levels while the 0.125 mg/ml plain MWCNT + Microwave treatment group showed reduced levels. The majority of groups given microwave treatment had lower levels of creatinine. All treatment groups showed an upregulation of NFKB and TNF, while IL6 and PTSG2 were downregulated and IL1B was not expressed at all. Results showed minimal to no effects two weeks after a single injection in mice with CNT-Ab followed by microwave hypothermia. Study is still ongoing to examine histopathological data.

## 2110 A Toxicological Assessment of Crystalline Nano-Cellulose in Mice

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Crystalline nanocellulose (CNC) is derived from the natural polymer, cellulose. Considering the numerous applications of CNC, a substantial number of workers could be exposed to CNC. This study investigates the time-course of onset, progression, and resolution, if any, of CNC-induced toxicity. For hazard ranking, the outcome of the CNC toxicity studies was compared to those induced by a larger-sized crystalline micro cellulose; CMC, and to a reference material, Mitsui multi-walled carbon nanotubes (MWCNT). Mice (male, C57BL/6J, 7 weeks old) were administered dispersion medium (DM) or DM containing CNC, CMC (50, 100, and 200 µg/mouse), or MWCNT (50 µg/mouse) as a single pharyngeal aspiration. At 1 day, 7 days, 1 month, 3 months, and 6 months post-exposure toxicity facilitated by exposure to the varying materials was assessed. Serum transaminases were measured to determine hepatotoxicity. Whole lung lavage was conducted to assess pulmonary toxicity (PMN number and LDH activity). Sperm counts and motility analyses were used to assess reproductive toxicity. All doses of CNC caused a significant increase in PMN number and LDH activity at 1- and 7-days post-exposure. For CMC, an increase in PMN number and LDH activity was also found at 1-day post-exposure, for all doses. However, the inflammatory response of CNC (number of infiltrating PMNs) was significantly greater when compared to the response facilitated by CMC. The 200 µg/mouse dose of CNC also caused significant increases in serum ALT and AST at 7-days post-exposure compared to controls. CMC exposure also facilitated a significant increase in serum AST at the 200 µg/mouse dose at 1-day post-exposure. MWCNT resulted in significant pulmonary inflammation. However, at 1-day post-exposure, CNC (50 µg/mouse) caused a 2-fold greater increase in PMN number compared to that by MWCNT. By 7-days post-exposure MWCNT caused a 3-fold greater increase in PMN number compared to CNC. Additionally, there was no treatment related changes in sperm count or motility for any dose/post-exposure time combination of CNC, CMC, or MWCNT. In conclusion, the physico-chemical properties, such as the enhanced surface area of the CNC, are likely driving the more robust pulmonary toxicity response as compared to the pulmonary toxicity caused by the larger micron-sized CMC. Furthermore, MWCNT-facilitated pulmonary inflammation that was more persistent than CNC.

## 2111 Pulmonary Responses to Subacute Cadmium Sulfide Nanoparticle Inhalation

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The uses and applications of engineered nanomaterials (ENMs) are expanding at an astounding rate. One such application is the manufacturing of quantum dots using cadmium sulfide (CdS) nanoparticles. CdS quantum dots are semiconductors with a direct intermediate bandgap and excellent thermal stability and thus have shown strong potential for wide scale use in solar cells, light emitting diodes, and specialty lasers. Industrial scale manufacturing of CdS quantum dots requires bulk quantities of CdS nanoparticles. To evaluate toxic risk associated with industrial CdS nanoparticle use, we performed a sub-acute nose-only inhalation exposure (3.49 ± 0.49 mg/m$^3$) to CdS aerosol (geometric mean mobility diameter 41 nm, geometric standard deviation 1.8) using a murine model. Analysis of bronchoalveolar lavage (BAL) fluid showed a 12-fold increase in total BAL cells per mouse immediately after 2 wk exposure accompanied with inflammatory cell infiltration (41% of these cells were neutrophils) that persisted through 3 weeks of rest after exposure (the number of neutrophils decreased only to 29%). Histopathologic analysis of lung tissue confirmed the presence of inflammatory cell infiltration. Exposed mice demonstrated an increase in inflammatory cytokines in the BAL fluid. There was evidence of lipid peroxidation in both lung tissue and serum of exposed mice (TBARS assay). Our analysis shows that sub-acute inhalation of CdS nanoparticles leads to pulmonary inflammation and an increase in ROS. Determination of Cd in selected tissues to assess deposited and cleared Cd after exposure as well as evaluation of pulmonary mechanics parameters is in progress. *Supported by: NIH U01 ES027252 & NIH P30 ES005605.*

