

chemical components and phenotypic effects of allergic lung disease. We conclude that coarse PM downwind and fine PM upwind of traffic promote allergic inflammation in OVA-allergic mice. (This abstract does not represent U.S. EPA policy.)

PS 1206 Diurnal Variation in Toxicological Effects of Size-Segregated Particulate Samples Collected from High-Air Pollution Situation in China

P. I. Jalava¹, O. Uski¹, S. Kasurinen¹, K. Kuuspalo¹, J. Ruusunen¹, H. Liqing¹, O. Sippula¹, M. Hoppo¹, H. Koponen¹, J. Nurmelä¹, Q. Wang², C. Gu², J. Jokiniemi¹ and M. Hirvonen¹. ¹University of Eastern Finland, Kuopio, Finland and ²University of Nanjing, Nanjing, China. Sponsor: M. Viluksela.

Air pollution is a wide recognized problem all over the world. The air pollution levels in ambient air are known cause for premature deaths, many respiratory and cardiac diseases as well as cancer. We studied the in vitro toxicological responses associated with the observed adverse health effects. Thus far, the daily variations in air pollution levels and the consequent health effects have been studied to a lesser extent. We collected the particles for toxicological and chemical analyses from University of Nanjing, Xianlin campus. The generally high air pollution situation in China allowed us to separate between the day and nighttime samplings due to sufficient collected mass. With this approach we also got information on how the atmospheric processes and changes in the emission sources alter the toxicity of the collected particulate mass. The samples were collected with high volume cascade impactor in four different size ranges (PM10-2.5; PM2.5-1; PM1-0.2; PM0.2) and then extracted from the sampling substrates. Mouse macrophage cell line was employed in detection of the toxicological responses of the particulate samples including cytotoxicity, genotoxicity and inflammatory responses. The results showed that both chemistry and toxicological responses change between day and night. Nighttime samples had much more PAHs than the daytime samples. Cytotoxicity was rather similar in the day and night samples. However, inflammatory responses and genotoxicity were significantly higher during the daytime when compared to the night. It is possible that breakdown products of the PAHs are responsible for the higher responses during the daytime. When compared to the previous results from less polluted situations in Europe, the responses were quite different. In conclusion, the toxicity of the particulate mass in polluted situation during day and nighttime showed large variation.

PS 1207 Particulate Matter from Saudi Arabia Induces Genes Involved in Cholesterol and Lipid Metabolism

J. A. Brocato¹, M. Shamy², M. Costa¹, M. Khoder², A. Mansour², H. Sun¹, L. Chen¹ and T. Kluz¹. ¹Environmental Medicine, NYU School of Medicine, Tuxedo, NY and ²Faculty of Meteorology, Environment, and Arid Land Agriculture, King Abdulaziz University, Jeddah, Saudi Arabia.

Airborne particulate matter (PM) exposure is a major environmental health concern and is linked to metabolic disorders, such as cardiovascular diseases (CVD) and diabetes, which are on the rise in the Kingdom of Saudi Arabia. This study investigated changes in mouse lung gene expression caused by administration of PM10 collected from Jeddah, Saudi Arabia. FVB/N mice were exposed to 100 ug PM10 or water by aspiration and euthanized 24 h later. The bronchoalveolar lavage fluid (BALF) was collected and analyzed for neutrophil concentration and TNF- α and IL-6 levels. IL-6 and TNF- α were significantly higher in mice exposed to PM according to an ELISA assay and neutrophil concentration was also increased in exposed mice. RNA was extracted from the lungs and whole transcript was analyzed using Affymetrix Mouse Gene 1.0 ST Array. Mice exposed to PM10 displayed an increase in neutrophil concentration and elevated TNF- α and IL-6 levels. Gene expression analysis revealed that mice exposed to PM10 displayed 202 genes that were significantly up-regulated and 40 genes that were significantly down-regulated ($p<0.05$). PM10 induced genes involved in inflammation, cholesterol and lipid metabolism. This is the first study to demonstrate that Saudi Arabia PM10 increases in vivo expression of genes located in pathways associated with diseases involving metabolic syndrome.

PS 1208 Compounds Collected from Indium-Tin Oxide Production Induce Inflammatory Responses from Cultured Macrophages and Bronchial Epithelial Cells

M. A. Badding¹, D. Schwegler-Berry¹, K. J. Cummings² and S. S. Leonard¹.

¹Health Effects Laboratory Division, NIOSH, Morgantown, WV and ²Division of Respiratory Disease Studies, NIOSH, Morgantown, WV.

