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TITLE

Zinc oxide nanoparticles selectively induce apoptosis in cancer cells mediated through reactive oxygen species

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inc oxide nanoparticles (ZnO NPs) are increasingly recognized for their utility in Libiological applications including biosensor and nanomedicine. It has been reported that ZnO NPs induce selective cytotoxicity to cancers cells. However, little is known about the toxicity mechanisms of ZnO NPs in cancer cells. This study was designed to investigate the possible mechanisms of apoptosis induced by ZnO NPs in human cancer cells. Cytotoxicity results showed that ZnO NPs have distinct effects on mammalian cell viability via killing cancer cells (A549, BEAS-2B and BALB 3T3) while posing no impact on normal rat astrocytes. To investigate the possible mechanisms of toxicity caused by ZnO NPs we have further utilized the human lung cancer cells (A549). ZnO NPs was found to induce reactive oxygen species (ROS) generation, oxidative stress and activities of caspase-3 & caspase-9 in a dose- and time dependent manner. Western blot results showed that ZnO NPs induced the expression of heat shock protein 70, a first-tier marker of cell damage and a cell-cycle checkpoint protein p53. Moreover, proapoptotic protein bax was up-regulated and the anti-apoptotic proteins, survivin and bcl-2 were down-regulated in ZnO NPs exposed cells. In conclusion, our data demonstrates that ZnO nanorod induced apoptosis in human lung cancer cells through ROS and oxidative stress via p53, survivin, bax/bcl-2 and caspase pathways. Reference: Ahamed et al. 2011. Nanomedicine: Nanotechnology, Biology and Medicine. doi.org/10.1016/j. nano.2011.04.011

Biography

Maqusood Ahamed is an Assistant Professor of King Abdullah Institute for Nanotechnology at the King Saud University of Riyadh in Saudi Arabia. He received his Ph.D. degree from Hamdard University in 2007 and did his postdoctoral research (2007-2010) at University of Dayton, Ohio. Dr. Ahamed has authored more than 30 peer-reviewed articles and serves as reviewers for more than 20 journals.