The impact of zinc oxide nanoparticles on metabolic activity in the cells of respiratory and reproductive system

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Because of expanding application of zinc oxide nanoparticles (ZnO-NPs) for industrial purposes, unintended human exposure in the work environment is highly probable, hence an evaluation of potential toxicity of ZnO-NPs is necessary. It is reasonable it more, that toxicological data on the toxicity of ZnO-NPs are inconclusive. The aim of this study was to assess the impact of ZnO-NPs on reactive oxygen species (ROS) formation in the cells of respiratory and reproductive system depending on the particle size of ZnO-NPs.

Toxicity of different sizes of ZnO particles, i.e., ZnO nanopowders (ZnO<50 nm; ZnO<100 nm particle size), microsized metallic ZnO powder (ZnO-micro) and stabilized commercially dispersion of ZnO<100 nm was evaluated on four cellular lines: Chinese hamster ovary cells (CHO), mouse testicular Sertoli cells (15P-1), human pulmonary cancer cells (A549) and mouse macrophage cells (RAW264.7) after 24-hour exposure. ZnO-NPs were characterized in terms of particle morphology by scanning electron microscopy (SEM) and particle size distribution by dynamic light scattering (DLS) methods.

The effect of ZnO-NPs on cell viability, metabolic activity, lipid peroxidation, generation of intracellular ROS and total oxidative and antioxidative capacity of cells was studied. Additionally bioavailability of zinc [Zn^{2+}] for cells was assessed by proton induced X-ray emission analysis (PIXE).

The SEM and DLS methods confirmed that there was a size difference between the ZnO-NPs, but there was strong tendency to aggregation of particles. All tested ZnO-NPs induced a dose-related negative effect on cell viability and caused oxidative metabolism disturbance assessed on the basis of lipid peroxidation (Fig. 1), intracellular ROS generation and total oxidative/antioxidative capacity of cells. It is worth noting that increase in total oxidative status of cells was observed at concentrations lower than cytotoxic doses. These results suggest that the toxicity of ZnO-NPs is caused by elevated oxidative stress.

The results of toxicity correspond well with the results of bioavailability of zinc oxide particles. The zinc oxide particles forming aggregates were weakly available for the cells so less toxic (Fig.2).

Conclusion: Determination of the physicochemical parameters of the nanoparticles is of fundamental importance in exposure assessment, since the physical properties (particle size distribution, state of aggregation) affect the toxic potency of nanomaterials. The phenomenon of aggregation of nanoparticles may be conducive working environment because it reduces their bioavailability and toxicity.

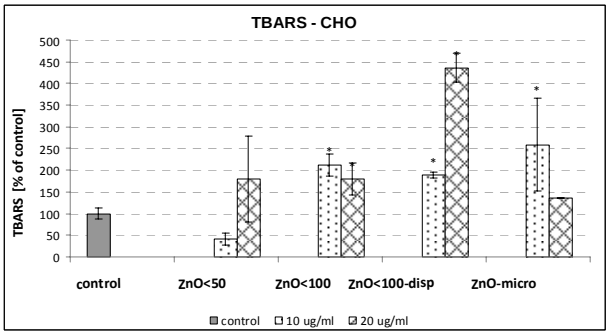

Figure 1. A comparison the effect of ZnO-NPs on lipid peroxidation in CHO cells. Cells were treated with 10, and 20 ug/ml ZnO for 24 h and controls received culture medium only. The thiobarbituric acid assay (TBARS) was used to detect lipid peroxidation. Results expressed as % of control. Each bar represents an average value ± S.D. of at least three independent experiments.

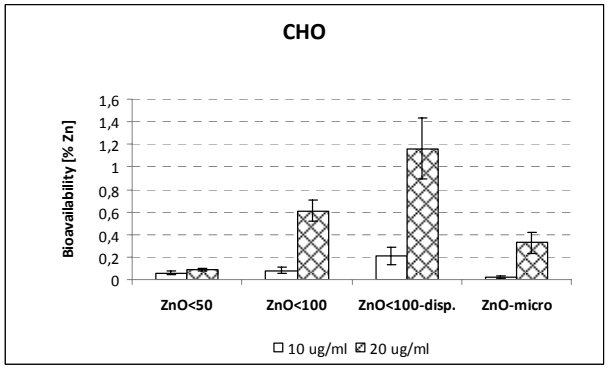

Figure 2. A comparison the bioavailability of Zn[2+] in CHO cells exposed to equimolar concentrations of ZnO-NPs compared to the concentration of Zn[2+] in the solution [%]. Each bar represents an average value ± S.D. from at least three experiments

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