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Oxidative stress and mitochondria damage contribute to silica nanoparticle-induced cytotoxicity in human endothelial cells

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Nanoparticles (NP) present possible dangers, both medically and environmentally. Most of these are due to the high surface to volume ratio, which can make the particles very reactive or catalytic. They are also able to pass through cell membranes in organisms, and their interactions with biological systems are relatively unknown. Given the central role that the endothelium plays in cardiovascular (CV) homeostasis and the involvement of endothelial cell (EC) dysfunction in CV diseases pathogenesis, the EC represent an excellent model to investigate the impact of NP on vascular (patho)physiology. In order to elucidate the NP-induced cytotoxicity and its mechanism, the effects of 100 nm silica NP (SiO_2) on cultured human EC line (ECV304) were investigated. Cell viability, mitochondrial function, reactive oxygen species (ROS) and apoptosis were assessed under control and silica exposed conditions. Moreover the cellular uptake of fluorescein isothiocyanate-coniugated (FITC-SiO_2) NP was also investigated by both fluorimetric analysis and fluoresce microscopy. Exposure to silica NP at dosage levels between 50 and 100 μg/ml decreased cell viability in a dose- and time-dependent manner. Increase of intracellular ROS level, reduction of mitochondrial membrane potential and cytochrome C release were also observed in silica nanoparticle-exposed ECV304 cells. Fluorimetric and microscopy fluorescence analysis revealed a dose-dependent uptake of FITC-SiO_2 NP by ECV304 cells. In summary, exposure to SiO_2 NP resulted in a dose-dependent cytotoxicity in cultured ECV304 cells that was associated with increased oxidative stress and mitochondria damage.

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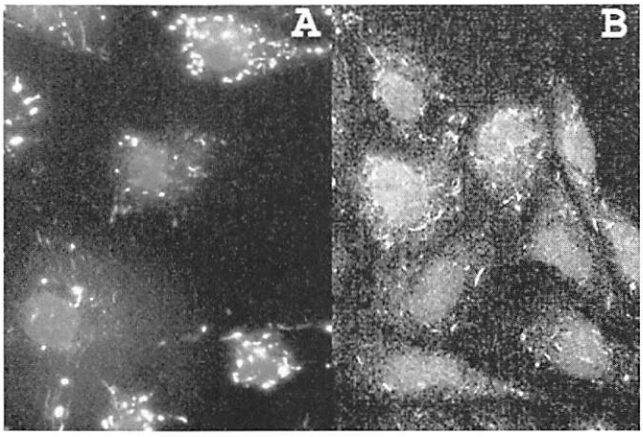

Figure - Mitochondria staining with JC-1 in control (A) and NP-treated (B) endothelial cells.