WP-36

Assessing Cancer Risk of Coal-fired Power Plant Workers Exposed to PAHs

F. Zeng[1], X. Xie[2]; [1]Renmin University of China, Beijing, China, [2]Institute of Acoustics Engineering Design and Research of Beijing Greentec, Beijing, China

Abstract: To study the relationship between the concentration of urinary 1-OH-Py,3-OH-BaP and the degree as well as the pathways of human exposure to PAHs, we collected 24-hour air, dietary and urine samples of 60 oven workers in a coal-fired power plant of Central China and 60 people in a general group not exposed to such PAHs in 2011. The concentrations of eight strong carcinogenic PAHs in air and dietary samples, and the concentrations of the urinary 1-OH-Py and 3-OH-BaP were determined. By studying the pathways of human exposure to PAHs, it is found that the main reason for coal-fired power plant workers' high cancer risk is the exposure to PAHs by breathing contaminated air, while the degree of exposure to PAHs through diet is similar to that of the general group. In order to reduce coal-fired power plant workers' cancer risk, occupational protection should be strengthened, and the working environment should be improved.

Keywords: A-biomarkers, A-exposure factors, A-risk assessment, C-food

WP-37

Assessment of the Contribution of Indoor Surface Residues to Children's Nicotine Exposure

E. Cooper, J. Shirai, A. Greenhall, J. C. Kissel; University of Washington, Seattle, WA

Abstract: The prevalence of smoking has declined significantly since the middle of the last century in the U.S. Most U.S. states have restricted the smoking of cigarettes in public places, which has had the effect of elevating the relative importance of residential exposure for non-smokers. Populations at risk for involuntary residential exposure to tobacco smoke include infants and children. While many smokers recognize the potential harm of secondhand smoke, and change their behavior to mitigate direct exposure to people around them, the extent to which tobacco smoke residues, termed thirdhand smoke (THS), persist in the indoor environment and contribute to exposure may be poorly understood. Review of available literature describing environmental nicotine levels and children's urinary excretion of cotinine suggests that direct inhalation or dust ingestion cannot fully explain apparent exposures. However, dermal exposure can plausibly explain the apparent shortfall. Chronic low-level dermal exposure to nicotine is inevitable in indoor environments contaminated by tobacco smoke. Nicotine provides an attractive case study for the investigation of the contribution of the dermal pathway to indoor non-dietary semi-volatile organic compounds (SVOC) exposure because 1) nicotine is well absorbed via skin, 2) nicotine is metabolized quickly and excreted in urine, and 3) nicotine is a common indoor contaminant for which relevant data are available. The work presented here applies a model that links a fugacity-based multi-compartment indoor environment component with a physiologically-based pharmacokinetic component to children's aggregate exposure to nicotine. The approach can be extended to SVOCs more generally. Other candidates for non-trivial contribution of dermal exposure to aggregate indoor exposure include common household materials such as pesticides, flame retardants and plasticizers.

Keywords: A-built environment, A-exposure models, A-indoor environment, B-SVOCs

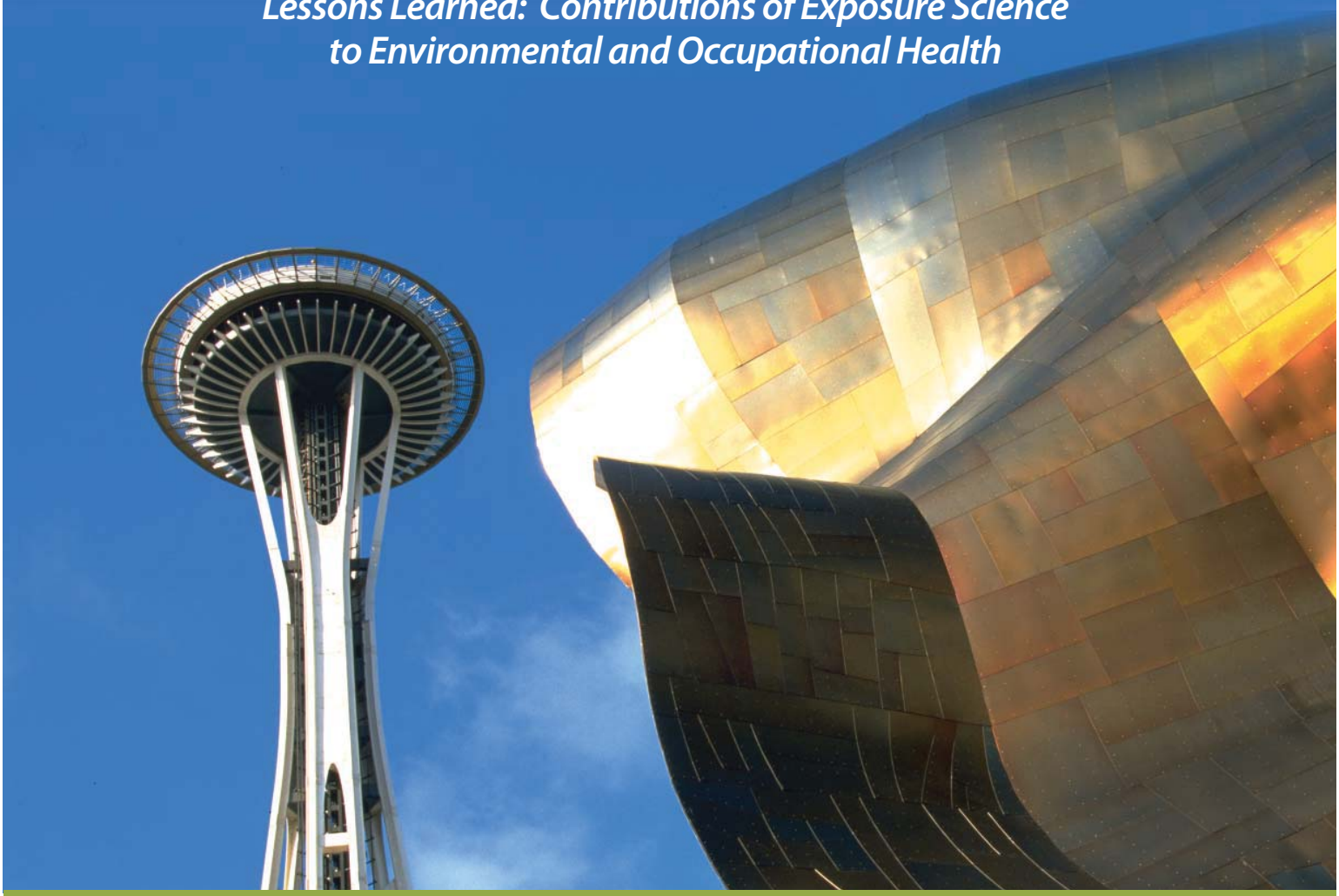
2012 ISES SEATTLE

Abstract Book

22ND ANNUAL MEETING OF

The International Society of Exposure Science

Lessons Learned: Contributions of Exposure Science to Environmental and Occupational Health



October 28 - November 1, 2012 — Seattle, Washington