ABSTRACT

The present study elucidated the profound protective role of vitamin B_{12} and folic acid against arsenic-mediated reproductive toxicity in female rats. Ingestion of sodium-arsenite via drinking water [0.4 ppm/100 g body weight (b.wt.)/day] followed by the co-administration of vitamin B_{12} and folic acid (0.07 μg and 4.0 μg respectively/100 g b.wt./day) in Wistar rats represented a notable protection against arsenic-induced disruption of female gonadal function, ROS generation and DNA fragmentation of ovarian and uterine tissues as well as disorganization of utero-ovarian histoarchitecture. However, arsenication impaired the action of the free radical scavenging enzymes superoxide dismutase (SOD) and catalase, peroxidase (POD) in ovarian and uterine tissue, with a concomitant elevation in lipid peroxidation in the reproductive organs. Arsenic ingestion resulted significant drop in the circulating follicle-stimulating hormone (FSH), leutinizing hormone (LH), and estradiol along with retarded activities of Δ^5,3β -hydroxysteroid dehydrogenase (Δ5,3β-HSD) and 17β-hydroxysteroid dehydrogenase (17β-HSD). Arsenicated rats showed irregular estrous cycle dominated by lengthy diestrus. Our previous work on arsenic-exposed humans was associated with DNA injury and carcinogenesis. Here, we demonstrated that the supplementation of aforesaid physiological/therapeutic dose of vitamin B_{12} and folate protected the rodents appreciably from arsenic-induced DNA damage (DNA fragmentation and comet assay) and ovarian and uterine tissue disintegration (histoarchitecture, HE staining). Vitamin supplementation mitigated the arsenic mediated reproductive injury by preventing abundant generation of free radicals. Restrained generation of free radicals may be correlated to the protection of DNA stability and reproductive organs morphology. This study highlighted that the decisive role of vitamin B_{12} and folic acid in ameliorating arsenic-mediated reproductive disorder.

Key words: arsenic in drinking water; female rat reproductive and metabolic profile; antioxidant systems; steroidogenesis; DNA fragmentation; utero-ovarian histoarchitecture; adverse pregnancy; hepatotoxicity; remedial supplementation: vitamin B_{12} ; folic acid.