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ZnO NPs exposure impairs cranial neural crest development by inducing excessive oxidant stress level during chick embryogenesis

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Zinc oxide Nanoparticles (ZnO NPs) could lead to defects in chick craniofacial skeleton during embryogenesis, but the further mechanism is still not clear. In this study, we found ZnO NPs exposure cause craniofacial development defect, including shorten coracoids, curved coracoids and impaired parietal bone, which is derived from the cranial neural crest cells. we observed that ZnO NPs could inhibit the production and migration of cranial neural crest cells, which mainly caused by altering expression of epithelial-mesenchymal transitions-related adhesion molecules. Moreover, we observed that ZnO NPs significantly induced the extent apoptosis in cranial neural crest cells but not affect the cell proliferation. Then, the level of oxidant stress of cranial neural crest cells is elevated by ZnO NPs treatment. The usage of antioxidant N-Acetyl-L-cysteine (NAC) proved that oxidant stress may involved in regulating the apoptosis and development of cranial neural crest cells in the present of ZnO NPs. In conclusion, our results suggest that ZnO NPs treatment could inhibit the development of cranial neural crest cells by inducing cells apoptosis, which might result from the disorder of oxidant stress level.