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Copper Oxide Nanoparticles as Antioxidants: Alleviating Kidney Injury and Promoting Autophagy Flux in a Cellular Model of Palmitate-Induced Lipid Accumulation

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Objectives : Obesity stands out as one of the most significant risk factors for the initiation of new chronic kidney disease with the incidence of obesity-related glomerulopathy increasing tenfold in recent years. Copper oxide nanoparticles (CuNPs) have been recognized for their effectiveness as antioxidants, and their small molecular weight suggests high renal clearance. In this study, we explored the efficacy of CuNPs in alleviating kidney injury induced by lipid overload and explored the regulatory mechanisms in a cellular model of palmitate-induced lipid accumulation.
Methods : A cellular model of lipid accumulation was established by exposing HK-2, a proximal renal tubular epithelial cell, to palmitic acid. CuNPs were synthesized using a chemical reduction technique. Subsequent experiments involved PCR, immunoblotting, and immunoflourescence assays.
Results : Oil Red O staining revealed a reduction in palmitate-induced lipid deposition in HK-2 cells treated with CuNPs. mRNA expression related to lipid metabolism showed that the CuNPs treatment suppressed the expression of SREBF1, FASN and ACC. CuNPs were observed to effectively decrease oxidative stress and reduce fibrosis in lipid-accumulated HK-2 cells. TFEB, a crucial transcription factor involved in the regulation of lysosomal biogenesis and autophagy, exhibited reduced nuclear localization in palmitate-induced HK-2 cells. However, CuNPs treatment activated the nuclear translocation of TFEB, restoring autophagy flux function. CuNPs enhanced the nuclear localization of TFEB through the PI3K/Akt/GSK3β signaling pathway.
Conclusions : These findings demonstrated that CuNPs promote autophagy flux through the PI3K/Akt/GSK3β-TFEB signaling pathway, showcasing their antioxidative function. These results lead to the beneficial effects on renal lipid deposition and fibrosis. This provides new insights into potential therapeutics for obesity-induced kidney disease.