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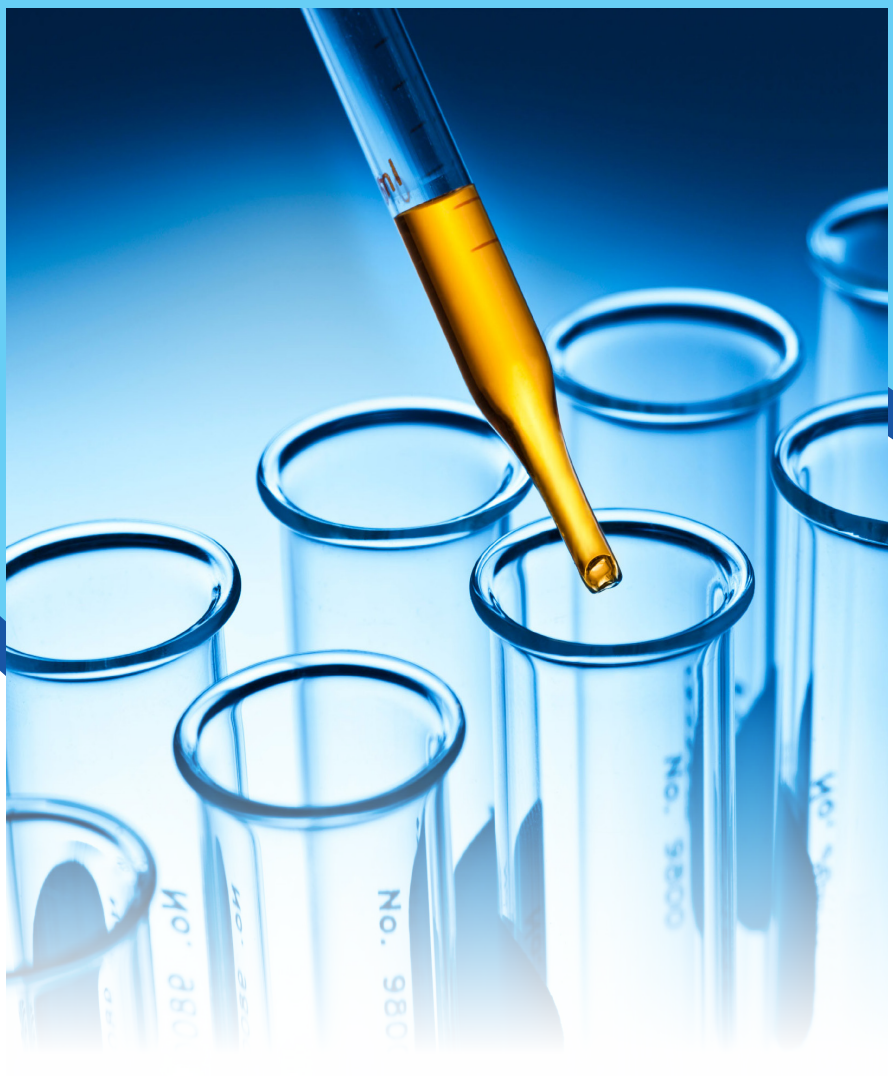
Toxicology and Pharmacology

May 06-07, 2019 Tokyo, Japan



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20th World Congress on

TOXICOLOGY AND PHARMACOLOGY

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Keynote Forum

Day 1

Alexei G Basnakian, Toxicol Open Access 2019, Volume 5
DOI: 10.4172/2476-2067-C1-007

