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Toxicity and Oxidative stress induced by metallic nanoparticles in renal cells

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Nanoparticles (NPs) possess the ability to cross the blood barrier, depending particularly on their size and surface properties. Unlike particles of microscopic size express local toxicity, NPs induce systemic effects on target organs such as kidney. In this study, we investigated in vitro interactions of manufactured NPs on human renal cells to highlight their induced toxic and biological responses. All studies were performed on glomerulus (IP15) and tubular (HK-2) cell lines after exposure to metallic NPs: TiO_2, ZnO and CdS. The aim of this study was first to characterize NPs in biological environment, to determine cell viability after 24h exposure and to compare NPs effects depending on their solubilization, composition and size. Secondly, we identified whether particle properties influence cytotoxicity by altering intracellular oxidative conditions.

Our data indicate that NPs could be internalized as agglomerates within cytoplasmic vesicle. Exposure to CdS and ZnO NPs lead to cell death (IC_{50} CdS: 5-6 µg/cm²; IC_{50} ZnO: 2-3 µg/cm²) partly by their solubilized fraction. TiO_2 NPs have no cytotoxic effect (IC_{50} TiO_2> 100 µg/cm²), probably due to their insolubility. The cytotoxic effects are related with reactive oxygen species (ROS) generation and intracellular oxidative stress measured by a decrease of reduced glutathione (GSH) (70-90 %, for CdS and ZnO) and oxidized glutathione (GSSH) production.

In this study, we used in vitro models to highlight the involvement of the oxidative stress on the cytotoxicity of metal NPs on kidney cells. Cytokine production and molecular

pathways (especially transcription factors such as NF-κB) must be further investigated to better understand mechanisms of cellular defense.

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