and bone formation on tungsten-mediated effects on bone biology, which would provide insight into the underlying molecular mechanisms. In addition, our work helps to identify susceptible subgroups of the population that could be susceptible to tungsten-mediated effects on bone biology.

1556 Alterations in Antioxidant/Oxidant Proteins Following Treatment of Transformed and Normal Colon Cells with Tellurium Compounds

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Tellurium (Te) is a metalloid, with no known physiologic role in humans. Increasing use of Te compounds in optical blue ray discs and photographic materials suggests that environmental exposure will increase in the future as these products make their way into land fills. Occupational exposure to individuals involved in production of these products may also occur. The neurotoxicity of Te compounds has been documented, particularly in animal studies. However, little has been reported regarding the gastrointestinal toxicity of Te, despite the fact that ingestion represents a likely exposure route. A previous study in our lab has demonstrated that tellurium tetrachloride (TeCl4) causes necrosis and diphenyl ditelluride (DPDT) causes apoptosis via the intrinsic pathway in transformed and normal colon cells. Antioxidant defense systems serve to counterbalance the effects of oxidants and prevent oxidative damage. The purpose of the current study was to evaluate the potential of tellurium compounds TeCl4 and DPDT to induce oxidative stress by examining the protein levels of cyclooxygenase-2 (COX-2), glutathione reductase (GR) and NADPH dehydrogenase quinone 1 (NQO1) in CCD-18Co cells and cytoglobin (CYGB) and neutrophil cytosolic factor-1 (NCF-1) in HT-29 cells. Upregulation of these respective genes were also noted in qPCR in both the cell lines. A significant increase in COX-2 activity was observed at concentrations of 500 µM-1000 µM DPDT and 125 µM-1000 µM TeCl4 after 24 hr exposures. Significant increase in NQO1 was also observed at concentrations of 500 µM-1000 µM of DPDT and TeCl4 in CCD-18Co cells. In HT-29 cells, increase in Cygb was noted at concentrations of 500 µM-1000 µM DPDT and TeCl4 while NCF-1 increases were noted at the 1000 µM concentration of DPDT and TeCl4. In conclusion, proteins levels of COX-2, and NQO1 were upregulated in HT-29 cells and NCF-1 and Cygb were upregulated in CCD-18Co.

1557 Protective Effect of Ethanolic Extract of *Grewia carpinifolia* Leaves on Vanadium-Induced Toxicity

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Pentavalent vanadium (metavanadate salt) toxicity is a challenging environmental hazard that affects living organisms. Studies have shown that plants play important roles in protecting against heavy metal toxicity. This study was designed to evaluate the protective activity of ethanolic extracts of *Grewia carpinifolia* leaves following vanadium toxicity. Twenty five male Swiss mice were randomly divided into five groups (A-E) of five rats each. Group A rats served as control and were given distilled water, Group B was administered with sodium metavanadate and a known antioxidant agent; α-tocopherol, Groups C and D were administered with sodium metavanadate and ethanolic extract of *Grewia carpinifolia* leaves orally at 100 and 200 mg/kg body weight respectively while Group E was administered with only sodium metavanadate. After a daily single oral dosing for seven days, changes in behaviour, haematology and serum biochemistry parameters were analysed. Sodium metavanadate caused a significant decrease ($p \leq 0.05$) in haematocrit levels, haemoglobin (Hb) concentration, white blood cell count, neutrophil count and serum cholesterol level. A significant ($p \leq 0.05$) lymphocytosis was also observed in the group administered with sodium metavanadate alone. *G. carpinifolia* extract given concomitantly with sodium metavanadate was able to restore PCV, Hb concentrations and serum total protein to levels comparable with the control and standard groups. *Grewia carpinifolia* also significantly reduced the elevated serum levels of AST and ALT after vanadium induced hepatotoxicity. Our findings suggest that *G. carpinifolia* extract protected against the toxicity induced by vanadium; the plant extract at 200 mg/kg however appear to offer a better protection.

