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Selenium abates reproductive dysfunction *via* attenuation of biometal accumulation, oxido-inflammatory stress and caspase-3 activation in male rats exposed to arsenic☆

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ABSTRACT

Frequent exposure to arsenic is well documented to impair reproductive function in humans and animals. Biological significance of inorganic selenium and organoselenium, diphenyl diselenide (DPDS), has been attributed to their pharmacological activities. However, their roles in arsenic-mediated reproductive toxicity is lacking in literature. The present study evaluated the protective effects elicited by selenium and DPDS in arsenic-induced reproductive deficits in rats. Animals were either exposed to arsenic alone in drinking water at 60 μg AsO_2Na L^{-1} or co-treated with selenium at 0.25 mg kg^{-1} or DPDS at 2.5 mg kg^{-1} body weight for 45 consecutive days. Results indicated that arsenic-mediated deficits in spermatogenic indices and marker enzymes of testicular function were significantly abrogated in rats co-treated with selenium or DPDS. Additionally, selenium or DPDS co-treatment prevented arsenic-mediated elevation in oxidative stress indices and significantly suppressed arsenic-mediated inflammation evidenced by diminished myeloperoxidase activity, nitric oxide, tumor necrosis factor alpha and interleukin-1 beta levels in hypothalamus, testes and epididymis of the rats. Moreover, selenium or DPDS abrogated arsenic mediated activation of caspase-3 activity and histological lesions in the treated rats. Taken together, selenium or DPDS improved reproductive function in arsenic-exposed rats *via* suppression of inflammation, oxidative stress and caspase-3 activation in rats.

1. Introduction

Arsenic is a ubiquitous sulfhydryl-reactive metalloid and environmental pollutant commonly released to the environment during volcanic and industrial activities (Zubair et al., 2017; Renu et al., 2018). In abundance, arsenic was rated 20th in the earth's crust, 14th in seawater and 12th in the human body (Pereira et al., 2010). The major cause of arsenic toxicity in human is related to arsenic contamination of drinking water from natural geological sources in addition to mining, smelting, pesticides and fertilizers (Missimer et al., 2018). Epidemiological studies indicated that chronic arsenic exposure is associated with increased risk of reproductive dysfunction evidenced by induction of erectile dysfunction, oligozoospermia and sperm chromosomal aberrance in countries including china, Taiwan, India and Mexico (Hsieh et al., 2008; Xu et al., 2012; Sengupta et al., 2013; Shen et al., 2013; Wang et al., 2016; Wang et al., 2017).

Previous studies have demonstrated that exposure of rodents to arsenic resulted in significant accumulation of arsenic in testes and accessory sex organs, decreased testicular weight, accessory sex organ weights and epididymal sperm counts with marked degeneration of germ cells (Guvvala et al., 2016; Ferreira et al., 2012; da Cunha de Medeiros et al., 2019). Arsenic reportedly elicits toxicity *via* generation of reactive oxygen species (ROS) due to its electrophilic nature as well as its interaction with iron (Jomova et al., 2011; Renu et al., 2018). Elevated intracellular ROS concentrations have been implicated in reproductive dysfunction due to their involvement in cellular oxidative damage to cell structures, nucleic acids, lipids and proteins (Lavranos et al., 2012). Moreover, arsenic reportedly caused testicular damage *via* induction of inflammation and apoptosis in animals (Shao et al., 2018; Zeng et al., 2019).

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