Indium-tin oxide (ITO) is used to make transparent conductive coatings for touch-screen and liquid crystal display electronics. Lung disease among workers in the ITO industry is an emerging occupational health concern as the demand for consumer electronics continues to increase. Epidemiologic studies have shown indium compound-exposed workers to have pulmonary alveolar proteinosis and fibrotic interstitial lung disease. However, the molecular mechanisms behind indium compounds' toxicity remain largely unknown. Thus, we aim to uncover how compounds encountered during ITO production affect cultured cells and ultimately, contribute to the pathogenesis of indium lung disease. We hypothesize that indium compounds (8 different samples collected from various stages at an ITO facility) cause lung pathology through direct cytotoxicity and/or via inflammatory signaling from exposed cells. Preliminary studies showed that exposure of RAW 264.7 monocyte macrophages and BEAS-2B bronchial epithelial cells to indium compounds resulted in significantly reduced viability. Microscopy techniques revealed that various indium compounds interact with and are engulfed by both cell lines within 1 to 3 hours, suggesting that cellular reactions may be occurring very rapidly. Indeed, nuclear factor kappa beta (NF κ B) activation occurs within 3 hours of treatment with compounds containing sintered ITO in both cell lines. Robust cytokine production (TNF α , IL-1 β , IL-6, and IL-8) following cellular exposures confirmed that pro-inflammatory responses are indeed occurring. Our results suggest that inflammatory responses to indium compounds by both pulmonary macrophages and epithelial cells may initiate and propagate indium lung disease. These findings have provided a better understanding of the molecular mechanisms behind an emerging occupational health issue and will aid in the discovery of biomarkers for disease prevention.

PS 1209 Differential Diagnosis of Airways Diseases Using the Ratio of FEV1/FVC in Subjects Occupationally Exposed to Powder Particles in a Pharmaceutical Industry in Nigeria

I. O. Omotosho^{1,2}, ¹Chemical Pathology, University of Ibadan, Ibadan, Nigeria and ²University of Ibadan, Ibadan, Nigeria.

Contribution of exposure to chemicals, fumes, dust and particles to the growing incidence of Chronic Obstructive Pulmonary Disease (COPD) has not been critically evaluated in this environment. This work was a preliminary report of the diagnostic differentials of COPD using pulmonary function indices determined in subjects occupationally exposed to chemical particles and dust in a pharmaceutical industry in Nigeria. 125 workers in the organization were screened for their pulmonary functions. Using Global Initiative for Obstructive Lung Disease (GOLD) as the mode of analysis and based on percentage FEV1/FVC ratio, 31.2% (39) had Normal Respiratory (NR) function; 24.8% (31) had Mild Restriction (MiR); 24% (30) had Moderate Restriction (MoR); 10.4% (13) had Moderately Severe Restriction (MSR); 5.6% (7) had Severe Restriction (SR); 2.4% (3) had Mild Obstruction (MO) while 0.8% (1) had Obstruction combined with Restriction (OR). Using the NR group as reference and towards stratifying the data, mean FEV1 and FVC obtained for the MiR, MoR, MSR, SR, MO and OR groups were compared with that of NR. The Mean values varied significantly in all the other groups ($P<0.005$) except with the MoR group ($P>0.005$). With this correction, 69 (55.2%) could be said to show normal pulmonary function while the remaining 53 (44.8%) had a form of respiratory abnormality or the other. The application of basic spirometry measurement and the need for enforcement of various industrial laws monitoring risk exposure towards reducing toxicity of chemicals by inhalation and the attendant airways diseases in our industries was further highlighted in this work.

PS 1210 Black Carbon Exposure Levels in New York City's Subway Stations

M. Vilcassim, G. D. Thurston and T. Gordon. Environmental Medicine, New York University, Tuxedo, NY.

The New York City subway is the main mode of transport for over 5 million passengers on average weekdays. Commuters are thus exposed frequently to the microenvironment in subway stations and airborne pollutants can have a significant impact on subway users if exposure levels are high. This study looked at black car-

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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 53rd Annual Meeting of the Society of Toxicology, held at the Phoenix Convention Center, March 23–27, 2014.

An alphabetical Author Index, cross referencing the corresponding abstract number(s), begins on [page 627](#).

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on [page 655](#).

The abstracts are reproduced as accepted by the Scientific Program Committee of the Society of Toxicology and appear in numerical sequence.

NOTE: Abstract numbers including a lower-case letter were programmed during second submission phase.

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