

1697 RISK CHARACTERIZATION USING DIFFERENT ASSUMPTIONS REGARDING SPECIATED ARSENIC IN FISH AND SHELLFISH.

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When only total arsenic tissue concentrations are available, assumptions are required regarding concentrations of speciated arsenic in fish and shellfish. Using consumption data from the Pacific Northwest, this work demonstrates the effect of different assumptions using tissue data for total arsenic in fish and shellfish recently collected for freshwater and saltwater species in the same area. The results of a worldwide literature search provide the basis for initial assumptions. Additionally, assumptions based on a small amount of speciated arsenic data from the Pacific Northwest are included. Total arsenic concentrations are higher in saltwater fish than in freshwater fish. However, the percentage of inorganic arsenic (As_{inorg}) is higher in freshwater fish than in saltwater fish. Based on sparse information, it appears that lower trophic-level fish tend to have higher percentage As_{inorg} . Total arsenic concentrations tend to be higher in shellfish than in finfish, with a great deal of species variability. The percentage of As_{inorg} in shellfish is higher than in finfish, as is the percentage of another toxic arsenical species, dimethylarsinic acid. Uptake of arsenic in an arsenic-contaminated system appears to be a complex process, and no generalizable trends were observed in the data. In an evaluation that assesses risks from multiple contaminants in the same animal species, the impact of the percent As_{inorg} depends on the overall contaminant concentration present in the species. The importance of the amount of As_{inorg} in animal tissues increases as overall contaminant concentrations decrease. While having speciated arsenical data is preferable in all situations, it is most critical when total contaminant concentrations are suspected to be low. Use of speciated arsenic data facilitates risk management decisions by avoiding risk characterizations in which As_{inorg} , based on assumptions rather than measurements, predominates the risk.

1698 ESTIMATION OF INORGANIC ARSENIC INTAKE FROM FISH: MARKET BASKET VS. RECREATIONAL CATCHES.

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Seafood contributes the greatest fraction of total As to the diet; however, the majority of the As in fish is composed of complex organic arsenicals. The contribution of fish to dietary intake of the more toxic inorganic As (As_i) is pertinent both for assessing background As exposures and for assessing the contribution of As in recreationally caught fish to As exposures from surface water. Due to the expense and difficulty of analyzing As_i , concentrations in fish, most available fish data are for total As (As_{tot}) only. Dietary intake estimates for As in fish are frequently made by assuming that a certain fraction of the As_{tot} reported in the fish is As_i . For example, NRC (1999) assumed that 10% of seafood As is inorganic. Limited data has been available to support such assumptions, but several recent studies suggest that the NRC (1999) assumption may be too high. Schoof, et al. (1999) report data for saltwater finfish, canned tuna, shrimp and freshwater fish purchased at markets. From these data the fraction of As_i vs. As_{tot} may be estimated. Whereas 2-8% of freshwater fish As is inorganic, <0.1 to 0.6% of the marine food As is inorganic. The As_i in all of these samples was very low, so the variation in fractional inorganic content is almost solely due to variations in the amount of As_{tot} reported. Buchet, et al. (1994) reported somewhat higher As_i content in marine fish, ranging from 0.4 to 3%. Since most seafood intake in typical diets is from marine sources, these and other recent studies suggest that the NRC assumption is an overestimate, and that As_i is closer to 1% of As_{tot} in seafood. New data also allow estimates of fractional As_i in wild freshwater fish. EVS (2000) report As_{tot} and As_i for fish caught in the Willamette River in Oregon. No As was detected in pike minnow, while the fraction of As_{tot} that was As_i averaged 5%, 4% and 13% in bass, carp and sucker, respectively. The implications of these findings for risk assessment of As in fish in surface water and the derivation of ambient water quality criteria for As will be explored.

1699 DOSE RECONSTRUCTION OF BENZENE EXPOSURE FOR PLIOFILM COHORT (1936-1976) USING MONTE CARLO TECHNIQUES.

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Regulatory standards or guidelines for occupational exposure to benzene are based predominantly on studies of workers. The cohort of rubber hydrochloride (Pliofilm) workers, in particular, serves as the primary basis for the OSHA Permissible Exposure Limit (PEL) and ACGIH Threshold Limit Value (TLV). Previous assessments of this cohort by Rinsky (1981), Crump and Allen (1984), and Paustenbach et al. (1992) have relied on varying assumptions about workplace

