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Impact of binary waterborne mixtures of nickel and zinc on hypothalamic-pituitary-testicular axis in rats

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HIGHLIGHTS

- Influence of Ni and Zn co-exposure on reproductive function was studied in rats.
- Reduction of hormones by Ni alone was abated in Zn and Ni co-exposed rats.
- Zn abrogated Ni-induced oxidative injury in hypothalamus, testes and epididymis of rats.
- Zn suppressed induction of inflammation and caspase-3 action by Ni in rats.
- Zn abated Ni-induced histological lesions in rats.

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ABSTRACT

Several evidences from the literature showed that the coexistence of nickel and zinc in polluted waters is related to the similarity in their geogenic and anthropogenic factors. Although most environmental exposures to metals do not occur singly, there is a paucity of scientific knowledge on the effects of zinc and nickel co-exposure on mammalian reproductive health. The present study investigated the influence of co-exposure to nickel and zinc on male reproductive function in rats. Experimental rats were co-exposed to environmentally relevant concentrations of waterborne nickel (75 and 150 μg $NiCl_2$ L^{-1}) and zinc (100 and 200 μg $ZnCl_2$ L^{-1}) for 45 successive days. Subsequently, reproductive hormones were assayed whereas the hypothalamus, epididymis and testes of the rats were processed for the assessment of oxidative stress and inflammation indices, caspase-3 activity and histology. Results indicated that co-exposure to nickel and zinc significantly ($p < 0.05$) abolished nickel-mediated diminution of antioxidant defense mechanisms while diminishing levels of reactive oxygen and nitrogen species and lipid peroxidation in the hypothalamus, epididymis and testes of the exposed rats. Additionally, co-exposure to zinc abated nickel-mediated diminutions in luteinizing hormone, follicle-stimulating hormone, serum and intra-testicular testosterone with concomitant enhancement of sperm production and quality. Further, zinc abrogated nickel-mediated elevation in inflammatory biomarkers including nitric oxide, tumor necrosis factor alpha, interleukin-1 beta as well as caspase-3 activity. The protective influence of zinc on nicked-induced reproductive toxicity was well supported by histological data. Overall, zinc ameliorated nickel-induced reproductive dysfunction *via* its anti-oxidant, anti-inflammatory, anti-apoptotic and spermato-protective activities in rats.

1. Introduction

Nickel (Ni) is the 24th most abundant element in the earth where it exists as compounds in soils, waters and fumes (Ciurli and Mangani, 2001; Rinklebe and Shaheen, 2017). The various forms of Ni are widely used in many industries for the production of many substances including watches, mobile phones, coins, belt buckles, coronary stents, dental and orthopedic implants (Schmidt and Goebeler, 2011). Furthermore, environmental contamination enhances human exposure to Ni *via* inhalation and ingestion of contaminated food. Recently, the contamination of groundwater and surface dregs by Ni in several countries including China,

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