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Effects of triclocarban on oxidative stress and innate immune response in
zebrafish embryos

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Abstract

Triclocarban (TCC) is used in many household and personal hygiene products. TCC has been widely detected in wastewater around the world. The present study reveals that TCC can activate oxidative stress, induce total antioxidant capacity expression and lipid peroxidation, and increase the activities of superoxide dismutase and other antioxidant enzymes to resist oxidative damage. A significant induction of concentrations of proinflammatory mediator and nitric oxide (NO), accompanied by an upregulated expression of inducible NO synthase gene, was detected in zebrafish embryos exposed to TCC. The transcription of immune-response-related genes, including *tnf- α* , *il-1 β* , *il-4*, *il-8*, and *cxcl-clc*, was significantly upregulated on exposure to TCC. Furthermore, we found that the exposure of zebrafish embryos to TCC decreased immune cell recruiting in the head. Expressions of *nf- κ b*, *trif*, *myd88*, *irak4*, and *traf6* were altered on exposure to TCC. These results demonstrated that exposure to TCC at environmental concentrations significantly affects the expression of immune-response-related genes in zebrafish embryos following oxidative stress and the release of proinflammatory mediators through Toll-like receptor signaling pathway. Thus, we assumed that the ecological risk of TCC on aquatic organisms could not be ignored.

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