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ENDOCYTOSIS OF CEMENT DUST EXPOSURE AS A CAUSE OF ALTERED MITOCHONDRIAL MEMBRANE POTENTIAL, APOPTOSIS, NADP/NADPH AND OXIDATIVE STRESS IN HUMAN TYPE II EPITHELIAL CELLS

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Mechanisms of cement dust induced toxicity in lung cells have been scarcely studied. In the present study, we investigated endocytosis of cement dust and clinker particles, apoptosis, mitochondrial membrane potential ($\Delta\Psi_m$) alteration, NADP+, NADPH and NADP/NADPH ratio, *in situ* DNA fragmentation and oxidative stress in human alveolar type II epithelial cells (A549) exposed to cement dust particles. The endocytosis of particles was evaluated using transmission electron microscope (TEM), 3-(4, 5-dimethylthiazol-2-yl)-2, 5 diphenyl tetrazolium bromide (MTT) assay, LDH leakage, NADP/NADPH by enzyme linked immunosorbent assay (ELISA) and $\Delta\Psi_m$ using JC-1 dye. Additionally, apoptosis, intracellular reactive oxygen species (ROS) production and reduction of intracellular reduced glutathione (GSH) were quantified using flow cytometry method, while in situ DNA strand break was examined using TUNEL assay. Alveolar epithelial cells evaluation shows endocytosis of cement dust or clinker particles into the cytoplasmic vacuoles. Though, cells exposed to clinker were found to internalized clinker predominantly at the membrane bound vacuoles. Also, both cement dust samples and clinker induced reduction of $\Delta\Psi_m$ and NADP/NADPH ratio with subsequent increased induction of apoptosis and ROS generation. All dust samples induced profound reduction NADP/NADPH ratio which suggests reduction in intracellular reducing power. Collectively, data from this study show that endocytosis of cement dust and subsequent reduction of $\Delta\Psi_m$ and NADP/NADPH ratio, increased apoptosis, intracellular ROS production and reduction of GSH in type II epithelial cells may be key mechanisms of cement dust induced lung cells damage and may contribute significantly to cement dust induced lung fibrosis.

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