ured by real-time PCR array and release of IL-8 and IL-6 was measured by ELISA. The PCR array showed that Zn induced different cytokine pattern than Fe, with high mRNA levels of CCL11, CCL26, CXCL5 and CXCL14. Both metals had high expression of CXCL8 and CCL20. The metals Cd, As and Zn were the most potent to induce the release of CXCL8 and IL-6, followed by Mn, Ni and V, and the least potent metals were Cu and Fe. The metals showed also differential effects in induction of apoptosis as well as necrosis in the epithelial lung cells. In conclusion, metals show marked differences with regard to inflammatory and cytotoxic properties in BEAS-2B cells. This may indicate that metals play a role in the inflammatory processes induced by particulate matter, and that the metals' contribution vary depending on types of sources.

PS 2177 HEPATIC AND PULMONARY DIFFERENTIAL TOXICITY AND PATHOGENICITY OF HEVAVALENT CHROMIUM, NICKEL, AND CADMIUM.

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Both civilians and military personnel can be exposed to toxic chemicals and materials from occupational sources, environmental pollution, or as the result of military activity. The goal in this first stage of our study was to measure oxidative stress markers after different transition metal exposures. We performed a toxicological study to examine these effects using rats treated through I.P. injection. Sprague-Dawley rats were dosed with NiCl2 (0.25. 0.5, 0.75 mmol/kg BW), Na2Cr2O7 (5, 10, 20 mg/kg BW) and CdCl2 (0.5, 1.25, 2.5 mg/kg BW) and humanely sacrificed 1, 3 and 7 days post exposure. Liver tissue, kidney tissue, blood and lung lavage fluid were collected and analyzed. Liver tissue showed an increase in oxidative damage, through lipid peroxidation and hydrogen peroxide production, in all metal exposures with Cr being the most dramatic and Ni showing damage at days 3 and 7. Kidney tissue demonstrated immediate damage from Cr and then showed recovery, while Cd and Ni showed effects at day 7, at the highest concentration. Electron spin resonance results showed an increase in hydroxyl radical formation in liver and kidney tissue from Cr, day 1, as well as Cd & Ni on days 3 and 7, at the highest exposure levels. Bronchiopulmonary lavage was also performed on the rats to yield macrophages and cell for differential measurements. From this a small rise in Cr exposed animals indicated a cross talk from the I.P. exposure. To summarize, Cr-induced oxidative damage at day 1 was reduced or resolved at day 7, while Cd and Ni produced more oxidative damage at days 3 and 7 at the higher exposure levels. This data will be combined with the second stage of our study which involved the analysis of blood biomarkers and gene transcripts to develop a method of identifying early biomarkers of transition metal exposure.

PS 2178 ACUTE SYSTEMIC INFLAMMATION TO WELDING FUME: COMPARISON OF VARIOUS TYPES.

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Adverse cardiovascular effects have been shown following particulate matter pulmonary exposure. In this study we evaluated acute systemic inflammation following aspiration exposure to three different types of welding fume (manual metal arc-stainless steel [MMA-SS]; gas metal arc-SS [GMA-SS]; GMA-mild steel [GMA-MS]). Fumes generated from SS electrodes are approximately 40-50% iron, 20-30% chromium with ~3-5% nickel, whereas MS fumes are typically >80% iron, with some manganese, and no chromium or nickel. Mice were exposed to 340ug of welding fume suspended in PBS. Lavage parameters from the right lung lobes, with collection of heart, aorta, left lung lobe, serum and whole blood were analyzed for various inflammatory parameters including serum protein profiling (59 in total), inflammatory gene expression (TaqMan array) and lung cytotoxicity. At 4hr post exposure the MMA-SS fume was the most cytotoxic to the lung in terms of lavage albumin and LDH. Inflammatory gene expression was equally increased for all fumes with some exceptions including IL-5 and IL-13 which were greater for the MMA-SS fume and the CXCL chemokines which were greater for the GMA fumes. IL-5 was the only increased serum protein. In the aorta and heart stress response genes (e.g. MT-1, MT-2 and Hif-3A) were elevated in the MMA-SS fume only. The primary soluble component of MMA-SS is chromium and when mice were exposed to chromium alone similar gene changes were seen in the aorta but to a lesser extent. At 24hr post exposure we found continued pulmonary lung inflammation mostly greater compared to 4hr. At this time point inflammatory gene expression was greatest in the GMA-SS fume. At 24hr, lung cytotoxicity was still greatest in the MMA-SS and the expression of the stress genes in the aorta were equally elevated compared to 4hr. In conclusion we found the MMA-SS fume induced more pulmonary cytotoxicity as compared to the GMA fumes, and this was reflected in a cardiovascular response. The soluble chromium in the MMA-SS fume may play a role in these effects.

