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Title	**The Involvement Of Mitochondria In Silica-Induced Apoptosis Of Pulmonary Phagocytes**
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Abstract	**Purpose.** We have previously shown that silica particles, a ubiquitous occupational fibrogenic agent that induces inflammatory responses through microtubule alterations, may activate caspase cascade leading to cell apoptosis. The purpose of this study is to investigate the potential involvement of mitochondria in silica-induced apoptosis. **Methods.** Rats were intratracheally instilled with saline or silica (20mg/kg) particles and were sacrificed 3 days post-exposure. Alveolar macrophages (AMs) were harvested by bronchoalveolar lavage. Apoptosis was measured by cell death detection ELISA with or without microtubule modifiers (taxol or tetrandrine). Intracellular generation of reactive oxygen species (ROS) was assayed by using confocal microscopy. Mitochondrial trans-membrane potential was examined using Jc-1 staining. Mitochondrial release of Cytochrome c and the resulting activation of caspase-9 and caspase-3 were determined by Western blots. **Results.** Silica particles increased the intracellular generation of ROS as compared to saline control. This effect was enhanced by microtubule modifiers taxol and tetrandrine. Silica along with the microtubule modifiers caused mitochondrial membrane depolarization and cytochrome c release. Caspase-9 and caspase3 were also induced by the silica treatment. **Conclusion.** The results show that silica particles induce AM apoptosis that involves alteration of microtubule structure and the activation of the mitochondria by oxygen radicals.