Alexei G Basnakian
University of Arkansas for Medical Sciences, USA

DNase/endonuclease network: A new target for mitigation of acute nephrotoxicity

Deoxyribonucleases (DNases) universally induce irreversible cell death by fragmenting DNA in response to cell injury. All of the nine known cell death DNases are endonucleases. Despite that most of the DNase activity is used after cell death, a genetic inactivation of DNases provide protection of cells and tissues against DNA breaks induced by cytotoxic stimuli and partially protect against tissue injury. Therefore, DNases act before the point-of-no-return in cell death and can be potentially used as therapeutic targets for tissue protection against injury. Our studies identified two DNases, DNase I and EndoG, as being responsible for tubular epithelial toxicity during acute kidney injury induced by Cisplatin, rhabdomyolysis or ischemia. However, inhibitors of DNases are not available. The aim of this study was to identify DNase inhibitors, which might be used for mitigation of acute kidney injury. To identify DNase inhibitors, we have developed a high-throughput screening assay based on a proprietary fluorescent probe. This assay allowed the identification of several new inhibitors of deoxyribonuclease I (DNase I), which were also active against two other DNases, endonucleases G (EndoG) and Deoxyribonuclease II (DNase II). The DNase inhibitors were able to significantly protect kidney tubular epithelial cells *in vitro* and mouse kidneys *in vivo* against acute kidney toxicity induced by Cisplatin, glycerol (rhabdomyolysis), or renal ischemia-reperfusion. The inhibitors showed no toxicity *in vivo* at 5x therapeutic doses. DNases can be used as a therapeutic target for mitigation of toxic or hypoxic acute kidney injury. The identified DNase inhibitors or similar compounds have a great potential for tissue protection against toxic kidney failure. Considering that DNases are expressed and responsible for cell death in all tested cells, tissues and animal models, it is likely that the same compounds may be used for universal tissue protection against various injuries.

Biography

Alexei G Basnakian has completed his PhD and DSc degrees from the Russian Academy of Medical Science, both in the field of DNA-degrading enzymes. He had Post-doctoral trainings in Molecular Biology at the Harvard Medical School and in Toxicology/Cancer Research at the National Center for Toxicological Research/U.S. Food and Drug Administration. He is a tenured Professor at the Department of Pharmacology and Toxicology and also Director of the DNA Damage and Toxicology Core Center at the University of Arkansas for Medical Sciences and Research Career Scientist at the Veterans Hospital in Little Rock, USA. He is the author of 87 peer-reviewed papers and 14 reviews or book chapters. He is an Editorial Board Member of four biomedical journals and also a Member of NIH, AHA and VA grant study sections. His research interests are in DNases/endonucleases and DNA damage associated with toxicity, anti-cancer therapy, tissue injury and cell death.

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Stephanie Lora Sharp, Toxicol Open Access 2019, Volume 5
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Stephanie Lora Sharp
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The role of the forensic pharmacologist as an expert witness

Forensic pharmacology is a fascinating discipline internationally, covering everything from drink driving to the influence of drugs and alcohol in rapes and murders. Scotland is in the unenviable position of being the worst country in Europe for illicit drug-related deaths. The scope of my interest is from medical negligence to drugs of abuse such as new psychoactive substances. I take a particular interest in drugs of abuse and further to that the impact scientific expert evidence has in determining the outcome of cases and how this may be subverted by the judicial process. I will be sharing specific case studies regarding the role of a scientist as an expert witness and the judicial outcomes of the cases. I will be addressing new psychoactive substances (Spice, synthetic cannabinoids, psychedelics, opioids) and medical misadventure resulting in death due to inappropriate therapeutic drug combinations. From the position that I am in, I feel that it is of great importance to highlight the systems in place for illicit drug use and regulation as the differences in these systems may have dire consequences for the user and society in general. In my opinion, decriminalization of drugs of abuse in Scotland, the United Kingdom and indeed, the rest of the World, will lead to a significant reduction in illicit drug-related harm as has been demonstrated in Portugal.

