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Copper oxide antioxidant nanoparticles alleviate cellular toxicity caused by colistin

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Objectives : Colistin acts as a bactericide by inducing changes in the permeability of the cytoplasmic membrane of Gram-negative bacteria (GNB). Intrarenal oxidative stress can contribute to colistin – induced nephrotoxicity. Copper is an essential element in humans, and copper nanoparticles (CuNPs) have exhibited excellent catalytic activity in scavenging H_2O_2 and O_2^- due to their strong quantum confinement of electrons in the ultrasmall size regime We investigated the efficiency and mechanisms of CuNPs in colistin-induced kidney cellular injury model.
Methods : Human proximal tubular epithelial (HK-2) cells were treated with colistin, with our without CuNPs. We conducted MTT assay and FACS analysis to assess cellular survival. Western blotting was performed to analyze the MAPK pathway and reinstating autophagy flux. Mitochondrial damage was evaluated using Mitotracker, while ROS production was detected through DCF-DA, DHE.
Results : CuNPs reduced the production of reactive oxygen species (ROS) production, decreased the level of pro-inflammatory markers (particularly mitogen-activated protein kinases) associated with colistin-induced cellular toxicity, and enhanced cellular survival as observed in MTT and Annexin V stained FACS analysis. Additionally, CuNPs showed the restoration of autophagy flux in colistin exposed HK-2 cells.
Conclusions : Collectively, these findings suggest that CuNPs ameliorate colistin-induced acute cellular injury, emphasizing a novel potential therapeutic drug for treating drug-related nephrotoxicity.