## 2112 Effects of Iron Oxide Nanoparticles (IONPs): Cytotoxicity and Genotoxicity in Wistar Rat Brain

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The utilization of iron oxide nanoparticles in several biomedical applications, has received much attention due to their unique properties, such as extremely small size, high surface-area-to-volume ratio, excellent magnetic properties and great biocompatibility. Although, the potential benefits of iron oxide nanoparticles are considerable, there is a distinct need to identify any potential risk associated with their usages. It has been demonstrated earlier that corpus striatum and hippocampus are the major sites of IONPs accumulation in brain. The high surface activity of these IONPs deposited for long term in brain may cause neurodegenerative disorders. Our study put forward a notion that in making of a perfect treatment vehicle, any nanoparticle has to pass through various parameters of cytotoxic and genotoxic evaluation. Thus, the present study was designed to evaluate the cytotoxic and genotoxic effects of iron oxide nanoparticles (IONPs) *in vivo*. In order to study the toxic effects, male Wistar rats (6-8 weeks old) were randomly divided into four groups, group I served as control, group II treated with 20.322 mg/kg body weight, group III treated with 40.644 mg/kg body weight and group IV treated with 81.288 mg/kg body weight. Animals of each group were intraperitoneally administered the selected dose daily for 28 days. They were sacrificed on post exposure day 7$^{th}$, 14$^{th}$ and 28$^{th}$ and brains dissected out for various biochemical assays such as antioxidant enzymes activity glutathione-S-transferase (GST), glutathione peroxidase (GPX), and glutathione reductase (GR), and genotoxicity assessment through single cell gel electrophoresis assay, in the tissue homogenates of four brain sub regions namely, frontal cortex, corpus striatum, hippocampus and cerebellum. The results revealed that the activity of antioxidant enzymes (GST, GR, GPx) elevated in IONPs treated groups in dose-dependent manner but non-significantly, however, activity of GR and GPx on 14$^{th}$ day of investigation for group IV animals was found significant. The comet assay results indicate that IONPs did not induce any significant DNA damage following exposure of IONPs at various dose levels. The present study concluded that IONPs are safe to use under prescribed doses. But it can induce oxidative stress as well as genotoxicity at higher doses of the particles with or without coatings. Therefore, there is a considerable need to address biocompatibility and biosafety concerns associated with their usage to avoid the adverse side effects of nanoparticles when used as biomedical tools.

## 2113 Time Course of Pulmonary Toxicity and Biodistribution during and after Subacute Inhalation Exposure to Copper Oxide Nanoparticles in a Mouse Model

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Research interest in nanotoxicity has dramatically increased due to the exponential growth of nanomaterial applications and nanotechnology because of their unique physicochemical properties compared to bulk materials. Copper oxide nanoparticles (CuO NPs) have been widely used in many applications such as antimicrobial agents, solar cells, catalysts, and electronic products. CuO NPs are highly cytotoxic compared to other metal oxide NPs. The inhalation is potentially the most significant route for unintentionally exposure especially in occupational settings. Several murine studies demonstrated that inhalation exposure to CuO NPs induces pulmonary inflammation. However, available data on the CuO NPs biodistribution relating to their toxicity are still limited. The goal of this study was to investigate the pulmonary toxicity and biodistribution of inhaled CuO NPs during and after subacute exposure. The time course of CuO NPs biodistribution will increase our understanding of CuO NP toxicity particularly in the pulmonary region. In this study, female BALB/CJ mice were exposed to CuO NPs at 3.75 mg/m$^3$ (geometric mean of aerosolized CuO NPs was 77.6 nm) using a nose-only exposure system for 4 hr/day, 5 days/week over a 2-week period and were necropsied on days 0, 3, 7, 10, 12, 17, 22, and 27. Sera, bronchoalveolar lavage (BAL) fluid and lung tissue were collected to measure 23 cytokine/chemokines levels, numbers of inflammatory cells, and lactate dehydrogenase (LDH). Histopathology of lungs was also evaluated. Whole blood, lung, brain, heart, kidney, liver, and spleen were collected to measure copper concentration by inductively coupled plasma mass spectrometry (ICP-MS). The number of macrophages, lymphocytes and neutrophils in BAL fluid were gradually increasing with a culmination on day 22, 17 and 12, respectively. Rate of weight gain, and LDH levels in BAL fluid exhibited similar changes as neutrophils. Significantly higher concentrations of many cytokines/chemokines (IL-12 (p40), KC, MCP-1, MIP-1a) persisted several days after the end exposure. The amount of copper in the lung tissues of all exposure groups increased significantly compared to the control group with a gradual increase observed during exposure and a decline subsequent to exposure. However, the copper concentration in whole blood exhibited a

SOT 2020

59th Annual Meeting & ToxExpo

Anaheim, California • March 15–19

# The Toxicologist

## Supplement to *Toxicological Sciences*


Toxicological Sciences

ISSN 1096-6080
Volume 174, Issue 1
March 2020

The Official Journal of the Society of Toxicology

OXFORD UNIVERSITY PRESS

SOT | Society of Toxicology

www.academic.oup.com/toxsci

Publication Date: February 21, 2020

# Preface

This issue is devoted to the abstracts of the presentations for the Continuing Education courses and Scientific Sessions of the 59th Annual Meeting of the Society of Toxicology, held at the Anaheim Convention Center, Anaheim, California, March 15–19, 2020.

An alphabetical Author Index, cross-referencing the corresponding abstract number(s), begins on page 542.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 580.

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To cite a 2020 SOT Annual Meeting abstract, please format as follows: *The Toxicologist*, Supplement to *Toxicological Sciences*, 174 (1), abstract #__, 2020, Title, First Author.

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