Involvement of oxidative stress and calcium signaling in oxide nickel nanoparticles - induced alterations in human pulmonary artery endothelial cells

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Objectives

Anthropic activities such as mining amplifies the natural erosion of nickel mines leading to atmospheric emission of oxide nickel nanoparticles (NiONPs). New Caledonia is particularly affected by nickel mining activities. After inhalation, NPs penetrate deeply into the airways and exert deleterious effects on cardiovascular system. Pulmonary artery endothelial cells (HPAEC) can be a direct target of inhaled particles. Alteration in oxidative stress and calcium homeostasis are critical events involved in the pathophysiology of vascular diseases such as pulmonary hypertension. Only a few studies have investigated the effect of NiONPs on pulmonary vascular endothelial cells and the cellular mechanisms remain unclear. The aim of this study was to assess the cytotoxic effects of NiONPs on HPAEC, especially oxidative stress and calcium signaling. HPAEC were exposed for 4 or 24 h to NiONPs (0.5 to 150 µg/cm²). Different endpoints were studied (i) ROS production (ii) cytoplastic and mitochondrial superoxide anion production (iii) superoxide dismutase activity and (vi) calcium signaling.

Materials and Methods

Results

Our results seem to show that NiO NPs-induced intracellular calcium homeostasis impairment is closely correlated to oxidative stress and thus could present a cardiovascular disease risk.

Conclusion