found with end-of-shift samples among workers with elevated exposure levels closer to the end of the work shift. The occurrence frequency and level of higher occupational exposure levels might better explain the relation between occupational exposure and biomonitoring outcome. Further study on the extent of influence from exposure variability on biomonitoring outcome is warranted.

217.

STATISTICAL FRAMEWORK FOR BIOLOGICAL MONITORING.

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For the application of biomonitoring to occupational health practice, it is crucial that appropriate statistical methodology be used for the modeling and analysis of the relevant exposure data. This talk aims to develop a statistical framework for the biomonitoring scenario for situations where simple linear regression models can adequately model the relationship between a biomarker and the exposure level. For the problem of biomonitoring based on a simple linear regression model, we identify two classes of problems: the problem of prediction, and the problem of calibration or inverse regression. For example, for predicting urinary benzene levels from airborne benzene, we can treat the airborne benzene as the independent variable x and the urinary benzene as the dependent variable y. We now have a prediction problem, where it will be necessary to quantify the uncertainty in the prediction using a prediction interval. Also of interest is the computation of a tolerance interval or a simultaneous tolerance interval that is expected to contain most of the urinary benzene levels. As an example of the calibration problem, suppose we want to estimate the blood lead level (say, x) among exposed workers. When x is difficult, expensive, or time-consuming to measure, we can use a related variable y that is quicker and cheaper to measure, for example, saliva lead levels. Knowing the linear regression relationship between the two variables, we can estimate the blood lead level after measuring the corresponding saliva lead level. The uncertainty in the estimate can be quantified using appropriate confidence intervals. This talk will briefly discuss the relevant statistical methodology for addressing the above problems. The results will be illustrated using the analysis of exposure data.

218.

TOTAL PHENOL AND T,T-MUCONIC ACID AS BIOMARKERS FOR INDIVIDUAL EXPOSURE TO BENZENE: EVIDENCE OF HIGH BACKGROUND CONCENTRATIONS IN SOME PERSONS.

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The OSHA benzene standard states that if a worker is exposed to benzene in an emergency situation and his/her end-of-shift urine contains phenol levels greater than 75 mg/L adjusted for specific gravity, further medical evaluation is required. ACGIH has published BEIs for benzene exposure of 500 µg t,t-muconic acid (ttMA)/g creatinine adjusted and 25 µg s-phenylmercapturic acid/g creatinine. However, much of the published literature puts into question the usefulness of these tests for persons exposed to less than 5 ppm in an 8-hour workday. A voluntary study was conducted where a worker was exposed to a mixture of chemicals for four days. The 8-hour TWA benzene exposures ranged from 0.1 to 0.83 ppm (mean = 0.47 ppm). Twenty-nine urine samples were collected during the four days of the study. Approximately 28% of the urine samples contained levels of phenol greater than 75 mg/L and 31% of the samples had levels of ttMA greater than the 500 µg/g creatinine. Thirty background urine samples were also collected from the volunteer, and approximately 7% had levels of phenol above 75 mg/L and 30% had levels of ttMA above the BEI. Statistically, phenol corrected for specific gravity was significantly greater in the urine samples collected during the exposure period than in the urine collected during the background period ($p = 0.003$). However, the levels of uncorrected phenol, phenol corrected for creatinine, uncorrected ttMA, and ttMA corrected for creatinine were not statistically different from background concentrations for this volunteer ($p = 0.31$, 0.79, 0.21, 0.21, respectively). These results support other studies that have shown that spot urine samples are clearly not reliable indicators of benzene exposure when exposures are around 1 ppm. Multiple background samples are essential to determine whether or not high levels of excreted metabolites correspond to workplace exposure rather than simply inter-individual differences in metabolism of foods in the diet.

219.

AN INVESTIGATION OF DIOXIN EXPOSURE AMONG RUSSIAN FIREFIGHTERS. J. Grassman, Brooklyn College, Brooklyn, NY; Y. Chernyak, Insititute of Occupational Health and Human Ecology of Siberia and Institute of Biophysics of Angarsk State Technical Academy, Angarsk, Russian Federation; E. Brodsky, Severtsov Insitute of Ecology and Evolution, Moscow, Russian Federation; A. Shelepchikov, E. Mir-Kadyrova, D. Feshin, Severtsov Institute of Ecology and Evolution, Moscow, Russian Federation; A. Merinova, Institute of Occupational Health and Human Ecology of Siberia and Institute of Biophysics of Angarsk State Technical Academy, Angarsk, Russian Federation.

In December 1992, more than 700 firefighters from the region near Shelekhov, Russia, were exposed to combustion products during a catastrophic three day industrial fire. The fire suppression took place inside a warehouse that was sufficiently large to permit the entry of firefighters and their trucks. An estimated 22 to 57 g of dioxins (in I-TEQ_{DF}) were formed during the combustion of 1,000 tons of various raw materials, mainly polyvinylchloride and polyethylene. Respiratory protection was not available for the majority of firefighters. Moreover, many of the firefighters with access to closed-circuit respirators were hesitant to use them. To assess their body burdens of dioxins, we collected serum samples in January 2004. Assessment of dioxin body burdens this long after the exposure event is informative because several dioxin congeners have half lives of 10–20 years. 7 polychlorinated dibenzo-p-dioxin (PCDD), 10 polychlorinated dibenzofuran (PCDF), and 12 polychlorinated biphenyl (PCB) congeners were measured in 20 firefighters randomly selected from the cohort of 165 firefighters. Firefighters were divided into groups according to their symptoms and participation in the Shelekhov fire. Overall, the firefighters had a total mean arithmetic TEQ of 153 pg/g lipid, with a median of 123 pg/g. This compares to serum levels of 20–30 pg/g lipid reported in populations with only environmental exposures. Dioxins were not significantly related to disability status, respirator use, or days at fire. Officers tended to have lower serum concentrations of PCDDs than lower-ranking firefighters. Firefighters participating in the Shelekhov fire had higher serum concentrations of PCDDs than the controls. These preliminary results suggest that this is a population with a high degree of dioxin exposure, but it does not conclusively identify participation in the Shelekhov fire or firefighting in general as the source.

220.

CO-EXPOSURE OF ARSENITE AND BENZO(A)PYRENE: EFFECT OF GLUTATHIONE ON DNA ADDUCT LEVELS.

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Humans are considered the most sensitive species to arsenic exposure with increased risk to skin, lung, and bladder cancer. Epidemiologic studies of workers simultaneously exposed to benzo[a]pyrene (BaP) and arsenite (As) report additive to multiplicative effects. These studies are supported by both in vitro and in animal studies demonstrating an increase in BaP DNA adduct levels when co-treated with BaP and arsenite than when treated with BaP alone. Glutathione, the major thiol compound responsible for maintaining redox homeostasis, may provide cellular protection against arsenite's ability to increase the likelihood of DNA damage. We characterized the effect of modulating glutathione levels, through the use of buthionine sulfoximine (BSO) and glutathione ethyl ester (GSHEE) treatment as well as by using glutathione-deficient knockout mice, on the formation of DNA adduct levels after co-exposure to arsenite and benzo(a)pyrene. Lung and skin tissues were analyzed for DNA adducts using ^{32}P-postlabeling. Arsenic co-treatment increased average BaP adduct levels in both lung and skin; the increase was statistically significant in the lung ($p = 0.048$). A reduction in intracellular glutathione level increased BaP adduct levels, although only significantly in the skin of mice treated with BSO ($p = 0.028$). Treatment with GSHEE reduced adduct levels, although not significantly ($p > 0.05$) in any tissue measured.