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Prophylactic treatment with antioxidant nanoparticles attenuate ischemia/reperfusion injury in BALB/c mice

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Objectives: The kidney is more susceptible to hypoxic damage than other organs because most of its oxygen consumption is used to move various solutes across its membranes. Oxidative stress is one of the important causes of hypoxia-induced kidney injury. The ceria nanoparticle (CNP) is known to exhibit free radical scavenger and catalytic activities. When zirconia is attached to CNPs (CZNPs), the ceria atom tends to remain in a Ce^{3+} form and its efficacy as a free radical scavenger increases. We studied the effectiveness of CNP and CZNP antioxidant activities against hypoxia-induced acute kidney injury.

Methods: We performed *in-vitro* and *in-vivo* studies in HK-2 cells and BALB/c mice under hypoxia condition with 2-3nm CNPs and CZNPs.

Results: We found that the effects of hypoxia on ROS levels were significantly reduced by the presence of CNPs and CZNPs in both the cytoplasm and mitochondria. CNP and CZNP exposure downregulated proinflammatory markers and reduced caspase 3/7 activity in hypoxic kidney (HK-2) cells. The number of mitochondria was recovered and mitochondrial fission was reduced by treatment with both types of nanoparticle. In vivo experiments further revealed that CZNPs could effectively attenuate hypoxia-induced acute kidney injury in BALB/c mice.

Conclusions: Collectively, we found from our analysis that CNPs and CZNPs has a successful uptake into HK-2 cells and that both can effectively counteract ROS induction by hypoxia, in addition to attenuating hypoxia-induced acute kidney injury in vivo. This suggests that treatment with these particles could be a novel approach to controlling hypoxia-induced acute kidney injury.