Biography

Stephanie Lora Sharp is a Forensic Pharmacologist, completed her Master of Science in Pharmacology from the University of Glasgow and PhD in Pharmacokinetics from the University of Dundee and Certificates in Civil and Criminal Law from the University of Cardiff. She has worked as a Research Scientist for eight years at University of Cape Town in South Africa and the University of Dundee. She is currently working as a Co-Director of the Glasgow Expert Witness Service Ltd. and is a registered Expert Witness with the Law Society of Scotland Directory of Expert Witnesses and also Professional Member of The Chartered Society of Forensic Sciences and a registered Expert Adviser on the National Crime Agency (NCA) database.

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Ming-Tsang Wu, Toxicol Open Access 2019, Volume 5
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Ming-Tsang Wu
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Environmental melamine exposure and renal diseases

We are still exposed to low-dose melamine in daily-life environment, even after 2008 toxic milk food scandal. One of the main sources is the intake of melamine chemical from the migration of melamine-made tableware, when contacted with high-temperature soup/water. Our previous study has found that chronic low-dose melamine exposure is associated with the risk of renal stones in adults, but the data about the relationship between environmental melamine exposure and the risk of renal damage in humans is still lacking. In this talk, I will present our recent findings about that link from different susceptible populations and propose the mechanisms behind that.

Biography

Ming-Tsang Wu has completed his MD from Chung Shan Medical University in Taiwan and PhD from Harvard School of Public Health in the USA. He is a Professor in the Department of Public Health and also the Director in Research Center for Environmental Medicine, Kaohsiung Medicine University, Taiwan. His major research interest is on the interactive effects of environmental and occupational exposures, genetic factors and biomarkers on the health outcomes.

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Day 2

Lidia Zapor, Toxicol Open Access 2019, Volume 5
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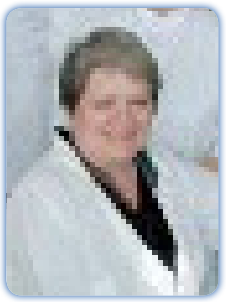

Lidia Zapor
Central Institute for Labour Protection, Poland

Cytotoxicity of molybdenum trioxide nanoplates

Introduction & Aim: Nanostructured molybdenum trioxide (MoO_3-NPs) is promising material in many applications, in coatings, plastics, textiles, pigments, lubricants, ceramics and glass production, as antimicrobial agents and for the detection of Dopamine in pharmaceutical and clinical preparations. In recent years, it was noted that in the case of nanomaterials should carefully evaluate the risks of their use, as they may pose a health risk. The main aim of this study was to assess the cytotoxic activity of MoO_3-NPs in human pulmonary cells.

Methods: The cytotoxicity of MoO_3-NPs was assessed on the alveolar carcinoma epithelial cells (A549) and normal bronchial epithelium cells (BEAS-2B) after short and long-term time of exposure. Cytotoxicity studies included the effect of MoO_3-NPs on cell viability, cell membrane integrity (NRU assay), mitochondrial metabolic activity (MTT assay) and the ability of the cells to proliferation (Clonogenic assay).

Results: MoO_3-NPs induced a dose- and time-related negative effect on the viability of both kids of the cells in the cytotoxic doses range 50-300 µg/ml, depending on cytotoxicity endpoint. In long-term exposure (7 day), MoO_3-NPs at concentrations about 100 µg/ml impaired proliferations, implying their potential chronic toxicity. A549 cells were less sensitive than BEAS-2B one, to all measurement parameters.

Conclusion: The sensitivity of BEAS-2B cells to MoO_3-NPs is of particular concern. These cells form a defense line of the body against the penetration of particles into lungs. Inhibition of the ability of BEAS-2B cells to proliferate under the influence of MoO_3-NPs may be an unfavorable phenomenon for predicting their long-term effects of exposure.

Biography

Lidia Zapor is currently working in the Department of Chemical, Aerosol and Biological Hazards in the Central Institute for Labour Protection, National Research Institute (CIOP-PIB). She is the Head of Laboratory of Toxicology. The main area of her professional interest is problems of human in the working environment and the toxicity of chemical substances as well as methods of estimating the toxicity of substances *in vitro*.

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