PS 2179 BREVETOXIN INHALATION ALTERS THE PULMONARY RESPONSE TO INFLUENZA A IN THE F344 RAT.

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Results of epidemiological studies indicate that emergency room visits for respiratory indications increase during periods of Florida Red Tides. The purpose of this study was to examine whether repeated daily brevetoxin inhalation, as may occur during a Red Tide, alters viral clearance, and pulmonary responses to influenza A. Male F344 rats were divided into four groups: 1) sham aerosol/ no influenza; 2) sham aerosol/ influenza; 3) brevetoxin/no influenza; and 4) brevetoxin/ influenza). Animals were exposed by nose-only inhalation to vehicle or 50 µg brevetoxin 3/m3, 2 hr/day for 12 days. On the sixth day of aerosol exposure, Groups 2 and 4 were administered 10,000 plaque forming units of non-adapted influenza A, strain HKX-31 (H3N2). Subgroups were euthanized 2, 4, and 7 days post influenza treatment. Left lungs were taken for histopathologic evaluation and right lungs evaluated for viral load and cytokine content. Influenza virus was cleared from the lungs over the 7 day period, however, there was significantly more virus (1.4 times) remaining in the Group 4 lungs compared to Group 2 lungs. At 7 days following influenza instillation, the severity scores for perivascular and peribronchiolar infiltrates were higher in Group 4 compared to Group 2, however, the severity score for alveolar macrophage hyperplasia in Group 4 was approximately half that in Group 2. The severity scores for bronchiolitis were approximately equal in Groups 2 and 4 at 48 hours post influenza treatment. Bronhchiolitis persisted, with low incidence and severity, only in Group 4 at 4 and 7 days. Influenza significantly increased interleukins 1-α and 6 and monocyte chemotactic protein-1 in lung, compared to Group 1 rats; brevetoxin exposure significantly increased the influenza – induced responses. These results suggest that repeated inhalation exposure to brevetoxin may slightly impair clearance and enhance the pathogenicity of influenza A in the rat lung. Research conducted under NIEHS P01 ES10594.

PS 2180 CARBON NANOFIBERS AND NANOTUBES DIFFER IN THEIR ALLERGY-PROMOTING CAPACITY IN MICE.

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There is a growing concern regarding the health impact of various nanoparticles. The aim of the present study was to compare the allergy-promoting capacity of carbon nanofibers (CNF) and nanotubes (CNT). We also aimed to identify major physicochemical characteristics important for the biological effects of CNF and CNT. Four qualitatively different CNF samples from one manufacturer, as well as single-walled (sw) and multi-walled (mw) CNT, were tested in two allergy mouse models. The particles were given by s.c. injection into the footpad or intranasally to BALB/cA mice together with the allergen ovalbumin (OVA). After an allergen booster, the allergic response was determined by measuring OVA-specific IgE in serum and eosinophil numbers in the bronchoalveolar fluid (BALF). OVA-specific IgG1 and IgG2a in serum and inflammatory cells and mediators (MCP-1 and TNF-α) in BALF were also measured. The four CNF samples and the sw and mw CNT all increased the OVA-specific IgE levels. The IgE response was markedly stronger for the sw and mw CNT, which also was associated with an eosinophil inflammation in the lung. The four CNF samples with different physicochemical characteristics had similar IgE adjuvant capacity in the airways. However, two of the CNF samples with several characteristics in common stood out from the other CNF with regard to a number of the other endpoints. Overall, we demonstrate that CNF and CNT promote allergic responses in two allergy models in mice, and that nanotube-specific properties appear to be especially potent in inducing allergic adjuvant effects. Further, our data suggest that particle properties like the tube structure, fiber or tube width, relative surface area, metal content and structural defect sites all deserve attention in future toxicological studies, to enable optimalization of the production process towards less toxic nanoparticles.