practices and processes over time, as well as different exposure data and assumptions, thereby yielding significantly different estimates of benzene dose for certain job categories. Given the inherent limitations and uncertainties involved in estimating historic exposures for this cohort, a "distributional" rather than a "point estimate" approach should be more informative for this cohort. In this paper, we re-evaluate the Pliofilm data using Monte Carlo techniques and discuss the impacts of various exposure parameters on estimated doses. A key objective of this work is to address criticisms that previous exposure parameter values were either too high or too low. Estimated benzene doses are reported as equivalent 8-hour time-weighted averages for more than ten job categories from 1936 to 1965 (Akron I and II facilities) and 1940 to 1976 (St. Mary's facility). The key sources of uncertainty addressed in the analysis include characterization of benzene air concentrations (background and peak), uptake of benzene from dermal exposures, engineering controls over time, extended work hours and plant shut down in early years, accuracy of monitoring devices, and effectiveness of personal protective equipment. Sensitivity analyses are used to illustrate areas of uncertainty that have the greatest impact on estimated benzene dose. The results of this analysis provide a more thorough and defensible characterization of benzene exposures among the Pliofilm cohort than those previously reported. These results may also be used, in conjunction with the available mortality data, to provide a more refined assessment of benzene's chronic toxicity in humans.

1700 RATE OF HEXAVALENT CHROMIUM REDUCTION BY HUMAN GASTRIC FLUID.

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Hexavalent chromium [Cr(VI)] is reduced to the essentially non-toxic trivalent state [Cr(III)] in the human stomach. This study investigates the rate of Cr(VI) reduction under a variety of simulated gastric conditions, and in real human gastric juice collected from fasting volunteers. The rate of Cr(VI) reduction in simulated gastric fluid was measured at pH values from 1.5 to 4.5, at starting Cr(VI) concentrations ranging from 100 to 400 ppb, in diluted stomach fluid (10-fold), following ingestion of Rolaid[®], and in the presence of food. Stomach conditions were simulated by preparing an aqueous mixture of the primary components of gastric fluid (pepsin, mucine, gastric lipase, gastric amylase, etc.), acidifying with HCl, and providing continuous stirring and heating to approximately 37°C. All experiments were conducted for 60 minutes, with Cr(VI) measured using a Hach AccuVac ChromaVer3 instant test kit. The findings of these studies are: 1) real human stomach fluid has a 10-fold greater capacity to reduce Cr(VI) than simulated stomach fluid; 2) within the first 2 to 5 minutes, 0.3 to 1 mg Cr(VI)/L is reduced in real stomach fluid collected under fasting conditions; 3) increasing gastric pH from 1.5 to 4.5 reduces the rate and extent of Cr(VI) reduction by at least one-third; 4) the amount and rate of Cr(VI) reduced is constant regardless of the starting Cr(VI) concentration, which suggests that these results may be extrapolated to lower concentrations of Cr(VI) in drinking water; 5) antacids dramatically increase the pH of the stomach (from pH 1.5 to 8.2) but appear to have little effect on the Cr(VI) reduction rate or capacity as compared to reduction at pH 4.5; and 6) the presence of food substantially increases Cr(VI) reduction in simulated stomach fluid, with 10 mg/L reduced within the first 4 minutes. These results suggest that ingested Cr(VI), at the federal MCL of 100 ppb, would exist in the stomach, even under fasting conditions, for less than 1 minute before being reduced entirely to Cr(III).

1701 EXACERBATION OF UROPORPHYRIA DEVELOPMENT IN MICE HOMOZYGOUS FOR GENETIC DISRUPTION AT THE HEMOCHROMATOSIS (HFE) GENE.

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Two recently developed strains of genetically modified mice have made it possible to study the contribution of mutations at the hemochromatosis (Hfe) and uroporphyrinogen decarboxylase (URO-D) genes to porphyric susceptibility. Homozygosity for the null mutation of the URO-D gene (URO-D^{-/-}) was lethal. Homozygosity for the null mutation of the Hfe gene caused high levels of iron to accumulate in the liver; heterozygosity was without effect. Neither URO-D nor Hfe gene disruption alone was sufficient to cause the development of uroporphyrin. We have investigated polyhalogenated aromatic hydrocarbon-induced uroporphyrin in male and female mice bearing either or both these genetic variables. In mice heterozygous for a null mutation at the URO-D gene, mild uroporphyrin develops 21 days after a single dose of Aroclor 1254, a polychlorinated biphenyl mixture. The presence of high hepatic iron-load from a disruption of the hemochromatosis gene in addition exacerbates the porphyric response. The hepatic porphyrin