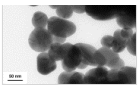
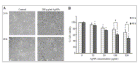
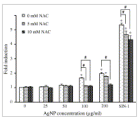
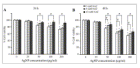
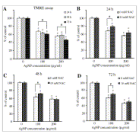
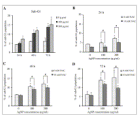
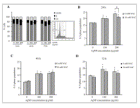

Toxicology in Vitro

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Silver nanoparticles induce toxicity in A549 cells via ROS-dependent and ROS-independent pathways

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Abstract

Silver nanoparticles (AgNPs) are incorporated into a large number of consumer and medical products. Several experiments have demonstrated that AgNPs can be toxic to the vital organs of humans and especially to the lung. The present study evaluated the *in vitro* mechanisms of AgNP (<100 nm) toxicity in relationship to the generation of reactive oxygen species (ROS) in A549 cells. AgNPs caused ROS formation in the cells, a reduction in their cell viability and mitochondrial membrane potential (MMP), an increase in the proportion of cells in the sub-G1 (apoptosis) population, S phase arrest and down-regulation of the cell cycle associated proliferating cell nuclear antigen (PCNA) protein, in a concentration- and time-dependent manner. Pretreatment of the A549 cells with N-acetyl-cysteine (NAC), an antioxidant, decreased the effects of AgNPs on the reduced cell viability, change in the MMP and proportion of cells in the sub-G1population, but had no effect on the AgNP-mediated S phase arrest or down-regulation of PCNA. These observations allow us to propose that the *in vitro* toxic effects of AgNPs on A549 cells are mediated via both ROS-dependent (cytotoxicity) and ROS-independent (cell cycle arrest) pathways.

Highlights

► Mechanisms of silver nanoparticle toxicity in A549 cells was investigated. ► Cytotoxicity, reduced MMP correlated with ROS induction. ► Antioxidant pretreatment improved the adverse effects from ROS. ► S phase cycle arrest and decreased PCNA protein expression were independent of ROS. ► ROS-dependent (cytotoxicity) and -independent (anti-proliferative effects) pathways.

Keywords

Silver nanoparticles; A549 cells; Mitochondrial membrane potential; NAC; Cell cycle; PCNA

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