout the year, both sites would exceed the recommended annual exposure suggested by the World Health Organization. We anticipate that there will continue to be variation between sampling sites due to source differentiation, which is a concern for community members living in the area. The addition of the remaining seasonal data and chemical characterization results will help profile air quality in the region. In conclusion, acknowledging that $PM_{2.5}$ exposure is a growing environmental justice concern in Jackson, MS is essential for determining health impacts and eventually mitigating health disparities and promoting community well-being.

PS 4041 Kinase (PKC/p38 MAPK) Regulation of TRPA1-Mediated Lung Epithelial Cell Injury and Repair

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Background and Purpose: Transient receptor potential (TRP) channels regulate cellular homeostasis. Wood/biomass smoke particulate matter (WBSPM) and cigarette smoke (CS) can cause acute and chronic respiratory damage and dysfunction. WBSPM is a potent transient receptor potential ankyrin-1 (TRPA1) and TRP vanilloid-3 (TRPV3) activator while CS activates TRPA1. Kinase signaling pathways also control cellular homeostasis, cell survival, and apoptosis. The hypothesis of this study was that the airway epithelial cell (AEC) injury and repair in the context of aberrant TRPA1 activation could be directionally modified by kinase inhibition. **Methods:** Human bronchial epithelial cell (HBEC3-KT) AECs were used. Proteomics/phosphoproteomics, qPCR, western blots, cell viability and live cell imaging assays, and selective kinase inhibitor treatments on AECs in the presence and absence of WBSPM or TRPA1 agonist (i.e., allyl isothiocyanate; AITC) treatment were performed. Biomarkers of oxidative stress, endoplasmic reticulum stress (ERS), and cytotoxicity were measured as well as alterations in TRP channel expression and function. **Results:** AECs treated with AITC exhibited robust morphological changes (rounding and detachment) within 2-4h, which reversed at times >6-8h. Proteomic analysis identified six significantly changed proteins at 2h, including loss of fibronectin and tubulin. At 6h, 36 proteins changed, including proteins responsible for structural/matrix regulation, the ERS response, and transcriptional/cell-cycle regulation. Phosphoproteome analyses revealed changes in 2672 and 1886 phosphosites on hundreds of proteins, and phosphosite-GSEA analysis identified EGFR, Akt, PKC, and p38 MAPK as differentially regulated by AITC treatment. A kinase inhibitor screen paired with cell viability assays revealed that EGFR, JNK 1/2, and Akt protected cells from damage while PKC, p38 MAPK, GSK3β, and ERK1/2 promoted toxicity. When temporal mRNA expression was examined, TRPA1 and TRPV3 (which counteracts TRPA1 driven ERS and cytotoxicity) exhibited a dynamic relationship. Western blots demonstrated increased phospho-EGFR and Akt, as well as decreased phospho-NF-kB, which regulates TRPA1 and TRPV3 expression. Calcium flux assays and qPCR analysis of AECs pre-treated with either PKC or p38 MAPK inhibitors demonstrated a decrease in TRPA1 expression and function as well as pre-emptive but muted TRPV3 expression. This correlated with a reduction in AEC damage triggered by treatment with either CS extract (89 µg/cm^2) or WSPM (20 µg/cm^2). **Conclusions:** PKC and p38 MAPK inhibition reduces TRPA1 expression while enhancing TRPV3. The combination of inhibitory effects protects AECs from damage triggered by toxic TRPA1 agonists and ERS. It may be possible to develop therapeutics targeting this network as a way to prevent damage by TRPA1-activating toxins.

PS 4042 Development of a controlled sawing apparatus for characterizing aerosols generated from cutting concrete composed of different formulations

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Background and Purpose: Concrete, a widely used and cost-effective construction material, often undergoes maintenance and alterations, including cutting, drilling, or grinding. These operations release airborne particles that have been observed to cause respiratory illnesses in construction workers. Additionally, concrete is a material constantly subject to examination for a range of applications adapting to changing environmental conditions and is continually refined through advancing technology. Recently, multi-walled carbon nanotubes (MWCNT) have been incorporated into concrete to enhance its compressive strength, abrasion resistance, reduced water and chloride penetration, and general increased durability. The aerosolization of MWCNT has been linked to lung inflammation, cancer, and other serious health risks potentially exacerbating respiratory hazards when added to concrete. This paper introduces a computer-controlled concrete aerosolization system designed for operation within a controlled laboratory environment to study the aerosols generated by cutting concrete that can contain additives such as engineered nanomaterials. The system can control saw travel speed, chamber air change rate, depth of cut, and time between cuts. This system represents the initial phase of a comprehensive study on the potential respiratory effects of aerosols generated during cutting using acellular, *in vitro*, and *in vivo* models. **Methods:** The current system integrates an industrial saw with a computer-controlled concrete feeding mechanism, enclosed within a mobile metallic chamber with a sealable door. The system is equipped with multiple ports facilitating the transfer of concrete particles to real-time particle measuring instruments and various offline sampling devices tailored to different applications. Standardized concrete specimens, with a 4” diameter and 8” length, commonly used in mechanical testing, were consistently employed in the experiments. The specimens to be cut were prepared with varying percentages of MWCNT: 0, low, and high wt %. **Results:** To maintain consistency, each concrete specimen was cut for approximately 54 seconds with multiple cuts averaged, ensuring a representative cross-section while collecting samples and preventing sampler and equipment overload. Results indicated a singular main mode with a peak particle size of approximately 0.72 µm as determined by using a Micro Orifice Uniform Deposit Impactor to measure particle sizes generated across all concrete specimens, regardless of the presence of MWCNT. No observable shift in particle size distribution occurred when comparing the aerosols generated from the sawing of all three concrete samples. Moreover, the mass median aerodynamic diameter fell within the respirable size range (< 6 µm) and remained similar for all specimens. Scanning and transmission electron microscopic analysis did not detect any free MWCNT in the collected aerosolized particles. **Conclusions:** A computer-controlled, laboratory-based concrete cutting aerosolization system was constructed that produces consistent respirable aerosols. It was determined that addition of MWCNT to concrete does not shift the particle size range or release free MWCNT by the methods of determination during cutting of the concrete. The developed system holds the potential to function as a standard concrete or other masonry material aerosolization system for studying various additive formulations in the future.

PS 4043 Role of RAGE Signaling in Cardiopulmonary Response to Sub-chronic $PM_{2.5}$ Exposure

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Background and Purpose: Exposure to fine particulate matter ($PM_{2.5}$) is hypothesized to induce both inflammation and oxidative stress, but a specific mechanism of action has yet to be discovered. One signaling pathway involved with chronic inflammation and oxidative stress is the receptor for advanced glycation end-products (RAGE). RAGE is a self-promoting receptor that is initiated from multiple ligands including (i.e. AGEs, amphoterin). $PM_{2.5}$ exposure has been found to increase RAGE activation in human bronchial epithelial cells, but has not yet been investigated in a sub-chronic *in vivo* exposure. This study explored the role of RAGE in cardiopulmonary responses following sub-chronic exposures to $PM_{2.5}$. **Methods:** C57BL6 wildtype and RAGE knockout (RKO) male mice (n=10/

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