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Induction of AP-1-MAPKs Signaling by Copper Oxide Nanoparticles

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Abstract

Occupational exposures to copper dusts or fumes have been reported to be harmful to human health, with possible risk of cancer among copper smelter workers. Copper (II) oxide (CuO) nanoparticles have not, to our knowledge, been extensively examined for potential carcinogenic or genotoxic effects. To investigate the mechanisms of CuO-induced pathogenesis, the effect of CuO on AP-1-MAPKs and ROS generation were investigated. The results indicated CuO caused a 2-fold increase in AP-1 activity in JB6 cells. The induction of AP-1 activity in cultured cell lines was time and dose-dependent. The signal transduction pathways for AP-1 activation were also investigated. Western Blot analysis demonstrate that CuO stimulates phosphorylation of p38 MAPK and ERKs. CuO also generated ROS when incubated with the cells as measured by electron spin resonance (ESR). Nano-sized CuO generated more ROS than the fine sized particles when incubated with the cells. Comet assay suggested that exposure of the cells with CuO resulted in DNA damage. Unraveling the complex mechanisms associated with these events may provide insights into the initiation and progression of CuO-induced pathogenesis.

Footnotes

This abstract is from the Experimental Biology 2016 Meeting. There is no full text article associated with this abstract published in The FASEB Journal.