Submit your proposal online at www.toxicology.org
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.

Scientific Session Types:

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

The 2016 SOT Mobile Event App and Online Planner

The Mobile Event App and Online Planner are available via the SOT website and app marketplaces. These mobile tools enable you, the attendee, to engage with organizers, exhibitors, and each other, and to manage your time and maximize your experience during the Annual Meeting. You also can access some ePosters electronically via the Mobile Event App until May 11, 2016.

To cite a 2016 SOT Annual Meeting Abstract, please format as follows: The Toxicologist, Supplement to Toxicological Sciences, 150 (1), Abstract #__, 2016, Title, First Author.

Copies of The Toxicologist are available at $40 each plus shipping ($15 shipping & handling in the USA and $50 for overseas shipments) from:

Society of Toxicology
1821 Michael Faraday Drive, Suite 300 • Reston, VA 20190

www.toxicology.org

© 2016 Society of Toxicology

All text and graphics are © 2016 by the Society of Toxicology unless noted. For promotional use only. No advertising use is permitted.

This abstract book has been produced electronically by the Society of Toxicology. Every effort has been made to faithfully reproduce the abstracts as submitted. The author(s) of each abstract appearing in this publication is/are solely responsible for the content thereof; the publication of an article shall not constitute or be deemed to constitute any representation by the Society of Toxicology or its boards that the data presented therein are correct or are sufficient to support the conclusions reached or that the experiment design or methodology is adequate. Because of the rapid advances in the medical sciences, we recommend that independent verification of diagnoses and drug dosage be made.
Titanium dioxide (TiO2) has been used in a broad spectrum of consumer products, including food, cosmetics and various medical products. In recent years, the use of inorganic TiO2 in research has raised concerns about their safety. In the current study, we characterized TiO2 nanoparticles with different crystal structures and particle sizes through electron microscopy, diffraction and scattering techniques. Subsequently, we determined the impact of TiO2 nanoparticles on cell viability using LDH, ATP assays, and their adenosine differentiation capacity using Oil red O staining assay in human mesenchymal stem cells (hMSC). We further investigated whether the impact of TiO2 nanoparticles was associated with specific particle size and/or crystal structure. Data revealed that TiO2 nanoparticles exhibited minimal acute (up to 72 hours) cytotoxicity in hMSCs. There was a size- and crystal structure-dependent inhibition of hMSC adipogenic differentiation (21 days) by TiO2 nanoparticles. Cellular uptake and media “striping” studies indicated that the inhibition of hMSC adipogenesis was likely due to direct cellular response to TiO2 nanoparticles instead of a “charcoal-striping” effect on the HTM2 cell line leading to reduced growth factors in the cell culture media. Additional exploratory gene expression array analyses suggested that TiO2 nanoparticles inhibit hMSC adipogenesis down-regulating key genes involved in adipogenesis promotion, including FGF2, IRS1, CEBPA, CEBPB, and ACACB, etc. Findings from this study indicate that TiO2 nanoparticles, while exhibiting minimal acute cytotoxicity, may impair long-term differentiation of hMSC, adipogenic differentiation. Future planned studies will reveal the mechanism of TiO2 nanoparticle interaction in stem cell models. Disclaimer: The views presented in this article do not necessarily reflect those of the Food and Drug Administration.

Zinc oxide (ZnO) nanoparticles are one of the promising materials applied in the various kinds of the commercial products such as sunscreens. At the same time, there are growing concerns about untended toxic effects of ZnO. Many researches on the toxicity of ZnO have been studied, except for the epithelial-to-mesenchymal transition (EMT). EMT is one of the significant multistep processes. Epithelial cells reduce intercellular adhesion and increase cell motility which is crucial for the cancer metastasis. The aim of our study is to investigate the ZnO-induced EMT in the human alveolar epithelial A549 cells. At first, size distribution of ZnO nanoparticles was measured by dynamic light scattering (DLS). The cytoxicity test was performed to find the appropriate exposure concentration. Real-time PCR was used for the mRNA expression, related with EMT. In addition, the EMT-associated protein level was measured by western blot. The morphology changes were obtained by microscopy. The average size of ZnO showed 208 ± 16.10 nm measured by DLS. Cell viability as the cytotoxic effects was decreased in a dose-dependent manner. When A549 cells are exposed to ZnO nanoparticles, mRNA level of EMT-related transcription factor was increased including Snail. Furthermore, the protein levels related with EMT markers were found to be upregulated. After characterization, different concentrations and time periods were tested in regards of HepG2 viability, by employing MTT and Alamar Blue assays, and DNA integrity, by using comet assay. Results and Discussion: Results showed that all studied materials were capable to induce hepatocyte cell death in a dose dependent way and for the majority of the studied periods of exposure. Besides that, the HepG2 DNA was also affected after longer periods of exposure to TiO2 NPs, kaolinite HTM2 and NdMWCNTs. Conclusions: Nanocomposites are promising materials for different nanotechnological applications. Notwithstanding, it is of paramount importance to evaluate their potential toxicity. Data obtained suggests that other substrates must be tested to immobilize TiO2 NPs as kaolinite mineral was found to be both cytoprotective and genotoxic for the studied cell line. Acknowledgements: Financial support from TD1204 MODENA COST Action.