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Paraquat causes CIK cell apoptosis and programmed necrosis in a dose-dependent manner through oxidative stress/NF-κB pathway

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Paraquat (PQ) is widely used as a quick-acting herbicide. The abuse of PQ causes water pollution and fishery losses, but the toxicity mechanism of PQ to fish kidney cells (CIK) has not been reported yet. In this experiment, it was found through inverted microscope observation that PQ exposure can significantly change the morphology of CIK cells, and exposure to high concentrations for 48 hours basically lost cell morphology. Through the detection of CAT, SOD and MDA kits, it is found that PQ exposure affects the homeostasis and balance of the antioxidant system of CIK cells. Through RT-PCR and Western bolt methods, it was found that PQ promoted the expression of pro-inflammatory factors (MyD88, NF-κB, TNF-α) in CIK cells. Through cell flow cytometry, it was found that PQ exposure caused CIK cells to undergo dose-dependent apoptosis and programmed necrosis. The expression of Bcl-2, Cyt-c, Bax, MLKL, RIPK1 and RIPK3 increased, and the expression of p53 decreased. In summary, we found that PQ exposure induces apoptosis and programmed necrosis of CIK in a dose-dependent manner through the oxidative stress/NF-κB pathway. This experiment provides theoretical support for the safe use of pesticides and protection of the ecosystem and has certain reference value for the safety risk assessment of PQ and the safety protection of animals and humans.

Keywords: Paraquat; CIK; Oxidative stress/NF-κB; Apoptosis; Programmed necrosis;

Biography:

Female, 24, born on July 20, 1997, graduated from Northeast Agricultural University with a bachelor's degree. Now studying in Clinical Veterinary Medicine, School of Animal Medicine, Northeast Agricultural University, and supervisor is Professor Xu Shiwen