1558 Comparison of the Toxicity of Sintered vs. Unsintered Indium-Tin Oxide Particles on Murine Macrophage and Epidermal Cells

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Revenues from consumer electronics are at an all-time high, leading to an increased demand for indium-tin oxide (ITO) to produce touch screens and plasma and liquid crystal displays. Composed of 90% indium oxide (In2O3) and 10% tin oxide (SnO2) by weight, ITO is synthesized under conditions of high heat via a process known as sintering. Concerns have arisen over the health of workers in the ITO industry, as severe pulmonary toxicity and increased levels of indium in blood have been associated with occupational exposure to ITO. In the current study, murine macrophage (RAW 264.7) and epidermal (JB6/AP-1) cells were used to differentiate between the toxicological profiles of sintered ITO (SITO) and an unsintered ITO (UITO) mixture. We hypothesized that sintering would play a key role in free radical generation and cytotoxicity. Cells were treated with either 50 µg/ml, 150 µg/ml or 250 µg/ml of ITO and various endpoints were measured over time. Exposure of cells to both UITO and SITO caused a time and dose dependent decrease on the viability of cells. Intracellular ROS generation was inversely related to the concentration of both UITO and SITO, a direct reflection of the decreased number of viable cells observed at higher concentrations. Electron paramagnetic resonance, used to measure free radical generation, showed significantly increased hydroxyl radical generation in cells treated with UITO versus those treated with SITO. This is different from observed LDH release, which showed that SITO caused significantly increased damage to the cell membrane compared to UITO. Our results delineate the need for a better understanding of the mechanisms of toxicity and free radical production associated with workplace exposure to indium, allowing for earlier disease detection and improved health amongst workers.

1559 Prevalence of Anti-Nuclear Antibodies Among Navajo Birth Cohort Study Participants with Elevated Urine Uranium Levels

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Native American Tribal communities in the United States are concentrated in the Western US, a region home to the highest concentration of abandoned hardrock mines (over 161,000) in the country. The Navajo Nation currently has more than 500 abandoned uranium mines. The Navajo Birth Cohort Study (NBCS) is an ongoing prospective study enrolling mothers, fathers, and babies to investigate the impact of environmental exposure to metals found in mine wastes on pregnancy, birth outcomes and early child development. NBCS participant samples were analyzed for 36 metals at the CDC Laboratory in Atlanta GA. To date, 85.9% of tested samples (N=455) have urine uranium above the NHANES 50th percentile (i.e. >0.006µg/L) and 24.4% have levels greater than the 95th percentile (i.e. >0.031 µg/L). Development of anti-nuclear antibodies (ANA) has been associated with environmental exposure to metals, and increased levels of maternal ANA with or without a maternal autoimmune disease diagnosis have been associated with development of autism spectrum disorders in children, providing a potential link between environmental exposures, ANA, and child development. Serum samples from 80 NBCS participants (40 male, 40 female) were tested for ANA using a microparticle immunoassay for 10 common autoantigens (centromere B, ribosomal P, chromatin, ds-DNA, Smith, mRNP, SS-A, SS-B, Scl-70 and Jo-1). The ANA prevalence for the US as a whole is 13.8%, and more frequent in females and older individuals. Within this young cohort (average age of 27), 18.8% of the samples (15/80) were ANA(+), with equal distribution across gender. Statistical analysis showed an interaction between urine uranium, gender and ANA, with a positive relationship for both genders, though stronger in males ($p=0.05$), suggesting a relationship between environmental exposure to uranium and induction of ANA. Development of ANA can precede clinical disease diagnosis by many years, making ANA a potential biomarker of metal exposures and providing important insights into mechanisms of metals-induced immunotoxicity such as immune dysregulation, oxidative stress and/or DNA damage. These findings warrant additional investigation of mixed metal exposures in addition to uranium within this young population.

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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 55th Annual Meeting of the Society of Toxicology, held at the New Orleans Ernest N. Morial Convention Center, March 13–17, 2016.

An alphabetical Author Index, cross referencing the corresponding abstract number(s), begins on page 603.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 629.

The abstracts are reproduced as accepted by the Scientific Program Committee of the Society of Toxicology and appear in numerical sequence. Author names which are underlined in the author block indicate the author is a member of the Society of Toxicology. For example, J. Smith.

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- CE Continuing Education Courses
- EC Education-Career Development Sessions
- FS Featured Sessions
- HH Historical Highlights Sessions
- IS Informational Sessions
- PL Platform Sessions
- PS Poster Sessions
- RI Regional Interest Sessions
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- S Symposium Sessions
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