Zinc oxide nanoparticle energy band gap reduction triggers the oxidative stress resulting into autophagy-mediated apoptotic cell death

Manoranjan Arakha, Jyoti Roy, Parth Sarthi Nayak, Bibekanand Mallick, Suman Jha,

Department of Life Science.

The physico-chemical properties of nanoparticle (NP), such as particle size, surface defects, crystallinity and accessible surface, affect NP photocatalytic activity that in turn defines the NP cytotoxic propensity. Since zinc oxide nanoparticle (ZnONP) energy band gap falls in a range of a semiconductor, the particle possesses photocatalytic activity. Hence, the study correlates energy band gap with cytotoxic propensity of ZnONP. To this end, ZnONPs with varying energy band gap are fabricated by varying calcination temperature. Cytotoxic propensity of the fabricated ZnONPs against HT1080 cell indicates that the particle with least energy band gap shows highest cytotoxicity. The data also indicate that the cytotoxicity is triggered primarily through reactive oxygen species (ROS)-mediated pathway. Additionally, the comet assay and γH2AX activity assay reveal that decreasing energy band gap of the particle increases DNA damaging propensity. Furthermore, cell cycle analysis indicates that the cell treatment with decreasing energy band gap ZnONP results in significant increase in cell population fraction in subG1 phase. Whereas, acridine orange binding assay and increased expression level of LC3II indicate that the cell tries to recover the stress by scavenging damaged cellular biomolecules and ROS using autophagosomes. Nevertheless, cell with the non-recoverable damages led into apoptotic cell death, as confirmed by Annexin V apoptosis assay, DNA fragmentation assay and 4,6-Diamidino-2-phenylindole dihydrochloride (DAPI) staining. *Zinc oxide nanoparticle energy band gap reduction triggers the oxidative stress resulting into autophagy-mediated apoptotic cell death*. More in ***Free Radical Biology and Medicine (2017, Volume 110),***

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