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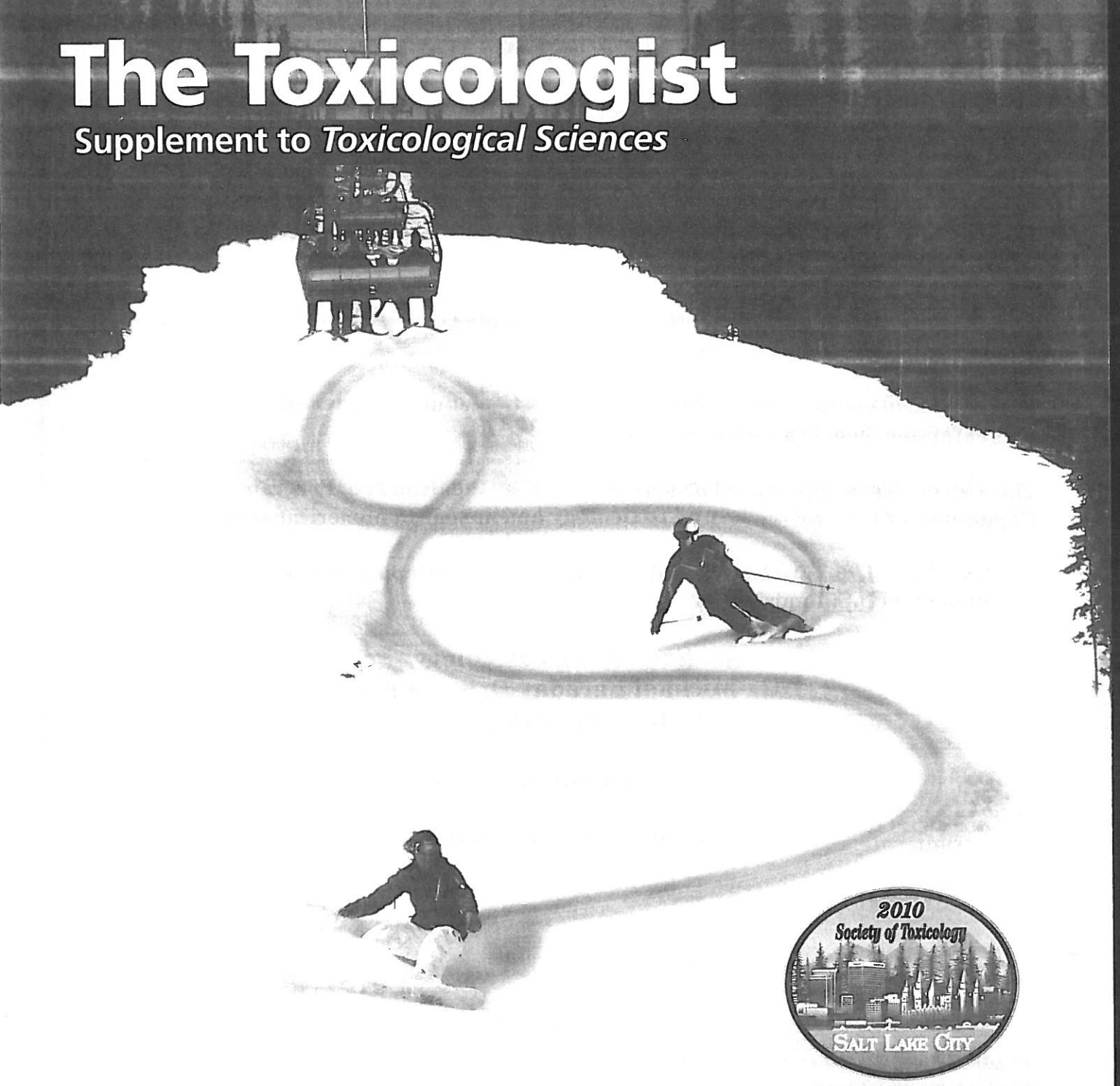

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Preface

This issue of *The Toxicologist* is devoted to the abstracts of the presentations for the Continuing Education courses and scientific sessions of the 49th Annual Meeting of the Society of Toxicology, held at the Salt Palace Convention Center, March 7–11, 2010.

An alphabetical Author Index, cross referencing the corresponding abstract number(s), begins on page 473.

The issue also contains a Key Word Index (by subject or chemical) of all the presentations, beginning on page 496.

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

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